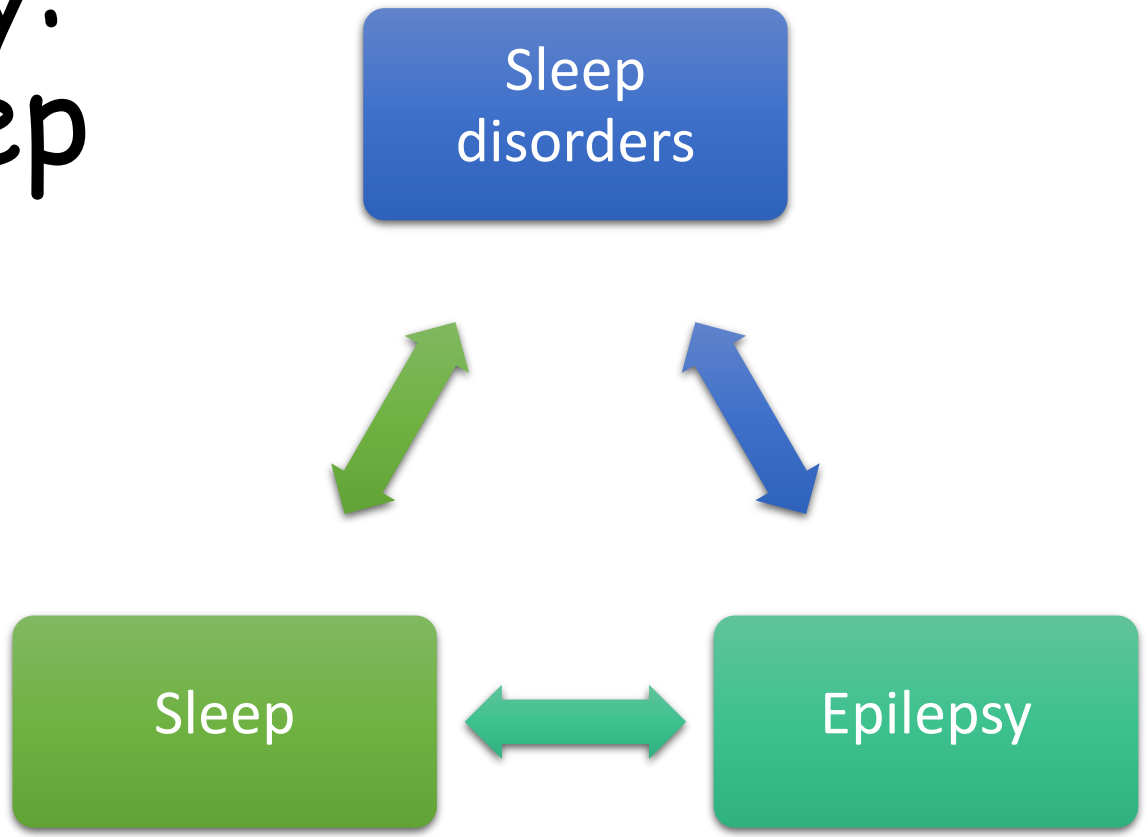
A complex interplay: Seizures and Sleep

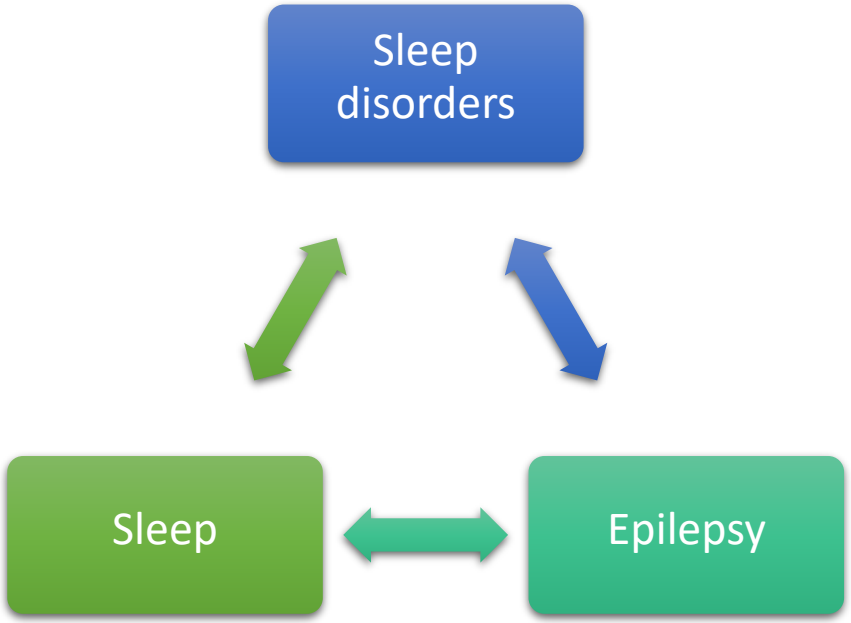
ณิรัชดา ทรัพย์อนันต์

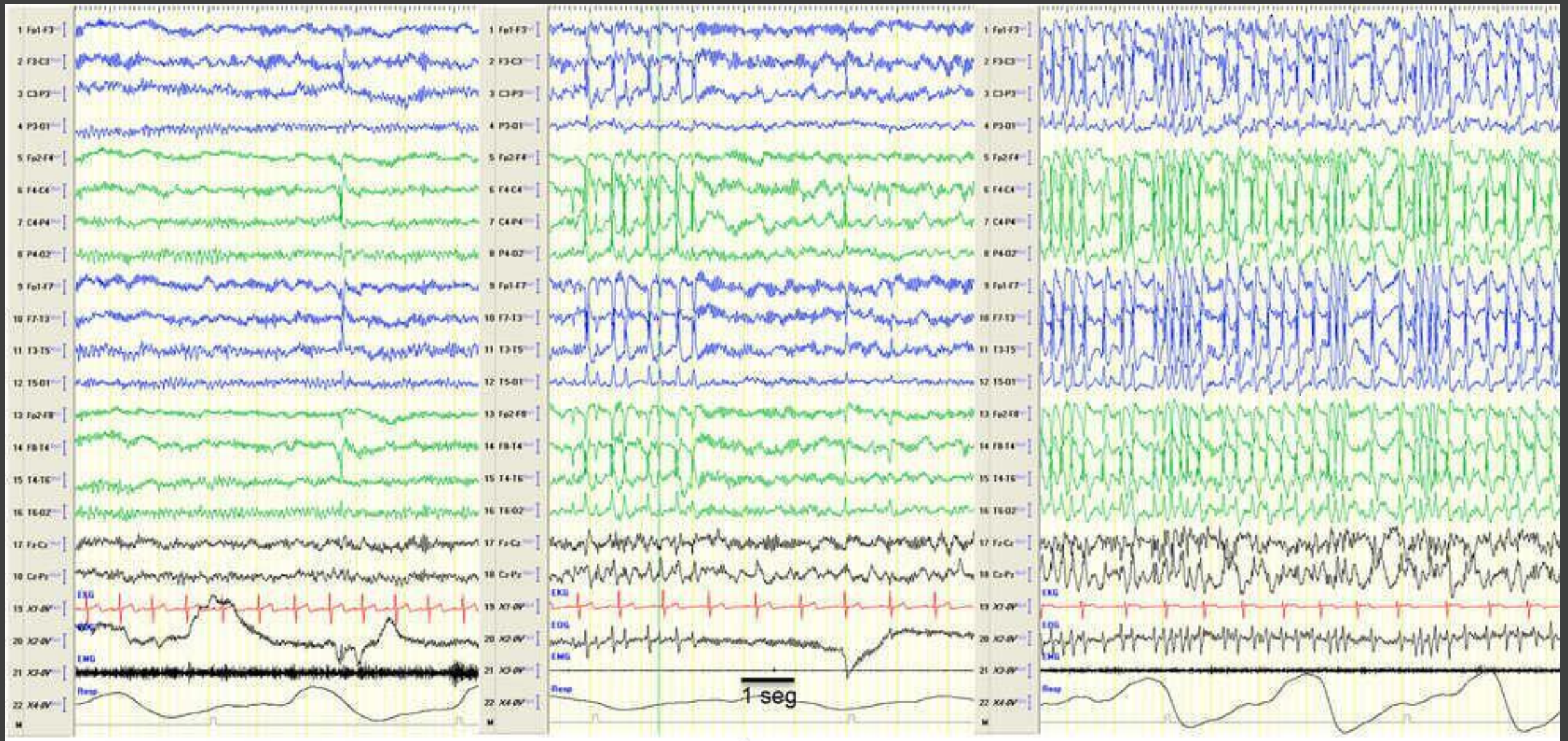
ลัลลิยา ธรรมประทานกุล



Sleep affecting distribution and frequency of interictal epileptiform discharges (IEDs)

- Total sleep deprivation activates interictal epileptiform discharges in 23% to 93% of patients with definite or suspected seizures.





