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Review

# Changes in sleep patterns after vagus nerve stimulation, deep brain stimulation or epilepsy surgery: Systematic review of the literature

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ABSTRACT

*Purpose:* Perform a systematic review of the literature on the effects of vagus nerve stimulation (VNS), deep brain stimulation (DBS) and epilepsy surgery in subjective and objective sleep parameters.
*Methods:* We performed a literature search in the main medical databases: Medline, Embase, Cochrane, DARE and LILACS, looking for studies that evaluated the effects of VNS, DBS or epilepsy surgery on sleep parameters. In all, 36 studies, coming from 11 countries, including reviews, cohort studies, case series and case reports were included.
*Results:* VNS induces sleep apnoea dependent of the stimulation variables. This condition can be reverted modifying these settings. Surgical procedures for epilepsy cause an improvement in objective and subjective sleep parameters that depend on the success of the procedure evaluated through ictal frequency control. There is evidence that non-pharmacologic treatment of epilepsy has different effects on sleep patterns.
*Conclusion:* It is advisable to include objective and subjective sleep parameters in the initial evaluation and follow-up of patients considered for invasive procedures for epilepsy control, especially with VNS due to the risk of sleep apnoea. More high quality studies are needed.


## 1. Introduction

In patients with epilepsy, multiple factors such as ictal frequency, interictal activity and anticonvulsant therapy affect sleep architecture and quality [1]. Daytime somnolenceis the most common complaint of patients with epilepsy is [1,2]. An increase in obstructive sleep apnoea/hypopnea syndrome (OSAHS) has been documented in patients with refractory epilepsy, in whom, with an incidence as high as 33% [3], associated with a decline in the quality of life [4].

The objective of this systematic review was to evaluate current evidence on the effects of non-pharmacological treatments, including vagal nerve stimulation (VNS), deep brain stimulation (DBS) or epilepsy surgery on both objective and subjective sleep parameters.

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## 2. Methods

### 2.1. Search strategy

We performed a systematic review of Medline (Pubmed), Embase, Cochrane Database of Systematic Reviews, Database of Abstracts of Reviews of Effects – DARE and Latin American and Caribbean Literature in Health Sciences – LILACS. Terms used and adapted for each database were:

(epilepsy OR epilepsies OR epileptic OR seizures)
AND
(surgery OR surgical OR "deep brain stimulation" OR DBS OR callosotomy OR lobectomy OR vagus)
AND
(Sleep OR REM OR insomnia OR somnolence OR sleepiness OR polysomnography OR somnography OR hypersomnia OR parasomnia OR osa OR apnea OR dreams)

We limited the search to articles with abstract available and published from January 1, 2000 to the present (search date June 15, 2016). There was no language restriction. We included meta-analyses or systematic reviews, randomized clinical trials, cohort studies, case series or individual case reports, focusing on the effect

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of epilepsy surgery, VNS or DBS on objective or subjective sleep parameters.

Initial exclusion of irrelevant articles, based on title and abstract, was done by 2 independent members of the research group (ORO, SGT); full-text versions were obtained for data extraction. The list of references of each study was analysed manually. Subsequently, the articles were classified according to the type of procedure, and according to the American Academy of Neurology's classification of evidence [5].

## 3. Results

Fig. 1 shows the selection process. The initial search retrieved 2717 references, of which finally 36 articles with a class of evidence III and IV were reviewed; 15 came from United States; five from Italy, four from France; three from Canada, two from Sweden, and one each from Australia, Austria, Brazil, Cyprus, Finland, Germany, and India; 27 articles referred to VNS, 8 to epilepsy surgery and 1 to DBS.

### 3.1. Vagus nerve stimulation

Of the 27 articles on VNS (Table 1), 11 were case reports, nine prospective descriptive studies and seven retrospective descriptive studies. These included a total of 209 patients between the ages of 3 and 58 years.

