Obstructive sleep apnea in epilepsy: a preliminary Egyptian study

Hala A. Shaheen

Prof and head of neurology department, Faculty of medicine, Fayoum university

A preliminary Egyptian study

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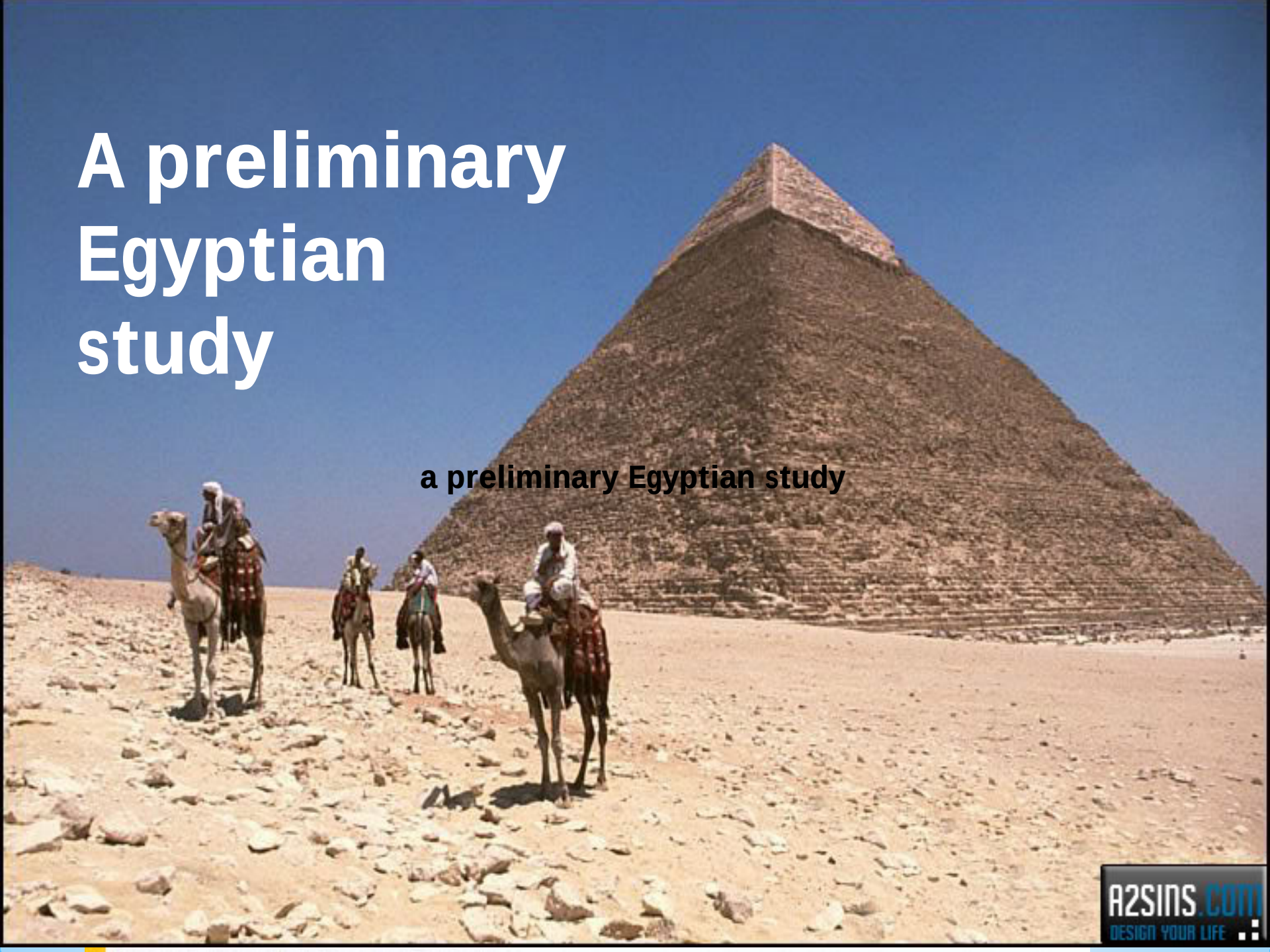


Hala A. Shaheen , Ann A. Abd El-Kader , Amira M. El Gohary , Neveen M. El-Fayoumy , Lamia M. Afifi
Faculty of medicine, Egypt

Epilepsy and obstructive sleep apnea are two common disorders that coexist

Introduction

Epilepsy and obstructive sleep apnea profoundly exacerbate each other's [Peppard etal 2000]

Introduction

Several mechanisms may contribute to the increased incidence of OSA in patients with epilepsy,

Introduction

The adverse effect of antiepileptic drugs

CNS depression,

Effect on upper airway tone,

Weight gain

Introduction

Reduced physical activity of patients with epilepsy

Also seizures prolonged REM where OSA became worse [Sharafkhaneh A, etal 2005)

Introduction

Appearance of OSA symptoms coincided with clear increase in seizure frequency or an emergence of status in patients with epilepsy [Nieto F, etal 2011]

Introduction

A variety of seizure-provoking mechanisms in OSA patients are postulated

Introduction

Introduction

Obstructive sleep apnea (OSA) is due to intermittent blockage of the upper airway with Consequent reduction or cessation of airflow during sleep [Frucht M, etal 2000]

It leads to

Cerebral hypoxemia,

cortical arousal,

Sleep fragmentation, and

Introduction

Decrease time spent in deep sleep, and

Sleep deprivation

[Wyler A, Weymuller E 2012].