a awake

b REM

c NREM

An Pediatr (Barc). 2019;91:18

Occurrence of IEDs in Different Sleep stages

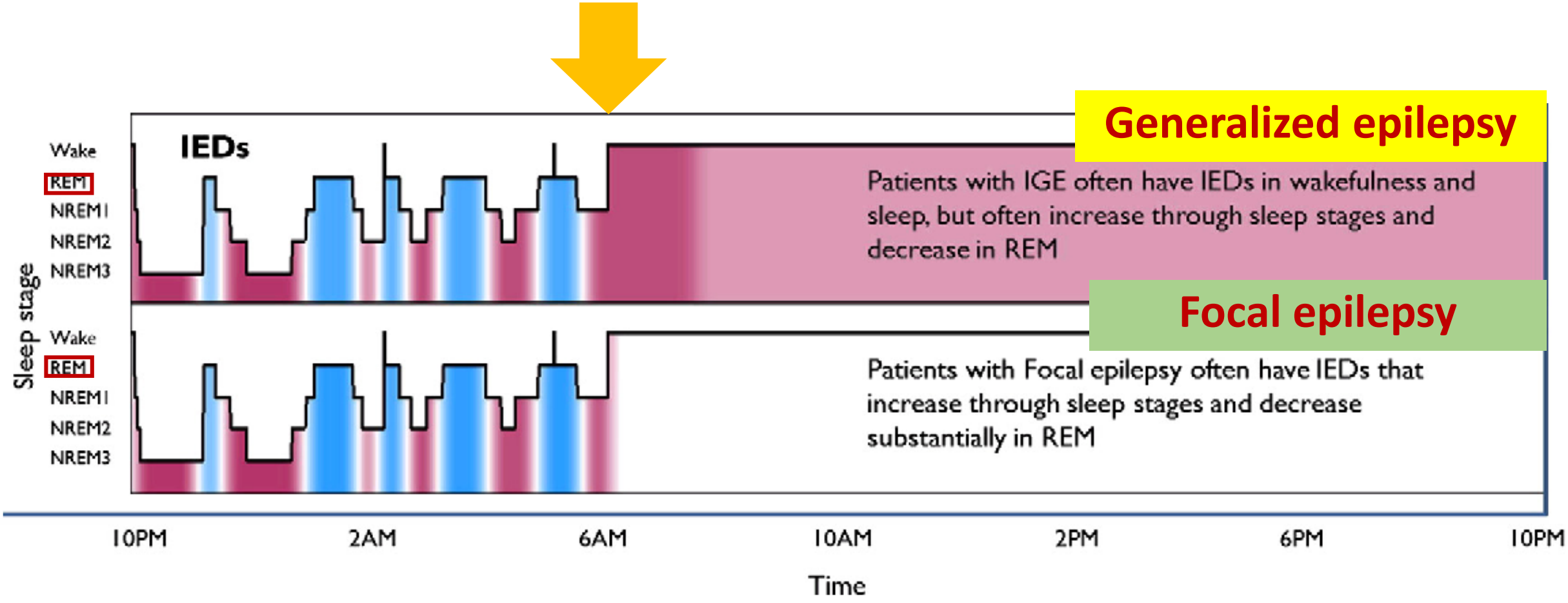
- Increased synchronization during NREM
  - “slow oscillation”
  - Enhance IED activity
  - Extensive and widespread IED
- Desynchronization during REM
  - “cholinergic modulation”
  - Enhance suppression effects of IEDs
  - Relatively suppressed and restricted IEDs
  - More accurate in localizing epileptogenic zone

Relative focal IED occurrence rate

(compared to REM)

- 1.11 times higher in W
- 1.75 times higher in N1
- 1.69 times higher in N2
- 2.46 times higher in N3

Sleep and Interictal Epileptiform Discharges (IEDs)



Badawy RA, Freestone DR, Lai A, et al. Epilepsy: ever-changing states of cortical excitability. Neuroscience 2012;222:89–99

Sleep Related Hypermotor Epilpesy (SHE)

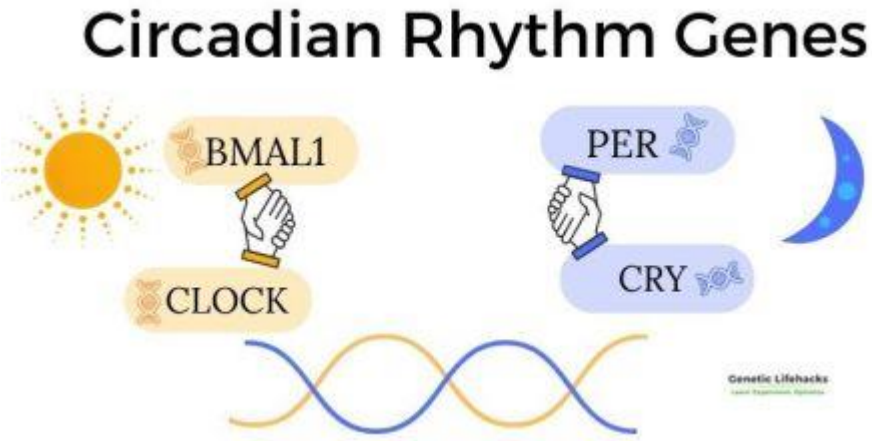
- Different intensity and durations ranging from paroxysmal arousals to nocturnal wandering that could occur in a single patient, during a single night

- Brief (< 2min)
- Abrupt onset and offset
- > 90% sleep (NREM) related
- Several episodes per night
- Level of awareness; not a crucial clinical signs

Mechanism of SHE

- Cholinergic hyperactivation
- Enhanced GABAergic function
- Cortical and subcortical networks involved in the mechanism of arousal → epileptogenesis of ADSHE
- Defects in CLOCK expression→ preferential occurrences of seizures during sleep
  - Circadian Locomotor Output Cycles Kaput
  - a transcription factor that regulated the circadian rhythm and the mTOR pathway