The effect of VNS on subjective parameters of sleep quantified by daytime sleepiness was evaluated in three studies. Rizzo et al. [6], using a sleep diary, described an improvement of daytime sleepiness with the chronic use of the stimulator. The study by Malow et al. [7] found that the score on the Epworth scale improved from $7.2 \pm 4.4$ to $5.6 \pm 4.5$, unrelated to a decrease in ictal frequency or changes in the sleep stages through polysomnographic evaluation. These findings contrast with the case report of St Louis et al. [8] in which a patient developed snoring and an increase in the score on the Epworth scale following implantation of the VNS, both of which improved after reducing its pacing times.

Carrosela et al. [9] reported the case of a patient with deep sleep status epilepticus in which there was an improvement in the perception of well-being, also finding an association between the decrease in the number of crises with improvement in cognition, language, school performance and decrease in irritability. In the study by Galli et al. [10], quality of life was evaluated, finding an improvement related to decreased sleep latency as measured by the Multiple Sleep Latency Test (MSLT).

Objective evaluation using polysomnography showed a relationship between sleep disturbances and VNS in 16 studies [3,8,11–24]. During the ON period [9,14,19], the discharge frequency and the configuration with fast cycling (ON/OFF) [13,18] were associated with an increase in the apnoea-hypopnoea index (AHI), the number of awakenings [14], and a decrease in respiratory effort and tidal without alteration in the AHI [3,15,20]. In all cases, the occurrence of apnoea was correlated with the ON period of the stimulator, and turning it off, or adjusting the setting with decreased stimulation parameters achieved an adequate control of respiratory symptoms in most patients [8,11,17], others required positive pressure ventilation [15,22].

Two studies with polysomnographic evaluation described the presence of vocal cord adduction during the ON stimulation period, evidenced by indirect laryngoscopy, associated with the onset or worsening of respiratory sleep disorder [23,25], suggesting a possible explanation for the mechanism by which the stimulator induces sleep apnoea.

In children, disturbances in the cardiorespiratory regulation were described during stimulation activation, including an increase in frequency and a decrease in respiratory amplitude, with variable changes from patient to patient in heart rate, which results in poor optimization of tissue oxygenation [21,26]. In adults, tachypnoea was the only disturbance reported after the ON period, with no significant changes in heart rate [27].

Other reported polysomnographic findings associated with VNS are: higher intensity of the stimulator attenuates the REM sleep, whereas lower intensity improves the wakeful state [27]. Stimulator discharges are associated with a significant increase in respiratory rate and a decrease in the range of abdominal distension, inducing a decrease in oxygen saturation [28]. Finally, VNS induces a significant increase in slow-wave sleep and decreases both sleep latency and stage 1 [29].

The objective evaluation by means of electroencephalographic parameters (EEG) found that VNS produced an increase of the spectral power in the delta and theta wave EEG in non-REM sleep, and alpha waves in REM sleep and wakefulness, without significant decrease in interictal activity during sleep [30]. In the epileptic state of deep sleep, there was no epileptiform activity at the follow-up year, associated with cognitive improvement [9]. A decrease in the number of interictal epileptiform discharges, especially during REM and delta sleep were also described,as well as other minor alterations in the EEG [27].

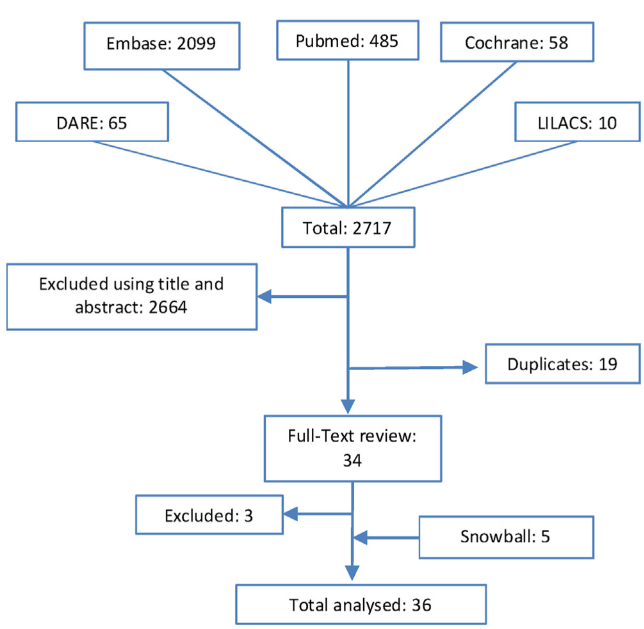


**Fig. 1.** Study selection process.

### 3.2. Epilepsy surgery

Eight articles refer to the effect of epilepsy surgery on sleep [32–39] (Table 2); two of these are case-control studies, two prospective case series, two retrospective, and two case reports, with a total of 207 patients aged between 3 and 62 years.