Introduction

OSA with subsequent significant morbidity such as

Introduction

Morbidity

increased risk of cardiac, respiratory, and metabolic consequences [Parra etal 2010]

Introduction

Introduction

Mortality

OSA postulated as a cause of sudden unexpected death in patients with epilepsy

[Malow B, etal 2008]

To make matters worse, obstructive sleep apnea is notoriously underdiagnosed particularly in patients with epilepsy [Decary A, etal 2000]

Introduction

The extent and clinical relevance of the association between epilepsy and sleep apnea are not previously studied in Egypt

Introduction

What we wanted to know was the

This study aimed to look for the frequency of sleep apnea in Egyptian patients with epilepsy in comparison to a control group.

Aim of work

Try to find out sleep apnea relation to

Clinical data, Sleep complaint

Polysomnographic findings

Aim of work

The identification and treatment of OSA may have far-reaching consequences in improving patient's quality of life [Trupp R, etal 2004]

Introduction

Introduction

Continuous positive airway pressure (CPAP) treatment was found to improve seizure control in those patients [Gami A, etal 2005]


Patients and
methods:


Patients

Patients and methods:

This is a case control study of 26 patients with epilepsy

and

26 normal controls Patients

Inclusion criteria

Children with idiopathic epilepsy

Patients and methods:


Patients and methods:
Exclusion criteria

Patients with any neurological disease apart from epilepsy; patients with psychiatric illness; patients with history of hypnotics or sedatives intake; or those with liver or kidney failure were excluded from the study.

The patients were divided into two subgroups according to apnea/hypopnea index (AHI):

Group (1) patients without OSA

group (2) patients with OSA

Patients and methods:

Healthy children their age and sex matched to our patients were also studied

Patients and methods: control group

Methods

Clinical assessment

Epilepsy history and detailed neurological examination

Epilepsy history: Neurologist interviewed patients and one of their close relative to inquire about age of onset of epilepsy and disease duration

Methods

Clinical assessment

Seizures characteristics

such as

Seizure frequency,

status epilepticus,

Circadian rhythm, and

Dosage of medications

Methods

Clinical assessment

Type of epilepsy was determined according to International League against Epilepsy classification

They were asked about total nocturnal sleep time, repeated awakenings during sleep, history of excessive daytime sleepiness, insomnia, and nocturnal snoring

Methods
Sleep history

Methods
Electrophysiological assessment

Electroencephalogram(EEG) was done for all patients using a Schwarzer GmbH medical diagnostic equipment

Video EEG monitoring was done for all patients using a Schwarzer GmbH medical diagnostic equipment and a digital video-camera Panasonic AG6040)

Methods
Electrophysiological assessment

Methods Electrophysiological assessment

Overnight polysomnography was performed for both patients and controls using a Schwarzer. Epos 32 GmbH, medical diagnostic polysomnogram, Germany

The software used was Somnologica version 3.1

Total sleep time (TST),

Sleep efficiency,

Percentage of each stage

Number of awakenings,

Arousal index and

leg movements

Methods

The polysomnographic parameters were :

Methods
Polysomnographic assessment

Apnea events were counted according to the criteria established by the American Academy of Sleep Medicine

Statistical package for social science (SPSS) version 15 were used for data management. Chi square test was used for comparison between qualitative variables groups

Statistical analysis:

Independent sample T test was used for normally distributed quantitative variables as comparing age mean, seizure duration, and sleep efficiency among patient subgroups.

Statistical analysis:

Statistical analysis:

Mann–Whitney test were used for non normally distributed quantitative variables as age of onset, number of awakening, sleep latency, PLM, and apnea and hypopnea index.

Pearson's correlation coefficient was calculated for the association between the apnea/hypopnea index and clinical and polysomnographic data.

Statistical analysis:

Statistical analysis:

The logistic regression analysis was done to test for significant predictors of OSA among the patients.