CLOCK gene

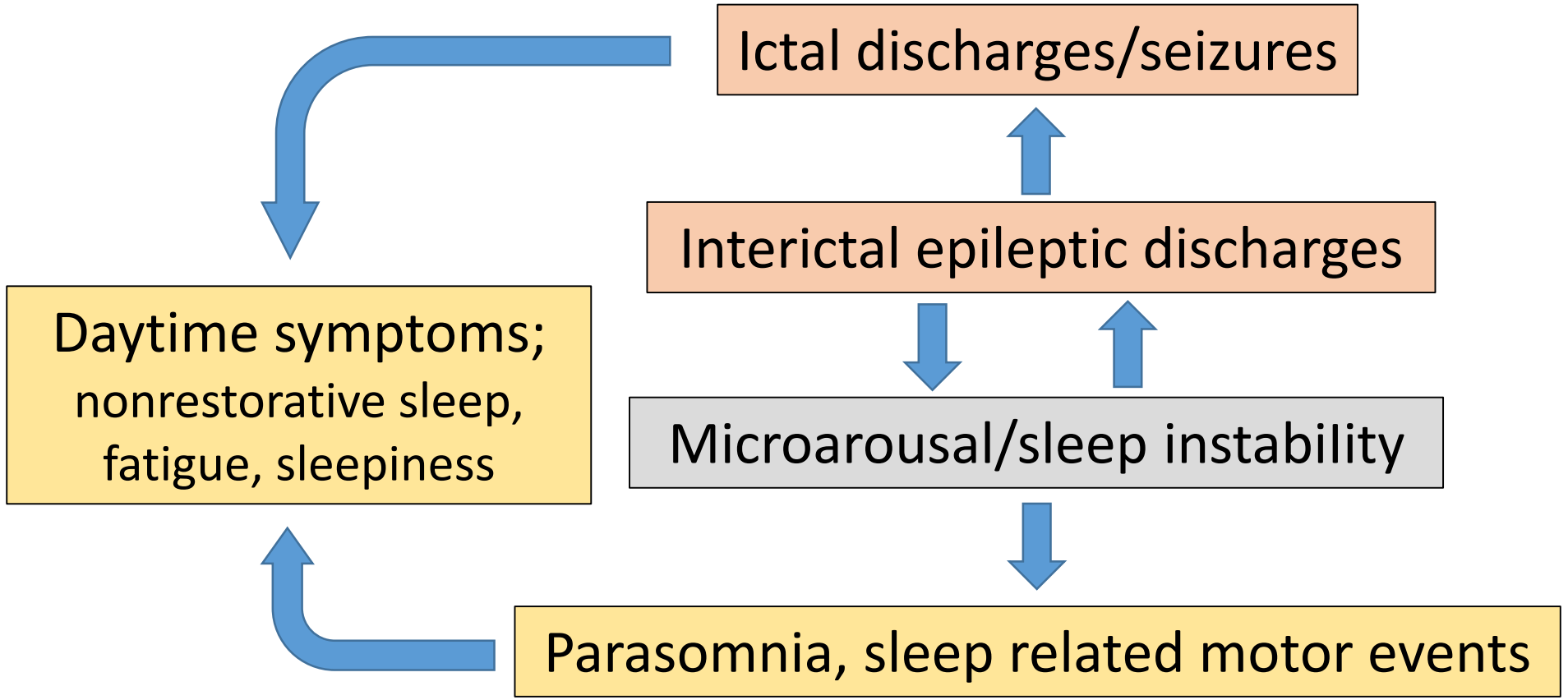


- Reduced expression of the CLOCK gene

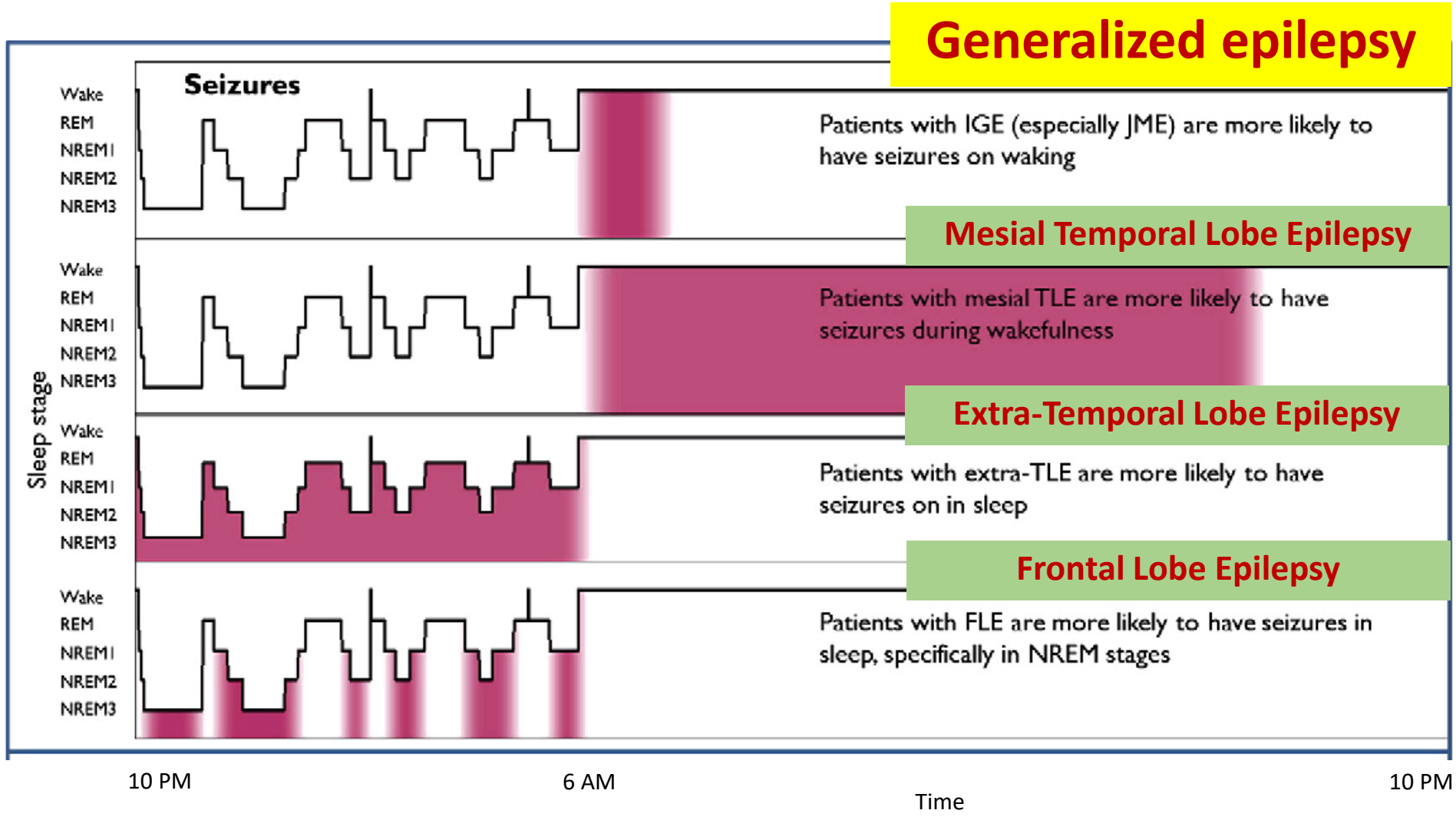
in epileptic brain specimens (FCD, TSC) compared to control tissue

- Mice with conditional deletion of CLOCK in the excitatory neurons, exhibits sleep-related seizures
- Disruption of the function of the core-clock genes may play an important role in the generation of focal epilepsy.
- However, most experiments are conducted on temporal lobe epilepsy; further studies on extratemporal lobe epilepsy are warranted.

Vicious loop of sleep-related epileptic discharges



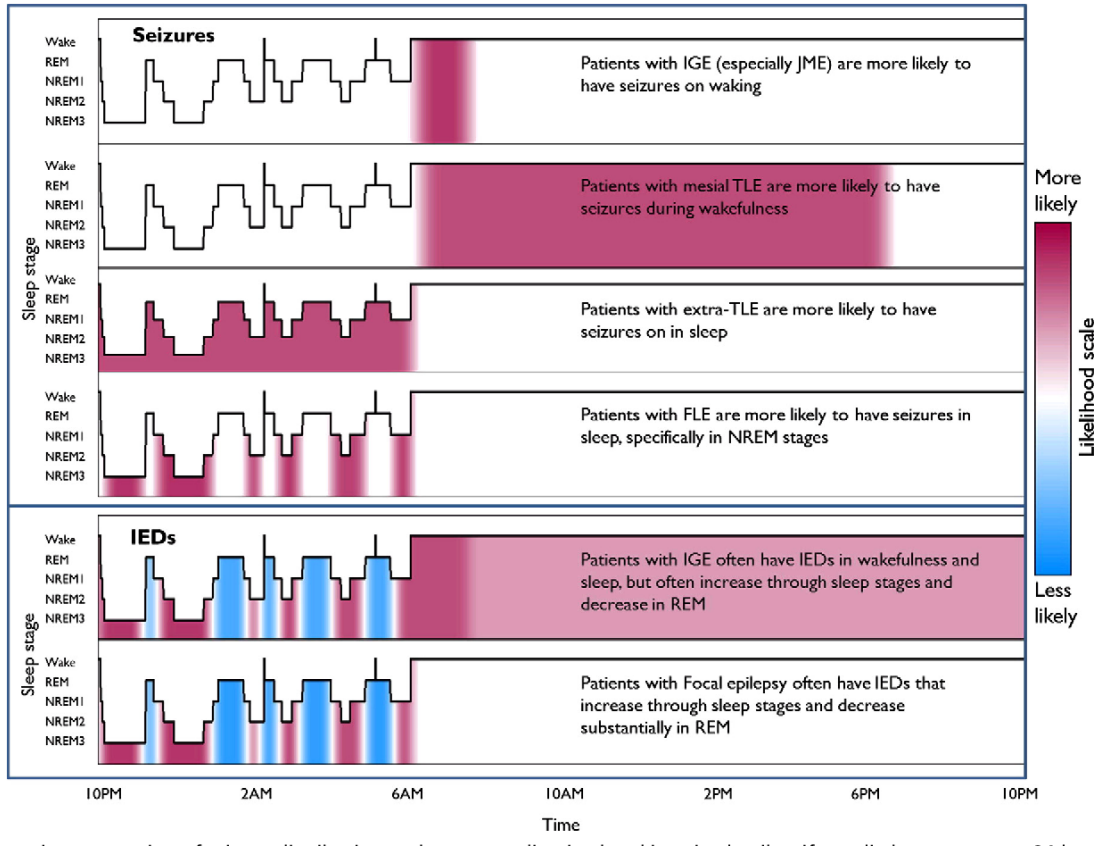
Sleep and Occurrence of Seizures



Badawy RA, Freestone DR, Lai A, et al. Epilepsy: ever-changing states of cortical excitability. Neuroscience 2012;222:89–99

Sleep affecting Epilepsy

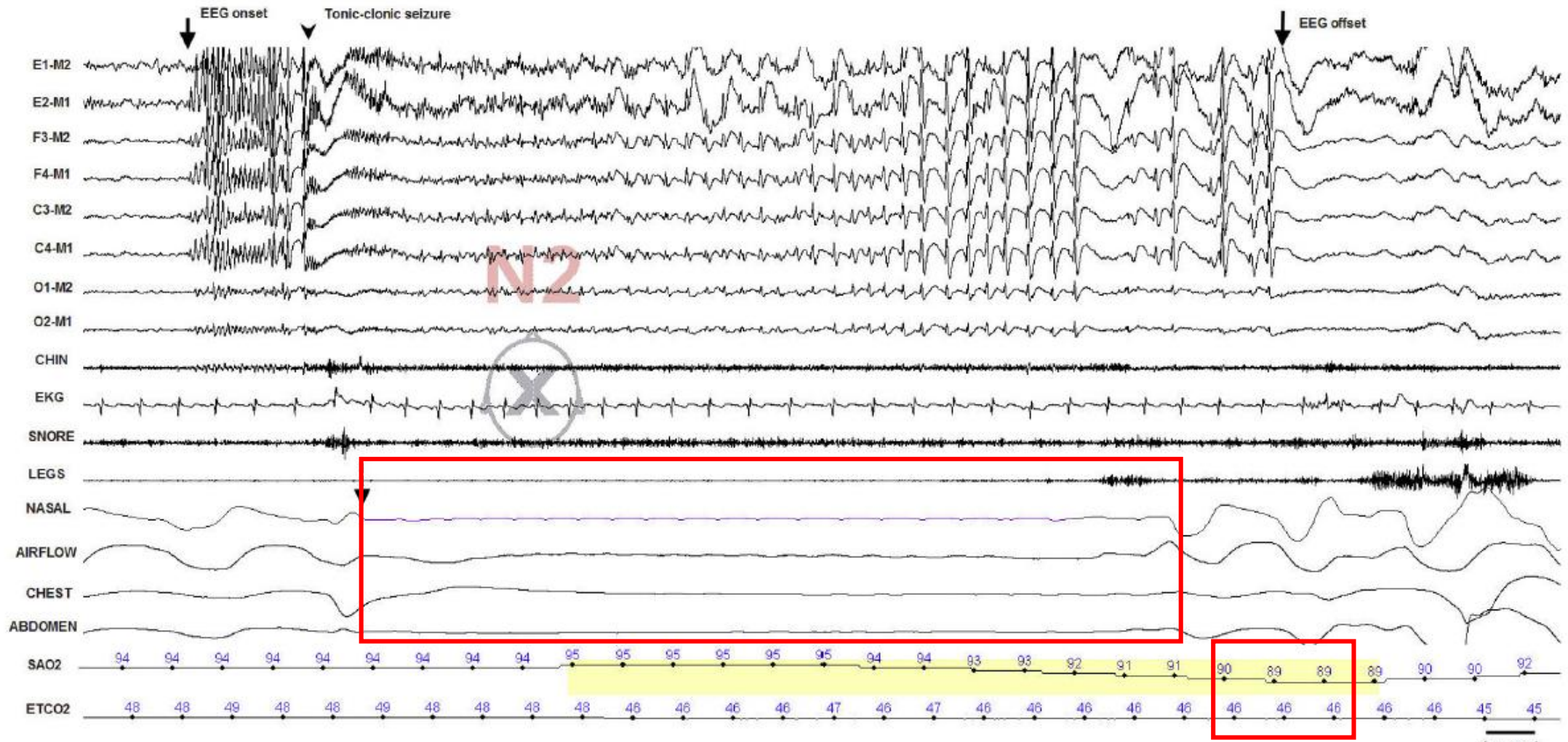
Sleep state modulates epileptic seizures and interictal epileptiform discharges.