In the class III study performed by Zanzmera et al. [32], 17 patients with refractory focal epilepsy, mostly of the mesial temporal type, followed by frontal and parietal, found a statistically significant reduction in ictal frequency ($p = .01$) with improvement in self-reported sleep parameters, such as the duration of night-time sleep ($p = .01$), total sleep duration ($p = .03$), and a reduction in Epworth score ($p = .02$) in patients with good postoperative outcome. In the polysomnographic study, a decrease in AHI was found in patients with good postoperative outcome with respect to the basal state and the poor outcome group.

**Table 1**
Studies of the effects of the vagus nerve stimulator on sleep.

| Author and year of publication | Type of study (type of evidence) | Number of patients | country | Age at time of evaluation | Type epilepsy | Quantification |
|---|---|---|---|---|---|---|
| Malow [3] | Case series (III) | 4 | United States | 35, 57, 31, 20 | Refractory epilepsy | Polysomnograhy |
| Rizzo [6] | Observational prospective (III) | 10 | Italy | 22–43 | Refractory epilepsy | Polysomnograhy sleep diary |
| Malow [7] | Prospective descriptive (III) | 16 | United States | 33.6 ± 11.1 | Refractory epilepsy | Polysomnograhy Multiple Sleep Latency Test Epworth |
| St Louis [8] | Case report (IV) | 1 | United States | 23 | Extratemporal focal refractory epilepsy | Polysomnograhy Epworth |
| Carosella [9] | Case report (IV) | 1 | United States | 12 | Electrographic status epilepticus in deep sleep, refractory | EEG Family information |
| Galli [10] | Prospective descriptive (III) | 8 | Italy | 43 ± 10.6 | Refractory epilepsy | Multiple Sleep Latency Test Visual reaction times Perceived well-being |
| Bhat [11] | Case report (IV) | 1 | United States | 22 | Refractory epilepsy | Polysomnograhy |
| Ebben [12] | Case report (IV) | 1 | United States | 54 | Focal Refractory | Polysomnograhy |
| Gschliesser [13] | Case report (IV) | 2 | Austria | 35 and 36 | Focal refractory | Polysomnograhy |
| Holmes [14] | Case report (IV) | 1 | United States | 21 | Refractory, primary generalized epilepsy | Polysomnograhy |
| Upadhyay [15] | Case report (IV) | 1 | United States | 14 | Refractory epilepsy, Stroke | Polysomnograhy |
| Parhizgar [16] | Case report (IV) | 1 | United States | 5 | Refractory epilepsy | Polysomnograhy |
| Joutsa [17] | Case report (IV) | 2 | Finland | 11 and 15 | Refractory epilepsy | Polysomnograhy |
| Khurana [18] | Descriptive retrospective (III) | 26 | United States | 3–17 | Refractory epilepsy | Polysomnograhy |
| Marzec [19] | Retrospective observational (III) | 16 | United States | 21–58 | Refractory epilepsy | Polysomnograhy |
| Nagarajan [20] | Case series (III) | 8 | Australia | 4–16 | Refractory epilepsy | Polysomnograhy |
| Zaaimi [21] | Observational prospective (III) | 10 | France | 7–18 | Refractory epilepsy | Polysomnograhy |
| Hsieh [22] | Case series, retrospective (III) | 9 | Canada | 6–20 | Refractory epilepsy | Polysomnograhy |
| Aron [23] | Case report (IV) | 1 | Canada | 10 | Lennox-Gastaut | Polysomnograhy |
| Papacostas [24] | Case report (IV) | 1 | Cyprus | *** | Refractory epilepsy | Polysomnograhy |
| Zambrelli [25] | Prospective descriptive (III) | 23 | Italy | 41.3 ± 10.3 | Refractory epilepsy | Polysomnograhy |
| Zaaimi [26] | Observational prospective (III) | 10 | France | 7–18 | Refractory epilepsy | Polysomnograhy |
| Murray [27] | Case series, retrospective (III) | 6 | United States | *** | Refractory epilepsy | EEG Cardiac and respiratory frequency |
| Zaaimi [28] | Case series, retrospective (III) | 10 | France | 7–18 | Refractory epilepsy | Polysomnograhy |
| Hallböök [29] | Prospective descriptive (III) | 15 | Sweden | 4–17 | Focal Refractory | Polysomnograhy Quality of life |
| Rizzo [30] | Observational prospective (III) | 10 | Italy | 22–43 | Refractory epilepsy | EEG |
| Hallböök [31] | Prospective descriptive (III) | 15 | Sweden | 4–17 | Refractory epilepsy Developmental delay | Telemetry |