P value 0.05 was considered significant

Results

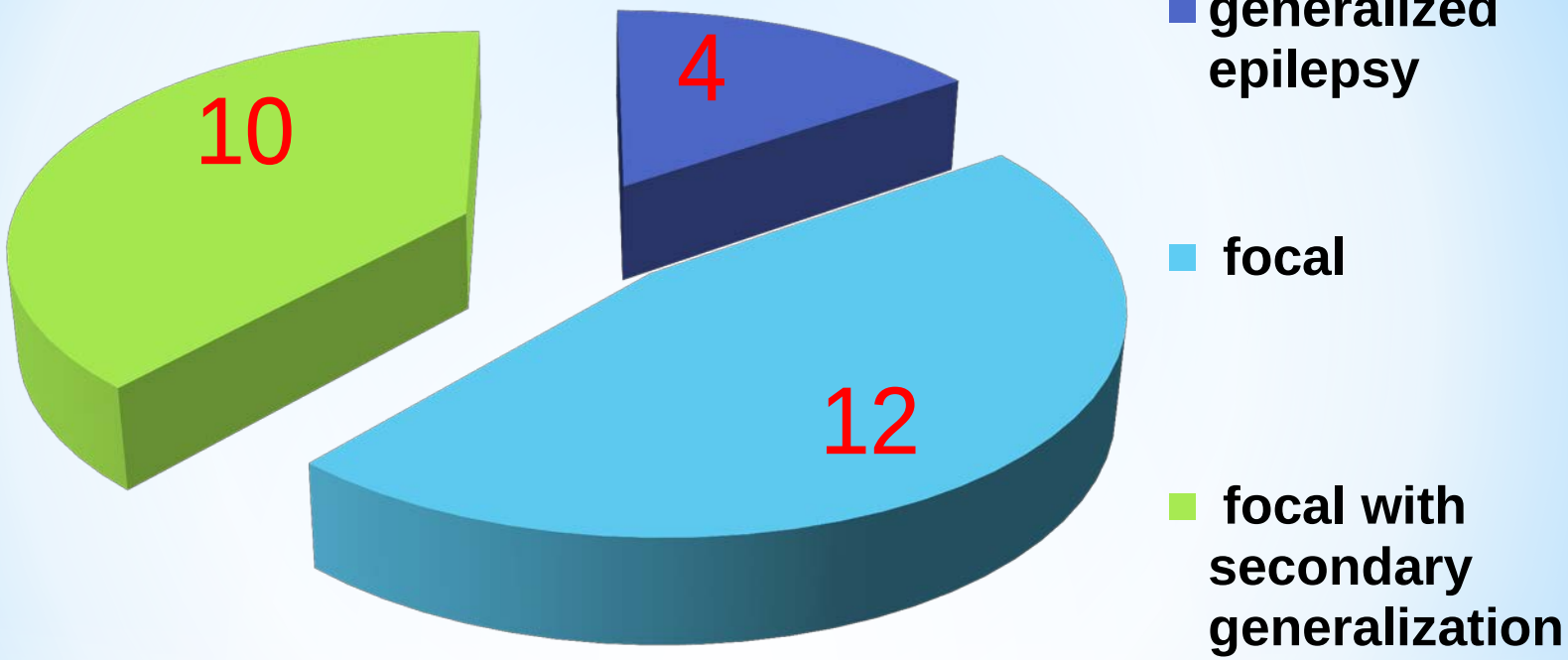
The patients' age ranged from 4.5 to 18 years.

Ten (38.5%) of them were girls. Sixteen (61.5%) were boys.

The control group were chosen to be age-and sex-matched to the patients group.

There were no obese children

Results



Type of epilepsy

Mean age of onset of epilepsy was 7.49 ± 4.2 years.

Mean duration was 5.09± 4.57 minutes.

Results Clinical

The seizure frequency ranged from once per month to six per day with mean of 47.6±53.2 seizures per month

Results Clinical

Seizures were uncontrolled (more than once per month) in 24 patients (92.3%)

and three patients (11.5%) had history of status epilepticus.

Results Clinical

Circadian distribution of the attacks was

diurnal in two patients (7.7%),

nocturnal in 12 (46.2%),

and both diurnal and nocturnal in 12 (46.2%).

Results Clinical

Six patients (23.1%) diagnosed had not received medications yet,

Ten (38.5%) were on monotherapy,

and 10 (38.5%) were on polytherapy

Results Clinical

Sleep clinical results

The patients' sleep complaint

Repeated awakenings during sleep in 15 patients (57.7%)

Daytime sleepiness in 13 patients (50%).

Snoring was encountered in only two patients (7.7%).