“Turning blue” during seizures

- 1899: Hughlings Jackson report that respiratory arrest could occur in humans during temporal lobe seizures originating near the uncus, leading to patients “turning blue.”
- 1996: During vEEG: 10 of 17 PWE (59%) during 47 seizures developed apnea (mean, 24 s; longest, 64 s) and six had seizures with oxygen desaturations in the range of 55% to 83%.
- Apnea was typically central; obstructive events were observed in only 30%
- Electrical stimulation of the amygdala and hippocampal head in 3 patients undergoing stereotactic depth electrode evaluation: elicited central apnea during the expiratory phase (ie, inhibiting inspiration).

Seizure induced apnea and desaturation



Somboon T. Chest 2019

SUDEP
(Sudden Unexplained Death in EPilepsy)

- Risk of 1% per decade in patients with uncontrolled epilepsy.
- "Prone position, sleep, refractory epilepsy"
- Potential etiologic factors
  - cardiac arrhythmia, myocardial ischemia, arrhythmogenic medications
  - electrolyte disturbances
  - transmission of the epileptic activity via the autonomic nervous system to the heart
  - central or obstructive apnea

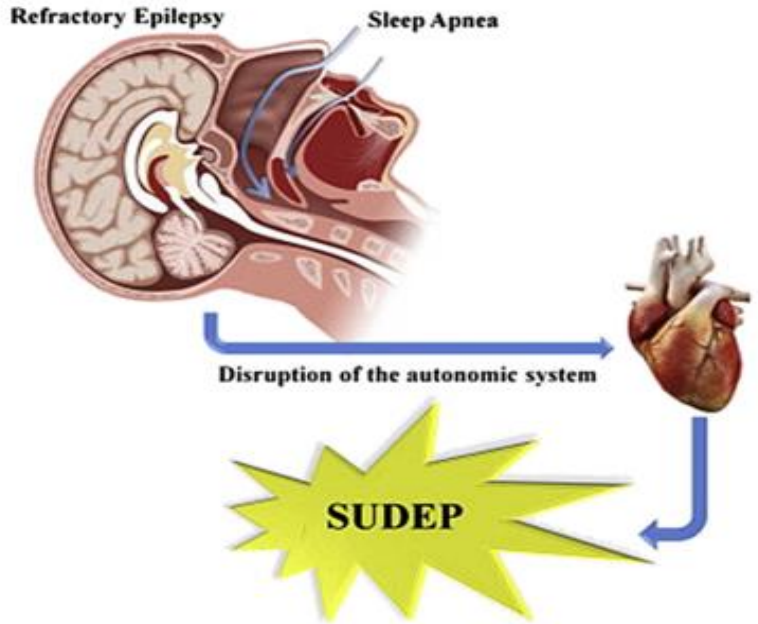
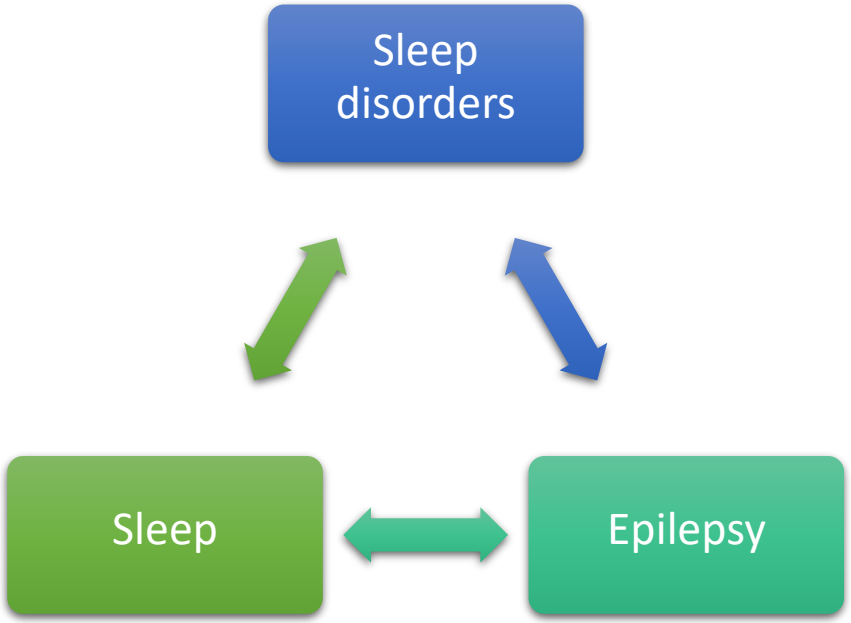


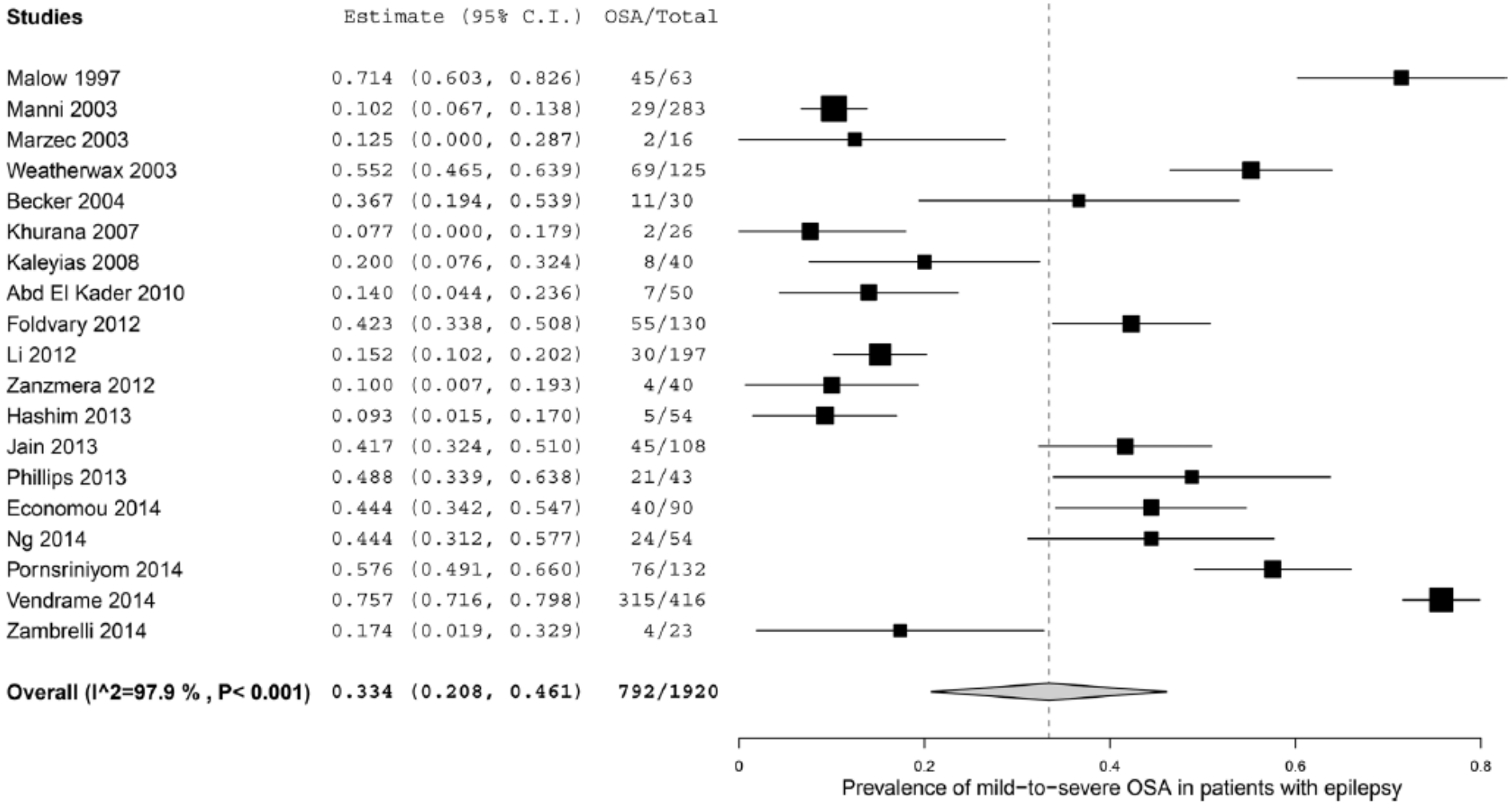
Fig. 1. SUDEP. The hypothesis that disruption of the autonomic system and SUDEP could be related to the occurrence of OSA in people with refractory epilepsy. (*From* Andersen ML, Tufik S, Cavalheiro EA, et al. Lights out! It is time for bed. Warning: obstructive sleep apnea increases risk of sudden death in people with epilepsy. Epilepsy Behav 2012;23(4):510–1.)