EEG: electroencephalogram.
*** Does not specify the age of the patients involved in the study.

Four studies evaluated the effects of temporal lobe surgery for hippocampal sclerosis on sleep parameters, and found that patients with refractory temporal lobe epilepsy have poor sleep quality, as measured by the Pittsburgh scale, which improved after the procedure [33]. The presence of interictal epileptiform discharges predicts the onset of aura or epileptic seizures in the postoperative period; EEG during sleep allows the identification of these patients, since half of interictal discharges occur during sleep [34]. The adequate postoperative outcome resulted in a reduction of epileptic seizures, a decrease of interictal discharges, and even cognitive improvement [35]. In addition to decreased ictal frequency and interictal epileptiform activity, there was anincrease in total sleep time, and in the percentage of REM sleep, with a decrease in the number of awakenings and in sleep latency [36].

In patients with nocturnal sleep epileptic status treated with hemispherectomy or local resection, there is improvement in ictal frequency and resolution of epileptiform activity, associated with improvement in neuropsychological tests [37]. In the case-control study of McCormick [38], patients with refractory generalized epilepsy treated with right hemispherectomy found lower sleep efficiency, with no difference in total sleep time, sleep stage time, or sleep latency. Foldvary-Schaefer et al. [39] presented a patient with refractory frontal lobe epilepsy, who achieved significant improvement in severe sleep apnoea following the surgical procedure, associated with an improvement in ictal frequency.

### *3.3. Deep brain stimulation*

A single prospective observational study of 9 patients undergoing DBS performed by Voges et al. [40] in Germany, with anterior thalamus stimulation for the treatment of refractory epilepsy, showed that this procedure increased the number of awakenings in a voltage-dependent manner with a consecutive increase in neuropsychiatric symptoms, which improved decreasing the stimulation voltage during sleep.

## 4. Discussion

This review compiles the literature published in the last 15 years on the effect of invasive procedures for the treatment of epilepsy on objective and subjective sleep parameters. These sleep patterns have been studied in a variety of procedures, and in populations of different ages. Conclusions are limited due to the number of studies and to their observational nature (class III and IV),;furthermore, results are not always consistent from one study to another. The review, however, highlights the importance of studying sleep in patients with epilepsy, not only because of the potential risk of seizures with sleep deprivation, but also because of the possibility of comorbidities associated with sleep disorders, such as cardiovascular risk posed by OSAHS. The association of sleep apnoea with VNS seems well substantiated, both in children and in adults. This association is consistent with the evidence of vagal inhibition as a means to reduce AHI in patients with severe