The patients had significantly

Higher arousal index and

percent of stage 2 from total sleep time but

lower SWS percent from TST and sleep efficiency,

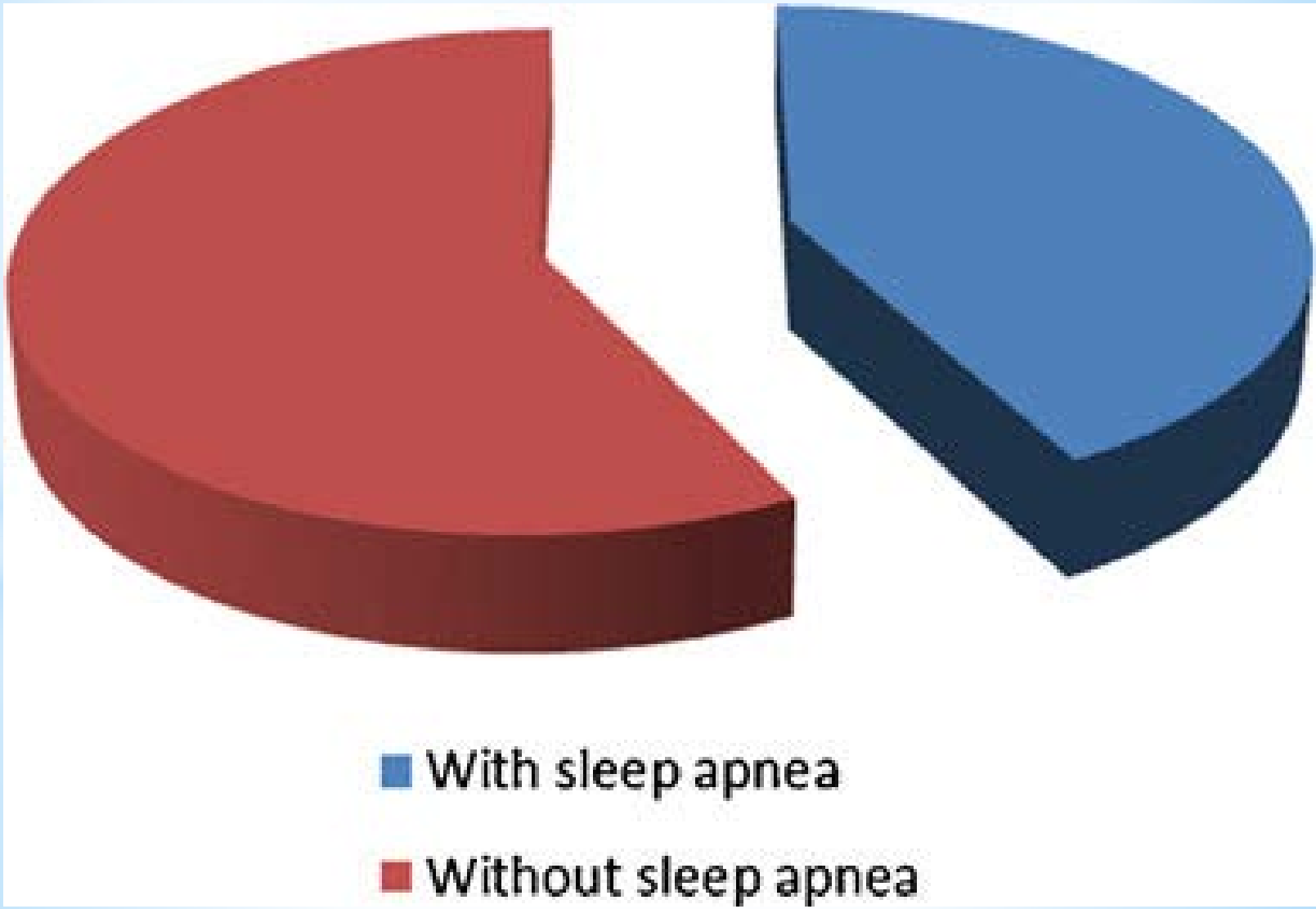
in comparison to the control group.

Polysomnographic results

Polysomnographic data	Patients group	Controls group	P-value
Sleep onset in min.	18.73 ± 17.5	14.1 ± 12.16	.53
Sleep efficiency %	73.17 ± 7.33	83.12 ± 10.59	.006
No. of awakenings	12 ± 6.03	13.67 ± 5.1	.449
Sleep latency to S1	102.01 ± 114.58	102.43 ± 176.07	.530
Sleep latency to S2	51.7 ± 74.76	17.35 ± 13.21	.157
Sleep latency SWS	81.82± 78.9	74.6 ± 47.55	.489
Sleep latency REM	196.88 ± 108.38	224.08 ± 90.67	.572
% of S1 from TST	8.18 ± 7.18	7.22 ± 6.86	.530
% of S2 from TST	57.01 ± 17.12	44.05 ± 14.98	.022
% of SWS from TST	23.11 ± 10.27	36.08 ± 14.9	.012
% of REM from TST	12.57 ± 9.33	12.62 ± 6.28	.706
Arousal index	8.23 ± 10.88	.89 ± .62	.000
Periodic Leg Movement index	.82 ± 1.33	.38 ± .35	.545
Apnea index REM	.39 ± 1.10	.00 ± .00	.109
Apnea index in NREM	.31 ± .39	.00 ± .1	1
Hypopnea index in REM	1.75 ± 4.91	.00 ± .00	.255
Hypopnea index in NREM	.7 ± .97	.00 ± .45	.775
Apnea hypopnea index	1.21 ± 1.46	.00 ± .75	.704