Sleep disorders affecting epilepsy

- Sleep disordered breathing
- Obstructive sleep apnea (OSA)
- Common comorbidity in epilepsy patients



Epilepsy patients more susceptible to OSA than healthy controls (OR 2.36; 95 % CI 1.33–4.18)



Lin Z,et al. Sleep Breath 2017

Subgroup	No. of studies	No. of OSA	No. of PWE	Prevalence (%, 95 % CI) or OR (95 % CI)	Heterogeneity		*P* value
					I^2, %	*P* value	
AHI >5	19	792	1920	33.4 (20.8–46.1)	97.9	<0.001	<0.001
AHI >15	10	86	856	9.7 (5.1–14.4)	86.1	<0.001	<0.001
AHI >30	9	34	726	4.4 (1.8–6.9)	66.1	0.003	<0.001
OR (PWE vs control)	5	94	650	2.36 (1.33–4.18)	29.6	0.22	0.003
Gender							
Male	11	229	536	44.8 (27.5–62.2)	95.6	<0.001	<0.001
Female	11	173	712	25.5 (15.4–35.0)	92.6	<0.001	<0.001
OR (M vs F)	11	402	1248	3.00 (2.25–3.99)	0	0.89	<0.001
Age							
Adults	11	641	1447	40.1 (21.6–58.6)	98.6	<0.001	<0.001
Children	4	66	204	26.3 (9.1–43.4)	88.2	<0.001	0.003
Seizure types							
Focal	7	160	560	32.2 (16.7–47.7)	94.6	<0.001	<0.001
Generalized	6	46	175	28.2 (12.1–44.3)	89.7	<0.001	<0.001
OR (focal vs G)	6	201	681	1.24 (0.80–1.92)	0	0.59	0.32
RE	4	37	188	19.5 (12.0–26.9)	37.2	0.18	<0.001
OR (RE vs CE)	3	41	287	1.61 (0.48–5.40)	22.3	0.27	0.43
AEDs							
≤1	7	116	481	23.2 (11.4–34.9)	90.6	<0.001	<0.001
≥2	8	103	410	25.6 (13.6–37.7)	89.4	<0.001	<0.001
OR (≤1 vs ≥2)	7	217	875	0.83 (0.59–1.17)	0	0.71	0.31
After VNS	4	26	70	43.1 (16.7–69.5)	83.6	<0.001	0.001
Sample size							
< 100	12	173	529	27.9 (15.8–40.0)	92.4	<0.001	0.06
≥100	7	619	1391	42.5 (19.3–65.8)	99.1	<0.001	0.001
Study quality							
<5	10	520	955	35.8 (17.1–54.6)	97.5	<0.001	<0.001
≥5	9	272	965	30.8 (16.9–44.6)	96.6	<0.001	<0.001

OSA obstructive sleep apnea, *PWE* patients with epilepsy, *CI* confidence interval, *OR* odds ratio, *AHI* apnoea/hypopnoea index, *M* male, *F* female, *G* generalized, *RE* refractory epilepsy, *CE* controlled epilepsy, *AEDs* antiepileptic drugs, *VNS* vagus nerve stimulation

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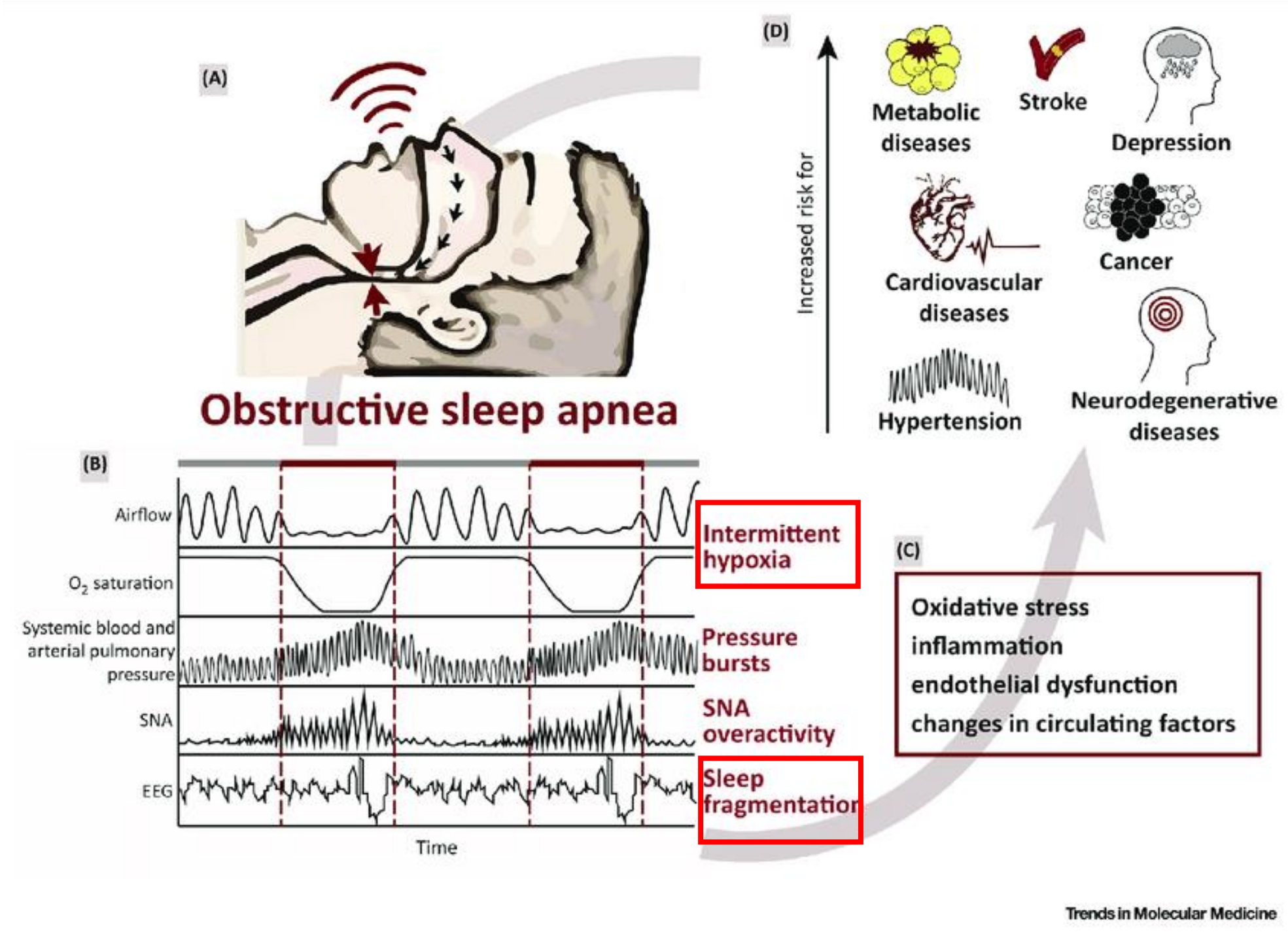
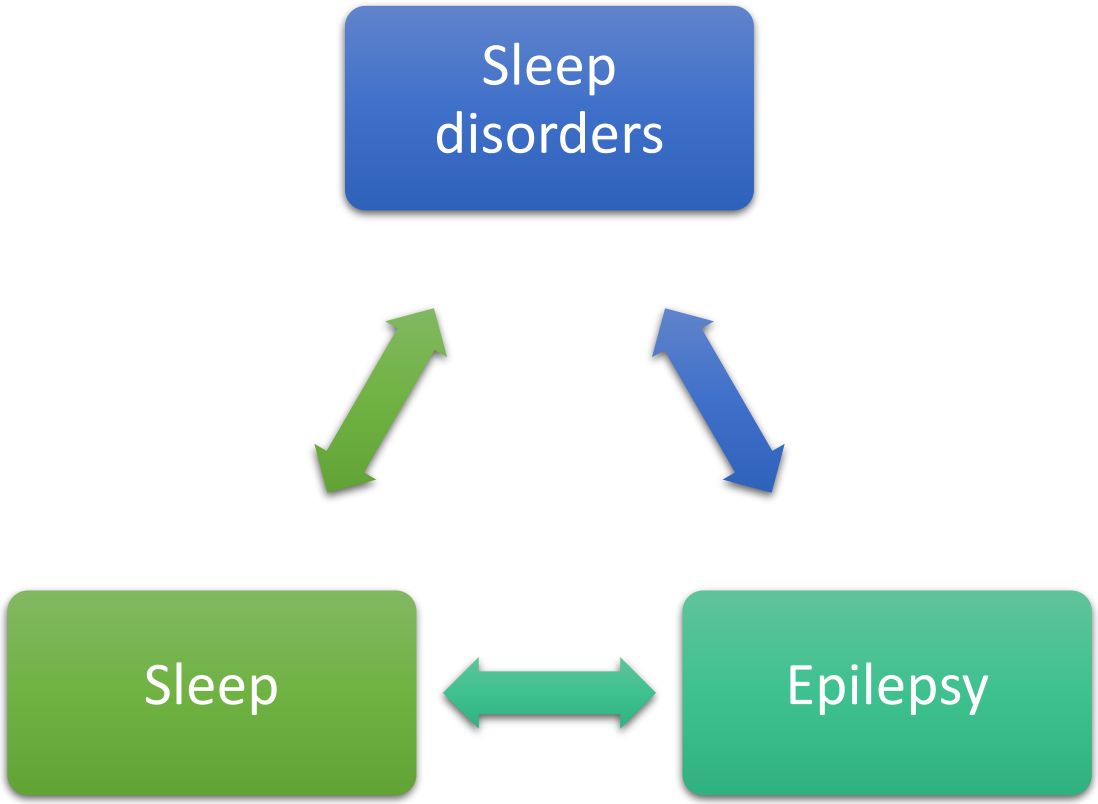

(A)
Obstructive sleep apnea
(B)
Airflow
O_2 saturation
Systemic blood and arterial pulmonary pressure
SNA
EEG
Time
Intermittent hypoxia
Pressure bursts
SNA overactivity
Sleep fragmentation
(C)
Oxidative stress
inflammation
endothelial dysfunction
changes in circulating factors
(D)
Increased risk for
Metabolic diseases
Stroke
Depression
Cardiovascular diseases
Cancer
Hypertension
Neurodegenerative diseases

Sleep disorders affecting epilepsy



OSA → hypoxia, frequent arousals, sleep fragmentation

Treatment of OSA improve seizure outcome

Poor seizure control
Daytime sleepiness
Poor tolerance to AEDs

Contents lists available at ScienceDirect

Epilepsy & Behavior

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Effect of positive airway pressure therapy on seizure control in patients with epilepsy and obstructive sleep apnea

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ABSTRACT

Previous studies suggest that treatment for obstructive sleep apnea (OSA) in patients with epilepsy can improve seizure control. We investigated the effect of positive airway pressure (PAP) therapy on seizures in adults with epilepsy referred to the Cleveland Clinic for polysomnography (PSG) from 1997 to 2010. Seizure outcome at baseline and 1 year later was compared in patients with no OSA (apnea–hypopnea index [AHI] <5), patients with PAP-treated OSA, and patients with untreated OSA. One hundred thirty-two subjects (age: 40.2 ± 13 (18–76) years, 65.4% female) were included. Seventy-six (57.6%) subjects had OSA; of these, 43 (56.6%) were on PAP therapy, and 33 (43.4%) were not on PAP therapy (either PAP-intolerant or refused therapy). Of the group with PAP-treated OSA, 83.7% were adherent (use ≥4 h/night at least 5 nights/week). The percentage of subjects with ≥50% seizure reduction and the mean percentage of seizure reduction were significantly greater in the group with PAP-treated OSA (73.9%; 58.5%) than in subjects with untreated OSA (14.3%; 17.0%). There were significantly more subjects with successful outcomes (with ≥50% seizure reduction or seizure-free at both baseline and follow-up) in the group with PAP-treated OSA (83.7%) than in the groups with no OSA (53.6%) and untreated OSA (39.4%). After adjusting for age, gender, body mass index, AHI, and epilepsy duration, we found that the odds of successful outcomes in subjects in the group with PAP-treated OSA were 9.9 and 3.91 times those of the groups with untreated OSA and no OSA, respectively. The group with PAP-treated OSA had 32.3 times the odds of having a ≥50% seizure reduction compared with the group with untreated OSA and 6.13 times compared with the group with no OSA. Positive airway pressure therapy appears to produce beneficial effects on seizures in adult patients with epilepsy and OSA.