**Table 2**
Studies of the effects of epilepsy surgery on sleep.

| Author and year of publication | Type of study (type of evidence) | Number of patients | country | Age at time of evaluation | Type epilepsy | Quantification |
|---|---|---|---|---|---|---|
| Zanzmera [32] | Prospective cohort (III) | 17 | India | Average 18.1 | Refractory (mesial, frontal and parietal) | Epworth One week sleep log Polisomnography |
| Carrion [33] | Case – control (III) | 59 | Brazil | 22–58 | Refractory hippocampal sclerosis | Pittsburg Epworth |
| Di Gennaro [34] | Descriptive retrospective (III) | 107 | Italy | 11–62 | Refractory hippocampal sclerosis | Video EEG |
| Moseley [35] | Case report (IV) | 1 | United States | 11 | Refractory hippocampal sclerosis | EEG |
| Serafini [36] | Prospective descriptive (III) | 11 | France | Average 36.2 | Refractory hippocampal sclerosis | IRLSSG Berlin questionnaire Hatoum's sleep questionnaire Epworth Video EEG |
| Loddenkemper [37] | Descriptive retrospective (III) | 7 | United States | 3 a 14 | Electrographic status epilepticus in deep sleep, refractory | EEG Neuropsychological tests |
| McCormick [38] | Case – control (III) | 4 | Canada | 14–28 Average 22.2 | Generalized refractory epilepsy right hemispherectomy | EEG Polysomnography |
| Foldvary-Schaefer [39] | Case report (IV) | 1 | United States | 18 | Focal frontal lobe epilepsy | Polysomnography |

EEG: electroencephalogram.
IRLSSG: International restless legs syndrome study group.

OSAHS, described by Garrigue et al. [41]. Apnoeas appear to occur during the ON mode and are dependent on specific stimulation parameters [8,13,14,18,19], meaning its modification can reverse the effects without the need for other interventions in most patients. Perhaps patient comorbidities and risk factors, like age, body mass index or craniofacial anomalies, which are rarely taken into consideration [42], might influence the appearance of apnoea. It is therefore advisable to perform a sleep study in candidates for VNS and to monitor them periodically, in order to prevent and treat changes that may occur as a result of the procedure. A single study of deep brain stimulation [40] also shows that the variables of stimulation (frequency and intensity) can be modified to change possible adverse effects; in this case, an increase in the number of awakenings.

Regarding epilepsy surgery, evidence is clearly insufficient to draw solid conclusions, but apparently the effect of the procedure on sleep depends on the success of the procedure, and is directly associated with good control of the ictal frequency and the absence of postoperative complications [33,36]. In patients with hippocampal sclerosis, a good postoperative outcome leads to a significant improvement in sleep quality [34].

A special case is status epilepticus of deep sleep, which is characterized by continuous slow-wave epileptic discharges during non-REM sleep, which leads to a progressive deterioration of cognition. This was studied in two cases [9,37], in which the control of ictal and interictal activity with hemispherectomy or VNS led to an improvement in both objective and subjective sleep parameters and cognition.

## 5. Conclusion

Given the importance of sleep, and the detrimental effect of related disorders in quality of life, it is advisable to include objective and subjective sleep parameters as part of the initial evaluation and follow-up of patients who are candidates for procedures for the control of epilepsy, in view of a new possible point of intervention in the integral management of the epilepsy patient.

## Conflict of interest

None of the authors has any conflict of interest to disclose.

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## Ethical statement

As a systematic review of the literature, this study did not involve interventions on patients nor following them in time. The work described adheres to the principles of the Committee on Publication Ethics and is consistent with the Journal guidelines for ethical publication. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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## References