Eleven patients with epilepsy(42.3%) have Obstructive sleep apnea

Results


With sleep apnea
Without sleep apnea

Comparison between clinical variables in epilepsy patients'groups with and without OSA

Seizure frequency was significantly higher in the patients with OSA. All other clinical epilepsy, sleep, and EEG findings did not differ significantly

Comparison Results

If sleep deprivation is the assumed mechanism, one might expect that seizures during both sleep and wakefulness would be facilitated as in our study.

Results

If sleep fragmentation and frequent stage shifts resulting from apneas are responsible for provoking seizures, then seizures during sleep may be facilitated preferentially

[Wyler A etal 1981]

Results

Clinical variables	Group (1) Patients without OSA No. (%)	Group (2) Patients with OSA No. (%)	P-value
Age of onset	7.97 ± 3.37	6.83 ± 5.27	.310
Duration of illness	5.23 ± 3.86	4.89 ± 5.6	.435
Sex Female	7 (46.7%)	3 (27.3%)	.315
Male	8 (53.3%)	8 (72.7%)	
Type			
Generalized	3 (20%)	1 (9%)	
Focal	5 (33.3%)	7 (63.6%)	.305
Focal with secondary generalization	7 (46.7%)	3 (27.3%)	
Seizure frequency	26.55 ± 36.18	63.07 ± 59.38	.046
Status epilepticus	2 (13.3%)	1(9%)	.738
Circadian rhythm			
Diurnal	2 (13.3%)	0 (0%)	
Nocturnal	7 (46.7%)	5 (45.4%)	.550
Nocturnal and diurnal	6 (40%)	6 (54.6%)	
Treatment			
No Medication	3 (20%)	3 (27.3%)	.910
Monotherapy	6 (40%)	4 (36.3%)	
Polytherapy	6 (40%)	4 (36.3%)	

Apart from apnea and hypopnea indices, all other polysomnographic sleep parameters did not differ between patients' subgroups

Results
Comparison

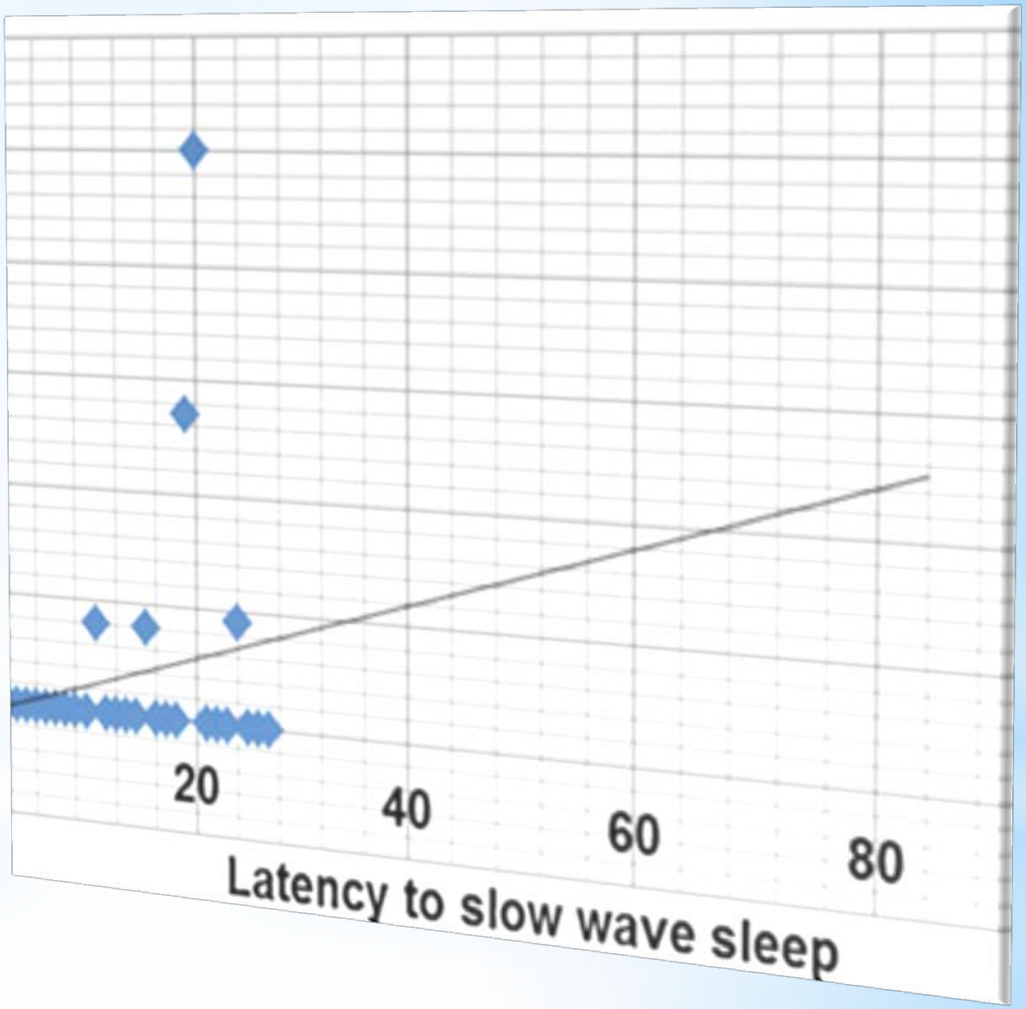
Polysomnographic data	Group (1) patients without OSA Mean ± SD	Group (2) patients with OSA Mean ± SD	P value
Sleep onset in min.	21.84 ± 21.46	14.49 ± 9.3	.659
Sleep efficiency %	85.3 ± 9.39	80.15 ± 11.83	.227
No. of awakenings	10.8 ± 5.02	13.64 ± 7.12	.404
Sleep latency to S1	92.66 ± 128.12	114.76 ± 97.61	.421
Sleep latency to S2	48.83 ± 69.57	55.63 ± 84.66	.659
Sleep latency SWS	79.23 ± 70.47	85.35 ± 92.67	.697
Sleep latency REM	182.69 ± 114.59	216.23 ± 101.34	.516
% of S1 from TST	8.53 ± 7.71	7.71 ± 6.72	.795
% of S2 from TST	57.81 ± 17.8	55.93 ± 16.94	.697
% of SWS from TST	22.07 ± 9	24.54 ± 12.08	.406
% of REM from TST	11.62 ± 9.88	13.87 ± 8.81	.311
Arousal index	5.57 ± 6.45	11.85 ± 14.57	.233
Periodic Leg Movement index	.86 ± 1.28	.75 ± 1.45	.546
Apnea index REM	.00 ± .00	.93 ± 1.58	.005
Apnea index in NREM	.16 ± .21	.51 ± .48	.062
Hypopnea index in REM	.03 ± .13	4.1 ± 7.05	.000
Hypopnea index in NREM	.23 ± .24	1.34 ± 1.22	.006
Apnea hypopnea index	.36 ± .27	2.36 ± 1.63	.000