Table 4

Seizure outcome in the groups with PAP-treated OSA, untreated OSA, and no OSA at follow-up.

	PAP Rx-OSA		Untreated-OSA		No OSA		p-Value
Total SZ/mo							0.11
Mean ± SD	43	1.46 ± 2.47	33	4.97 ± 8.96	56	2.69 ± 6.37	
Median [P25, P75]	43	0 [0, 2.25]	33	0.17 [0, 7]	56	0.04 [0, 1.62]	
Focal/dialeptic							0.060
Mean ± SD	43	1.05 ± 2.19	33	3.08 ± 6.17	56	1.53 ± 5	
Median [P25, P75]	43	0 [0, 0.5]	33	0.17 [0, 4]	56	0 [0, 0.17]	
Generalized motor							0.66
Mean ± SD	43	0.41 ± 1.01	33	1.89 ± 7.26	56	1.17 ± 3.61	
Median [P25, P75]	43	0 [0, 0]	33	0 [0, 0]	56	0 [0, 0]	
SZ change[a]							0.004
Mean ± SD	43	−3.2 ± 8.46	33	0.37 ± 1.08	56	−0.14 ± 1.81	
Median [P25, P75]	43	0 [−2, 0]	33	0 [0, 0]	56	0 [−0.29, 0]	
SZ change − SF							0.40
Mean ± SD	20	0.10 ± 0.45	12	0.17 ± 0.44	19	0.04 ± 0.09	
Median [P25, P75]	20	0 [0, 0]	12	0 [0, 0]	19	0 [0, 0]	
SZ change − NSF[b]							<0.001
Mean ± SD	23	−6.07 ± 10.86	21	0.48 ± 1.31	37	−0.23 ± 2.23	
Median [P25, P75]	23	−2 [−2.75, −0.17]	21	0 [0, 0]	37	−0.17 [−0.7, 0]	
% decrease[c]							0.004
Mean ± SD	23	58.52 ± 50.36	21	−16.97 ± 120.42	37	18.5 ± 134.17	
Median [P25, P75]	23	72.7 [45.8, 100]	21	0 [0, 0]	37	35 [0, 100]	
Responder, %[d]							<0.001[C]
Yes	17	73.91	3	14.29	15	40.54	
No	6	26.09	18	85.71	22	59.46	
Successful outcome[e]							<0.001[C]
Yes	36	83.72	13	39.39	30	53.57	
No	7	16.28	20	60.61	26	46.43	

Mean ± SD, median [P25, P75] for continuous variables, otherwise percentage.

Abbreviations: SZ = seizure; mo = month; SF = seizure-free at baseline; NSF = not seizure-free at baseline; responder ≥ 50% seizure reduction; C = Pearson's chi-squared test. Kruskal–Wallis rank sum test unless otherwise indicated.

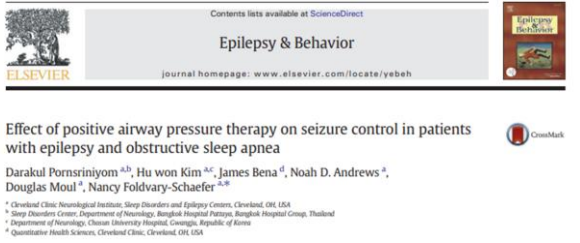
[a] Using a 0.017 significance level, the group with PAP-treated OSA differs from the group with untreated OSA ($p < 0.001$); the groups with PAP-treated OSA and no OSA do not differ ($p = 0.057$); the groups with untreated OSA and no OSA do not differ ($p = 0.060$).

[b] The group with PAP-Treated OSA differs from the other two groups ($p < 0.001$ vs. untreated OSA and $p = 0.002$ vs. no OSA); the groups with untreated OSA and no OSA do not differ ($p = 0.020$).

[c] The group with PAP-treated OSA differs from the group with untreated OSA ($p < 0.001$); the groups with PAP-treated OSA and no OSA do not differ ($p = 0.17$); the groups with untreated OSA and no OSA do not differ ($p = 0.046$).

[d] The groups with PAP-treated OSA and untreated OSA differ ($p < 0.001$). The groups with PAP-treated OSA and no OSA do not differ ($p = 0.024$); the groups with untreated OSA and no OSA do not differ ($p = 0.075$).

[e] The groups with PAP-treated OSA and untreated OSA differ ($p < 0.001$); the groups with PAP-treated OSA and no OSA differ ($p = 0.003$); the groups with untreated OSA and no OSA do not differ ($p = 0.28$).



Effect of positive airway pressure therapy on seizure control in patients with epilepsy and obstructive sleep apnea

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PAP-treated OSA had 32.3 times the odds of having a ≥50% seizure reduction compared with the group with untreated OSA

Pornsiriniyom D, et al. Epilepsy & Behavior 2014

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SLEEP BREATHING PHYSIOLOGY AND DISORDERS • ORIGINAL ARTICLE

Obstructive sleep apnoea in patients with epilepsy: a meta-analysis

Zhang Lin[1] · Qi Si[2] · Zou Xiaoyi[1]

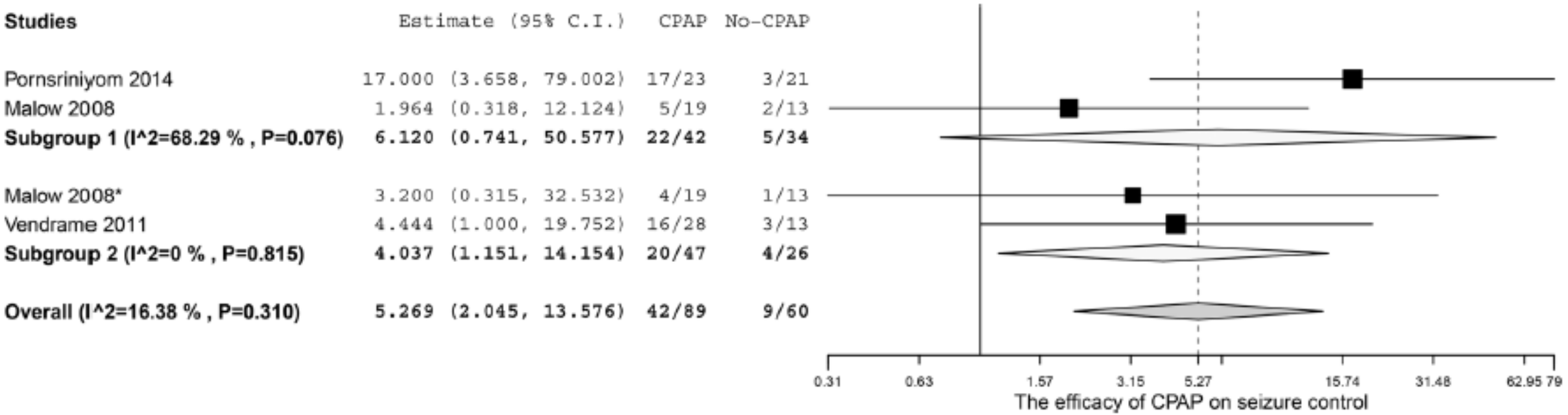

Fig. 3 The efficacy of CPAP on seizure control. Subgroup 1 = 50 % seizure reduction rate. Subgroup 2 = seizure-free rate. *CPAP* continuous positive airway pressure

Patients treated by CPAP have an increase in successful outcomes as compared to the untreated patients (OR 5.26; 95 % CI 2.04–13.5; P < 0.001)

Lin Z,et al. Sleep Breath 2017

Epilepsy Research 129 (2017) 37–40

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Short communication

The STOP-BANG questionnaire improves the detection of epilepsy patients at risk for obstructive sleep apnea

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ABSTRACT

Patients with epilepsy and obstructive sleep apnea (OSA) are at risk for worsened seizure control and quality of life. We performed a quality improvement project, evaluating for improvements in the screening of OSA in epilepsy patients using the STOP-BANG questionnaire. The electronic medical records of patients seen in our epilepsy clinic were screened for 4 months prior to the intervention. We subsequently implemented the STOP-BANG questionnaire for 3 months. Only 22/664 patients (3.3%) had their sleeping

on the STOP-BANG questionnaire saw sleep medicine; 11 (91.7%) were referred for polysomnography (PSG). Of the 10 patients who underwent PSG, 9 (90%) were diagnosed with OSA and offered treatment with continuous positive airway pressure (CPAP).

PPV of the STOP-BANG questionnaire
81.8% in epilepsy clinic population.