[1] Derry CP, Duncan S. Sleep and epilepsy. Epilepsy Behav 2013;26:394–404.
[2] 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.
[3] Malow BA, Levy K, Maturen K, Bowes R. Obstructive sleep apnea is common in medically refractory epilepsy patients. Neurology 2000;55:1002–7.
[4] Jain S, Glauser T. Effects of epilepsy treatments on sleep architecture and daytime sleepiness: an evidence-based review of objective sleep metrics. Epilepsia 2014;55:26–37.
[5] Gross R, Johnston KC. Levels of evidence: taking neurology to the next level. Neurology 2009;72:8–10.
[6] Rizzo P, Beelke M, De Carli F, Canovaro P, Nobili L, Robert A, et al. Chronic vagus nerve stimulation improves alertness and reduces rapid eye movement sleep in patients affected by refractory epilepsy. Sleep 2003;26:607–11.
[7] Malow BA, Edwards J, Marzec M, Sagher O, Ross D, Fromes G. Vagus nerve stimulation reduces daytime sleepiness in epilepsy patients. Neurology 2001;57:879–84.
[8] St Louis EK, Faber K. Reversible sleep-related stridor during vagus nerve stimulation. Epileptic Disord 2010;12:76–80.
[9] Carosella CM, Greiner HM, Byars AW, Arthur TM, Leach JL, Turne M, et al. Vagus nerve stimulation for electrographic status epilepticus in slow-wave sleep. Pediatr Neurol 2016;60:66–70.
[10] Galli R, Bonanni E, Pizzanelli C, Maestri M, Lutzemberger L, Giorgi FS. Daytime vigilance and quality of life in epileptic patients treated with vagus nerve stimulation. Epilepsy Behav 2003;4:185–91.
[11] Bhat S, Lysenko L, Neiman ES, Rao GK, Chokroverty S. Increasing off-time improves sleep-disordered breathing induced by vagal nerve stimulation. Epileptic Disord 2012;14:432–7.