Correlation between apnea/hypopnea index, Clinical and polysomnographic data

Apnea index in REM positively correlates with latency to deep sleep

Correlation

Correlation

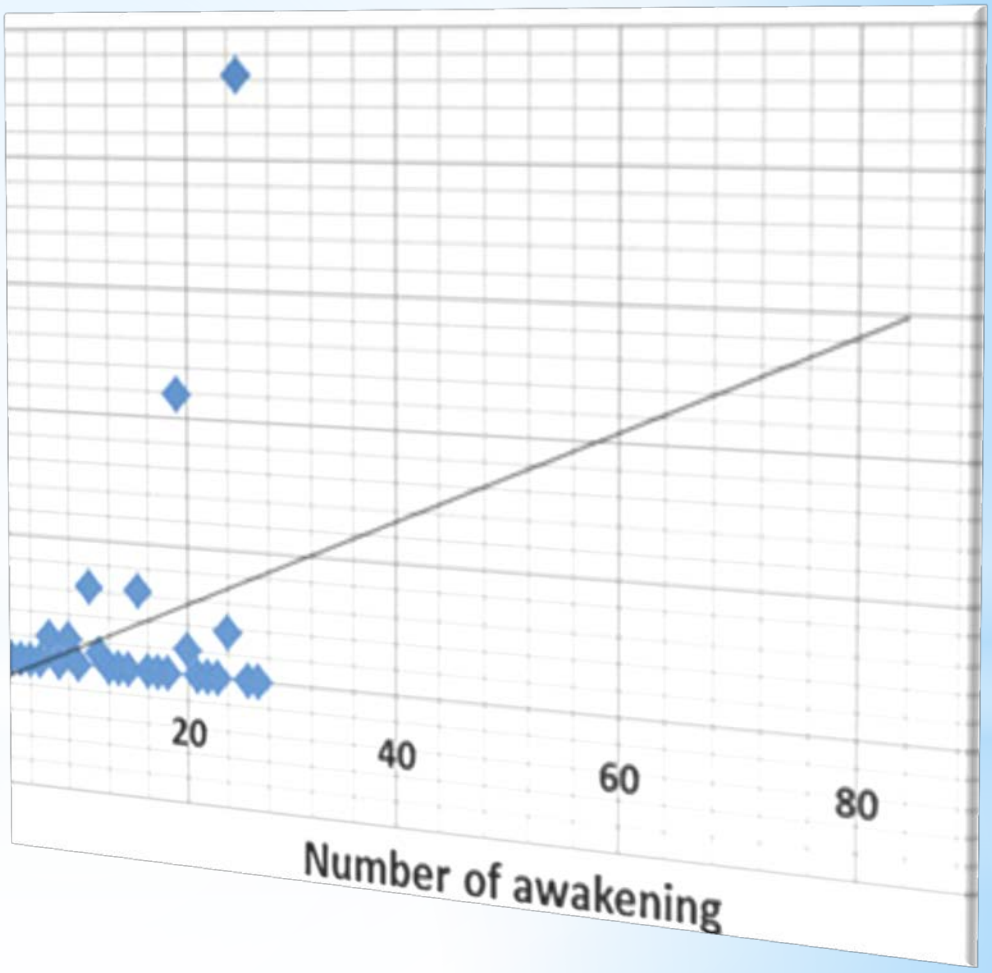


Correlation between Apnea index and latency to deep sleep

Hypopnea index in REM positively correlates with number of awaking (r=0.393, P=0.047).

Results
Correlation

Correlation



number of awakening and hypopnea index