Snoring
Tiredness
Observed
Blood **P**ressure
BMI
Age
Neck circumference
Gender

Table 1
The Snoring, Tiredness during daytime, Observed apnea, high blood Pressure, Body mass index, Age, Neck circumference, and Gender (STOP-BANG) questionnaire.

1. Snoring
Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?
Yes, No
2. Tired
Do you often feel tired, fatigued, or sleepy during daytime?
Yes, No
3. Observed
Has anyone observed you stop breathing during your sleep?
Yes, No
4. Blood pressure
Do you have or are you being treated for high blood pressure?
Yes, No
5. BMI
BMI more than 35 kg/m^2?
Yes, No
6. Age
Age over 50 yr old?
Yes, No
7. Neck circumference
Neck circumference greater than 40 cm?
Yes, No
8. Gender
Gender male?
Yes, No

Epilepsia, 49(8):1457–1459, 2008
doi: 10.1111/j.1528-1167.2008.01677.x

BRIEF COMMUNICATION

Resolution of obstructive sleep apnea with epilepsy surgery? Expanding the relationship between sleep and epilepsy

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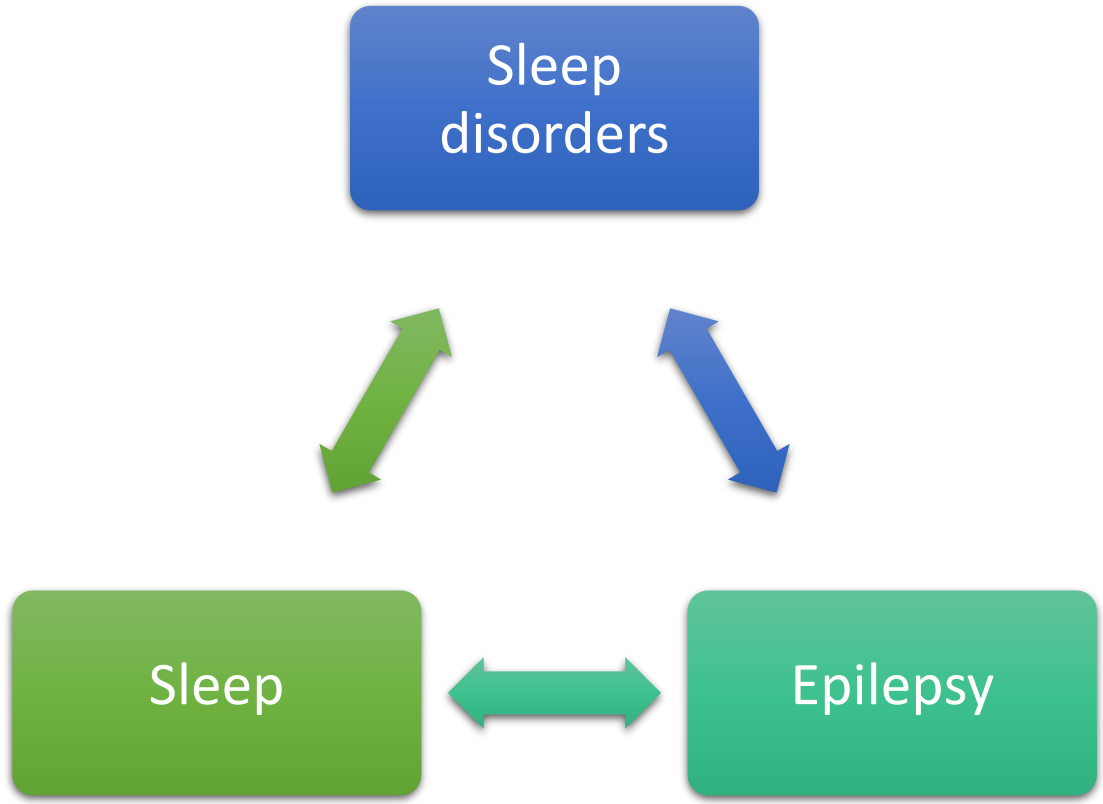
Table 1. Polysomnographic parameters

	Preoperative PSG	Postoperative PSG
Body mass index (kg/m^2)	21.2	23.1
Recording time (min)	374.1	491.0
Total sleep time (min)	367.4	367.0
Supine sleep time (%)	100.0	97.0
Sleep efficiency (%)	98.2	74.7
Sleep latency (min)	0.2	111.0
REM latency (min)	301.5	76.0
Stage 1 (%)	7.3	3.8
Stage 2 (%)	37.3	53.0
Stages 3–4 (%)	52.3	22.1
Stage REM (%)	3.1	21.1
Arousal index	11.3	14.4
AHI	24.3	1.1
REM AHI	47.1	2.3
Mean/Minimum SaO_2	92/62	98/91
% TST with $SaO_2 < 90\%$	30.6	0
Periodic limb movement index	1.0	15.2
Periodic limb movement arousal index	0	2.9
No. of recorded seizures	1	0
Spike rate[a]	867	27

[a] Interictal epileptiform discharges per hour, quantified over the first sleep cycle.

Foldvary-Schafer N, et al. Epilepsia 2008

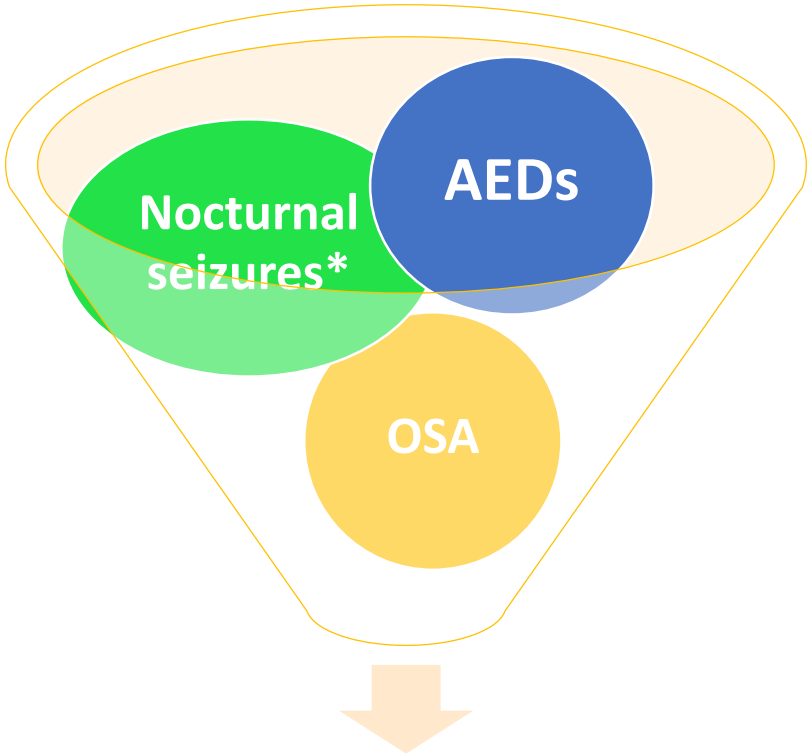
Epilepsy affecting Sleep



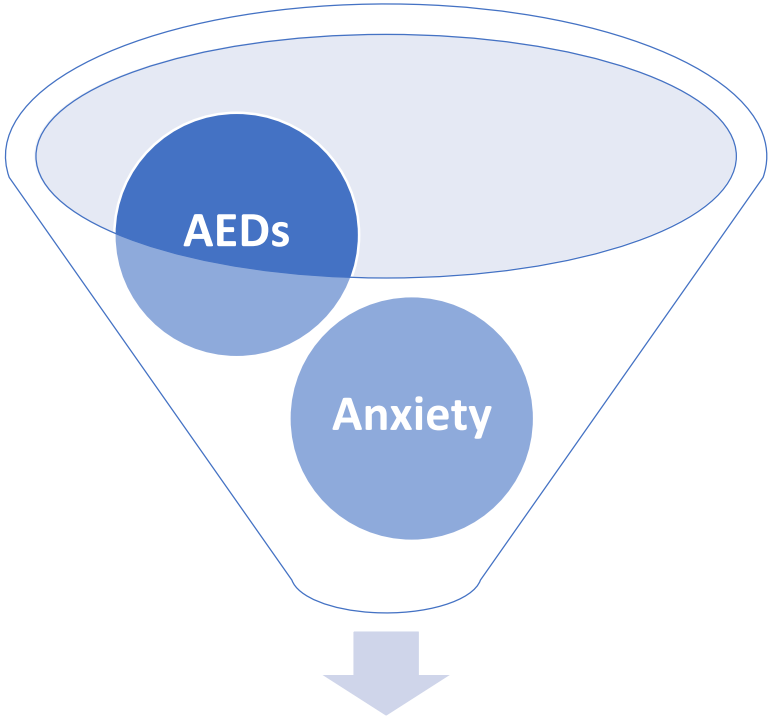
Insomnia
Daytime sleepiness
OSA

Anxiety
Nocturnal seizures
Epilepsy treatment
Anticonvulsants: hypotonia, wt gain
Vagal nerve stimulator