[12] Ebben MR, Sethi NK, Conte M, Pollak CP, Labar D. Vagus nerve stimulation, sleep apnea, and CPAP titration. J Clin Sleep Med 2008;4:471–3.
[13] Gschliesser V, Hogl B, Frauscher B, Brandauer E, Poewe W, Luef G. Mode of vagus nerve stimulation differentially affects sleep related breathing in patients with epilepsy. Seizure 2009;18:339–42.
[14] Holmes MD, Chang M, Kapur V. Sleep apnea and excessive daytime somnolence induced by vagal nerve stimulation. Neurology 2003;61:1126–9.
[15] Upadhyay H, Bhat S, Gupta D, Mulvey M, Ming S. The therapeutic dilemma of vagus nerve stimulator-induced sleep disordered breathing. Ann Thorac Med 2016;11:151–>.
[16] Parhizgar F, Rogers K, Hurst D, Nugent K, Raj R. Vagus nerve stimulator-induced apneas and hypopneas in a child with refractory seizures. J Pediatr Neurol 2012;10:53–6.
[17] Joutsa J, Suomela M, Jutila L, Erkinjuntti M, Soilu-Hänninen M, Müller M. Respiratory disturbance during sleep induced by vagus nerve stimulator. Duodecim 2015;131:1811–5.
[18] Khurana DS, Reumann M, Hobdell EF, Neff S, Valencia I, Legido A, Kothare SV. Vagus nerve stimulation in children with refractory epilepsy: unusual complications and relationship to sleep-disordered breathing. Childs Nerv Syst 2007;23:1309–12.
[19] Marzec M, Edwards J, Sagher O, Fromes G, Malow BA. Effects of vagus nerve stimulation on sleep-related breathing in epilepsy patients. Epilepsia 2003;44:930–5.
[20] Nagarajan L, Walsh P, Gregory P, Stick S, Maul J, Ghosh S. Respiratory pattern changes in sleep in children on vagal nerve stimulation for refractory epilepsy. Can J Neurol Sci 2003;30:224–7.
[21] Zaaimi B, Grebe R, Berquin P, Wallois F. Vagus nerve stimulation therapy induces changes in heart rate of children during sleep. Epilepsia 2007;48:923–30.
[22] Hsieh T, Chen M, McAfee A, Kifle Y. Sleep-related breathing disorder in children with vagal nerve stimulators. Pediatr Neurol 2008;38:99–103.
[23] Aron M, Vlachos-Mayer H, Dorion D. Vocal cord adduction causing obstructive sleep apnea from vagal nerve stimulation: case report. J Pediatr 2012;160:868–70.
[24] Papacostas SS, Myrianthopoulou P, Dietis A, Papathanasiou ES. Induction of central-type sleep apnea by vagus nerve stimulation. Electromyogr Clin Neurophysiol 2007;47:61–3.
[25] Zambrelli E, Saibene AM, Furia F, Chiesa V, Vignoli A, Pipolo C, et al. Laryngeal motility alteration: a missing link between sleep apnea and vagus nerve stimulation for epilepsy. Epilepsia 2016;57:e24–7.
[26] Zaaimi B, Grebe R, Berquin P, Wallois F. Vagus nerve stimulation induces changes in respiratory sinus arrhythmia of epileptic children during sleep. Epilepsia 2009;50:2473–80.
[27] Murray B, Matheson JST. Effects of vagus nerve stimulation on respiration during sleep. Neurology 2001;57:1523.
[28] Zaaimi BHC, Berquin PPM, Grebe RW. Vagus nerve stimulation induces concomitant respiratory alterations and a decrease in SaO2 in children. Epilepsia 2005;46:1802–9.
[29] Hallböök T, Lundgren J, Blennow G, Strömblad LG, Rosén I. Long term effects on epileptiform activity with vagus nerve stimulation in children. Seizure 2005;14:527–33.
[30] Rizzo P, Beelke M, De Carli F, Canovaro P, Nobili L, Robert A, et al. Modifications of sleep EEG induced by chronic vagus nerve stimulation in patients affected by refractory epilepsy. Clin Neurophysiol 2004;115:658–64.
[31] Hallböök T, Lundgren J, Kohler S, Blennow G, Strömblad LG, Rosén I. Beneficial effects on sleep of vagus nerve stimulation in children with therapy resistant epilepsy. Eur J Paediatr Neurol 2005;9:399–407.
[32] Zanzmera P, Shukla G, Gupta A, Goyal V, Srivastava A, Garg A, et al. Effect of successful epilepsy surgery on subjective and objective sleep parameters-a prospective study. Sleep Med 2013;14:333–8.
[33] Carrion MJM, Nunes ML, Martinez JVL, Portuguez MW, da Costa JC. Evaluation of sleep quality in patients with refractory seizures who undergo epilepsy surgery. Epilepsy Behav 2010;17:120–3.
[34] Di Gennaro G, Casciato S, D'Aniello A, De Risi M, Quarato PP, Mascia A, et al. Serial postoperative awake and sleep EEG and long-term seizure outcome after anterior temporal lobectomy for hippocampal sclerosis. Epilepsy Res 2014;108:945–52.
[35] Moseley BD, Dhamija R, Wirrell EC. The cessation of continuous spike wave in slow-wave sleep following a temporal lobectomy. J Child Neurol 2012;27:113–6.
[36] Serafini A, Kuate C, Gelisse P, Velizarova R, Gigli GL, Coubes P, Crespel A. Sleep before and after temporal lobe epilepsy surgery. Seizure 2012;21:260–5.
[37] Loddenkemper T, Cosmo G, Kotagal P, Haut J, Klaas P, Gupta A, et al. Epilepsy surgery in children with electrical status epilepticus in sleep. Neurosurgery 2009;64:328–37.
[38] McCormick L, Nielsen T, Ptito M, Ptito A, Villemure JG, Vera C, Montplaisir J. Sleep in right hemispherectomized patients: evidence of electrophysiological compensation. Clin Neurophysiol 2000;111:1488–97.
[39] Foldvary-Schaefer N, Stephenson L, Bingaman W. Resolution of obstructive sleep apnea with epilepsy surgery: expanding the relationship between sleep and epilepsy. Epilepsia 2008;49:1457–9.
[40] Voges BR, Schmitt FC, Hamel W, House PM, Kluge C, Moll CK, Stodieck SR. Deep brain stimulation of anterior nucleus thalami disrupts sleep in epilepsy patients. Epilepsia 2015;56:e99–103.
[41] Garrigue S, Bordier P, Jais P, Al E. Benefit of atrial pacing in sleep apnea syndrome. N Engl J Med 2002;346:404–12.
[42] Unterberger I, Gabelia D, Prieschl M, Chea K, Hofer M, Högl B. Sleep disorders and circadian rhythm in epilepsy revisited: a prospective controlled study. Sleep Med 2015;16:237–42.