It is well-known that sleep apnea is not the same throughout the course of the night. It tends to be worse in (REM) sleep

Discussions

Patients with epilepsy and OSA had significantly longer sleep latency and higher arousal index. A significant tendency towards light sleep than slow wave deep sleep

Discussions

In this study, no epilepsy or sleep data have been

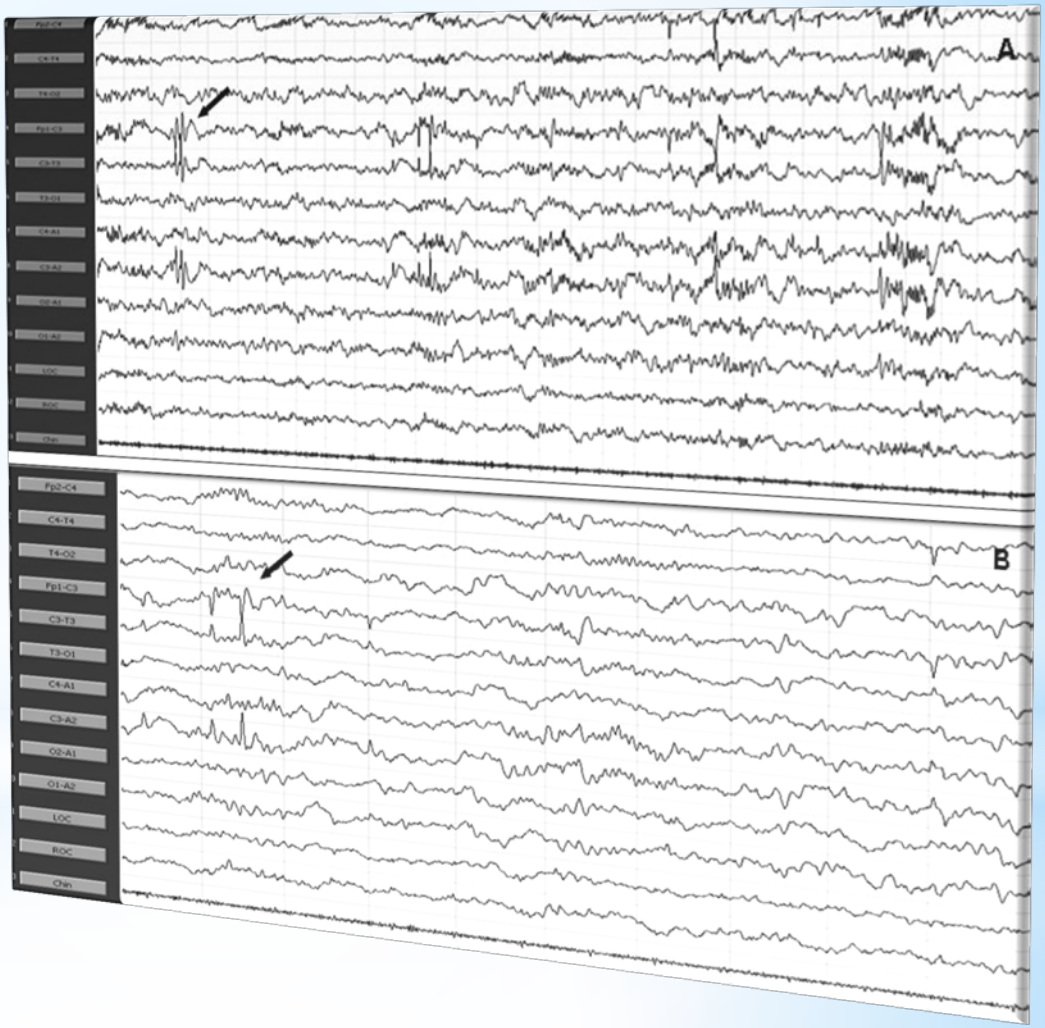
found to be significant predictors of OSA in patients with epilepsy