Epilepsy affecting Sleep



Excessive daytime sleepiness



Insomnia

* Sleep fragmentation from increased arousal

Anticonvulsants affecting Sleep

Sleep architecture change

Daytime sleepiness*

Insomnia: Lamotrigine, levetiraceam

Increased OSA

- Increased airway floppiness : benzodiazepine

- Excessive wt gain: valproate

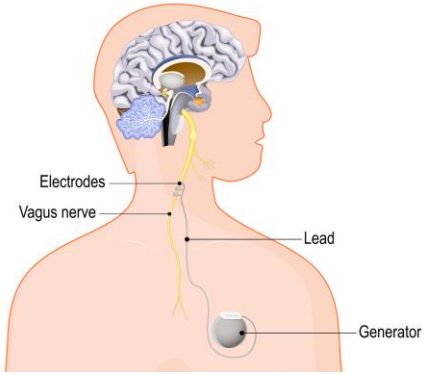
Increased RLS: Topiramate, Zonisamide

Table 1
Contribution of AED on sleep disorders and sleep architecture

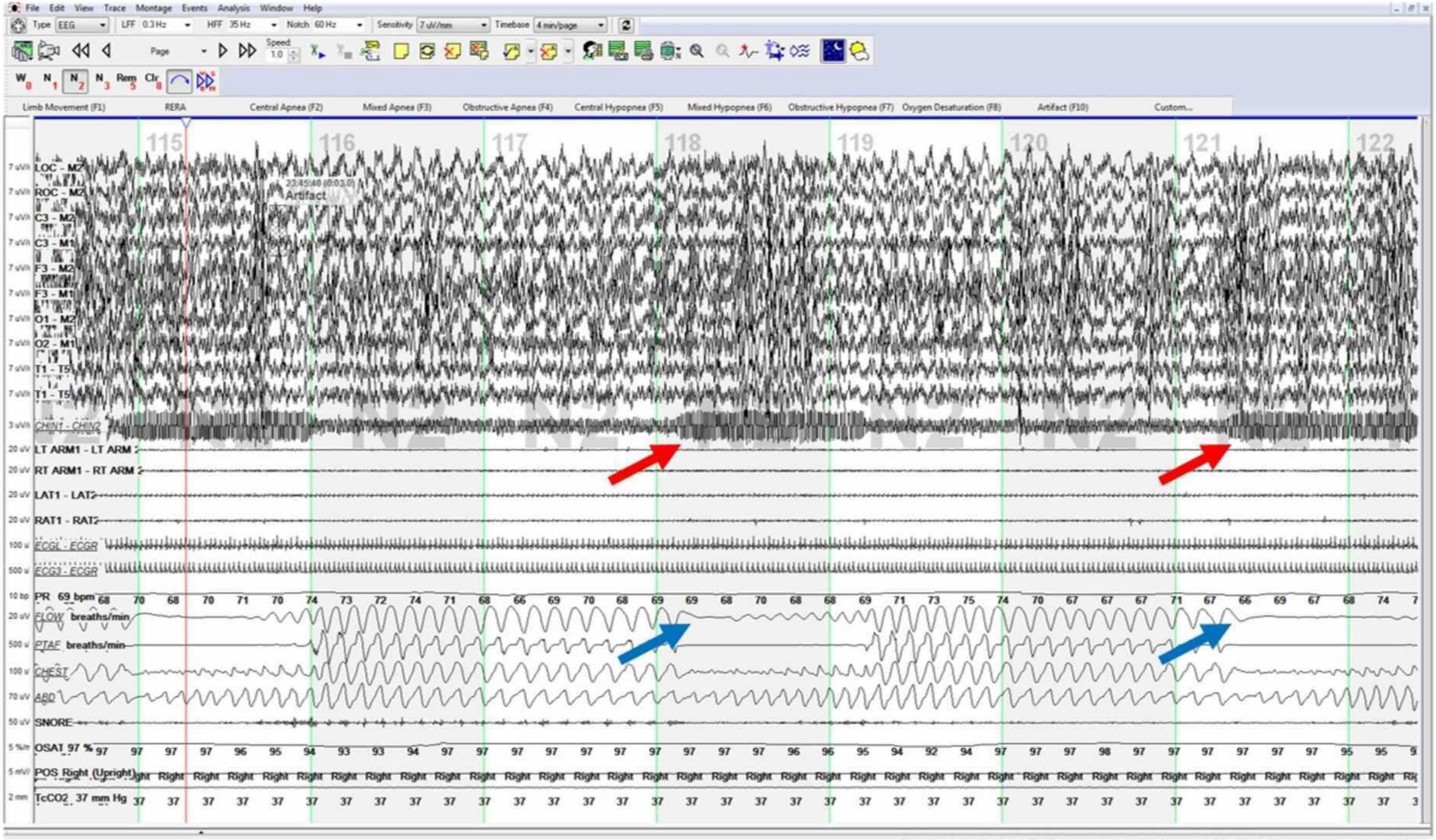
	Sleep Disorders		Sleep Architecture	
AED	**Positive Effects**	**Negative Effects**	**Positive Effects**	**Negative Effects**
Phenobarbital	Insomnia	Obstructive sleep apnea	↓SL	↓REM
Benzodiazepines	Insomnia, Willis Ekbom disease, REM sleep disorder	Obstructive sleep apnea	↓SL, ↓arousals, ↓CAP rate	↓REM ↓N3
Carbamazepine	Willis Ekbom disease	None	None	↓REM, ↑Sleep stage shifts
Valproate	Willis Ekbom disease	Obstructive sleep apnea	Sometimes no effect	↑1N Reduction in REM
Gabapentin	Willis Ekbom disease, insomnia	Obstructive sleep apnea	↑N3, ↓arousals ↑sleep efficiency	None
Lamotrigine	Consolidating sleep reducing arousals stage shifts	Insomnia; REM sleep behavior disorder	↓Sleep stage shifts, ↓arousals, ↑REM	↓N3(possible)
Levetiracetam	Willis Ekbom disease (case reports)	Insomnia	↑N3 Stage shifts and wake after sleep onset were significantly decreased	None
Pregabalin	Willis Ekbom disease, insomnia, daytime attention	Obstructive sleep apnea	↑N3, ↑REM, ↓arousals	None
Topiramate	Weight loss, Obstructive sleep apnea	Willis Ekbom disease	No changes	No changes
Zonisamide	Obstructive sleep apnea	Willis Ekbom disease	No changes	No changes

Abbreviations: AED, anti epileptic drugs; CAP, cyclic alternating pattern (A marker of sleep insta-

VNS affecting Sleep Disordered Breathing

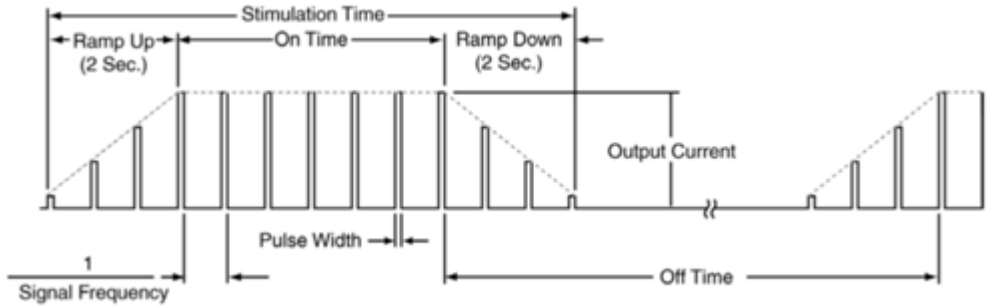


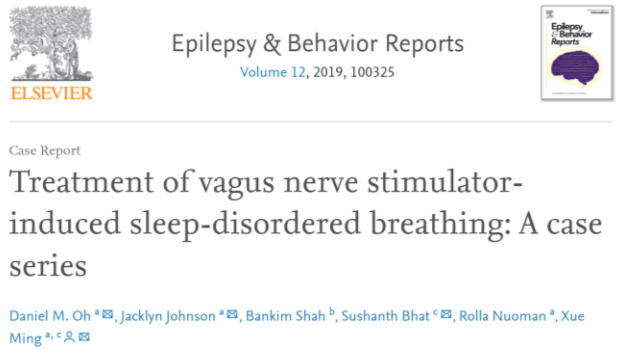
- Experimental stimulation of the vagus nerve in humans: → partial/complete inhibition of inspiration, prolongation of expiratory time, and modest changes inarterial pressure and bradycardia
- Patients with VNS can have central apneas, obstructive hypopneas, and obstructive apneas.
- Prevalence of OSA increased from 17% to 65% after VNS
- Awake endoscopic laryngoscopy postimplantation showed left vocal cord adduction during the stimulation ON phase
- Suggesting that reduction of the glottal space or lack of coordination between inspiration and the glottal aperture may have a role in OSA after VNS therapy.


File Edit View Trace Montage Events Analysis Window Help
Limb Movement (F1)
RERA
Central Apnea (F2)
Mixed Apnea (F3)
Obstructive Apnea (F4)
Central Hypopnea (F5)
Mixed Hypopnea (F6)
Obstructive Hypopnea (F7)
Oxygen Desaturation (F8)
Artifact (F10)
Custom...
115
116
117
118
119
120
121
122
LOC - M2
ROC - M2
C3 - M2
C3 - M1
F3 - M2
F3 - M1
O1 - M2
O2 - M1
T1 - T5
T1 - T5
CHIN1 - CHIN2
LT ARM1 - LT ARM 2
RT ARM1 - RT ARM 2
LAT1 - LAT2
RAT1 - RAT2
ECGL - ECGR
ECG3 - ECGR
PR 69 bpm
FLOW breaths/min
PTAF breaths/min
CHEST
ABD
SNORE
OSAT 97 %
POS Right (Upright)
TcCO2 37 mm Hg

VNS affecting Sleep Apnea: Management

- Pre VNS implantion OSA; improved with PAP treatment
- Post VNS implantation OSA; decreased stimulation of VNS
- Respiratory events during VNS ON time can usually be reduced or eliminated by
  - lengthening the duration of OFF time (increasing the cycling time to 300 s)
  - reducing the stimulation intensity from 30 to 20 Hz, and if needed 10 Hz.
- **Screening for OSA prior to VNS implantation?**

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Epilepsy & Behavior Reports
Volume 12, 2019, 100325
Case Report

Treatment of vagus nerve stimulator-induced sleep-disordered breathing: A case series

Daniel M. Oh [a], Jacklyn Johnson [a], Bankim Shah [b], Sushanth Bhat [c], Rolla Nuoman [a], Xue Ming [a, c]

Take home message

- Complex interplay epilepsy, sleep and sleep disorder
- Better sleep, better seizure outcome
- Screen for sleep problems particularly OSA in epilepsy patients