Discussions

Contradictory to previous study that reported that older, heavier male, and sleepier epileptics are more prone to have

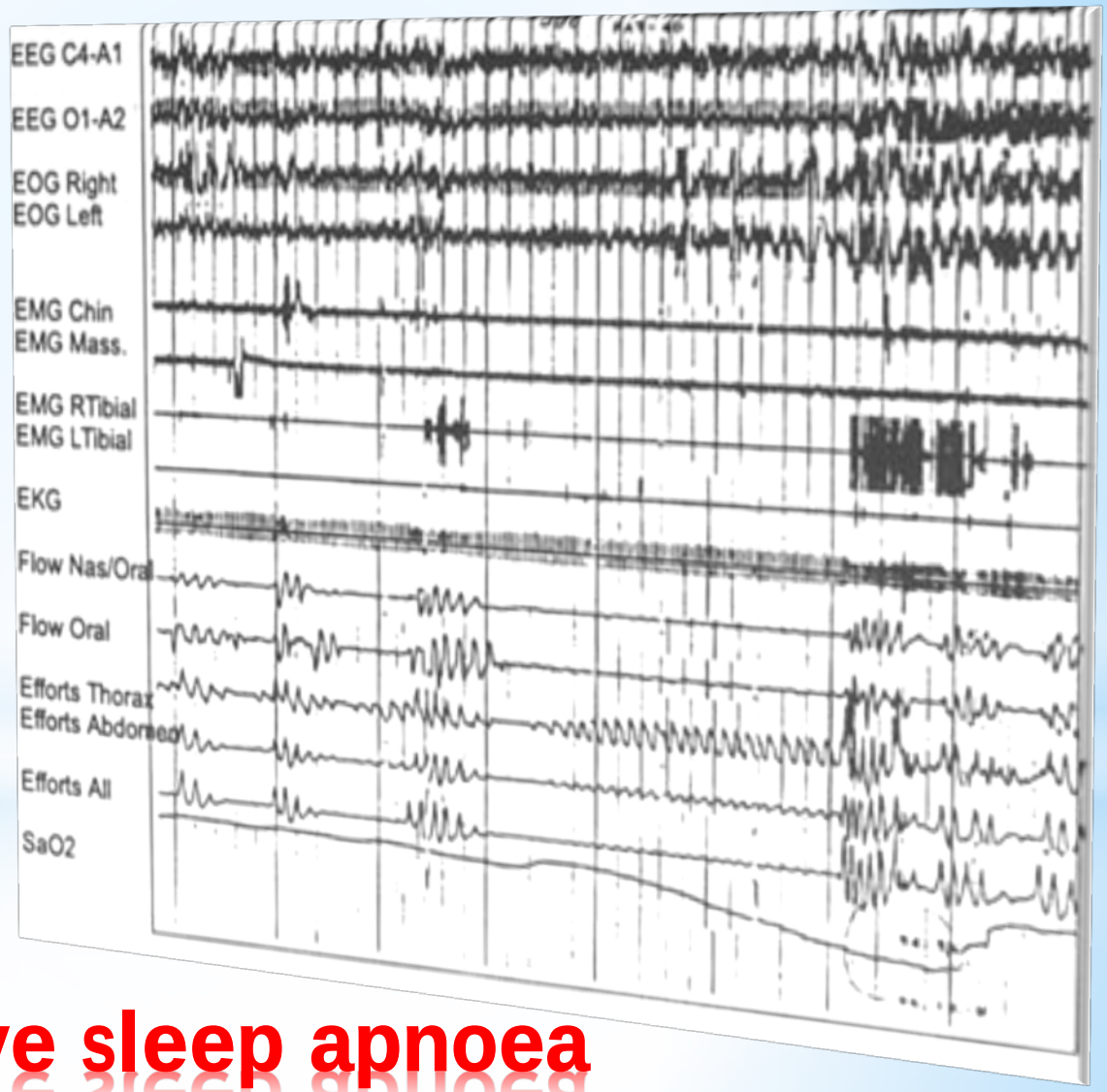
OSA [Raffaele M, etal 2003].

Discussions



left centrotemporal sharp waves

Polysomnographic recording



Obstructive sleep apnoea


Conclusions

Conclusions

Obstructive sleep apnea is frequent in patients with epilepsy.

Obstructive sleep apnea contribute to increase seizures frequency.

Investigating sleep apnea in all patients with epilepsy even those without sleep complaint

Recommendations

Egypt
Fayoum
neurology
Conference
10-12
November
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has01@
fayoum.edu.eg

Finally

Thank You