Review

The effects of obstructive sleep apnea syndrome (OSAS) on the executive functions of the brain before and after treatment

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ABSTRACT

PURPOSE: To summarize the information of published studies on the effect of OSAS on the executive functions of the brain in adult patients before and after any treatment. **METHOD:** An extensive literature search was performed on the Pub Med database. **RESULTS:** The OSAS inarguably causes neurocognitive deficit and reduced activation of brain regions responsible for cognitive function. The methods of treatment are: by a continuous positive air pressure (CPAP) device, mandibular treatment and tonsillectomy. These methods of treatment offer some protection to the cognitive areas of the brain. The severity of the syndrome correlates significantly with the scores of various neuropsychological batteries; these batteries vary even if they evaluate the same function. **CONCLUSIONS**: The severity of the syndrome is responsible for the degree of neurocognitive dysfunction. The most prevalent form of treatment is the use of a device with positive air pressure CPAP which acts as a protective factor in neurocognitive functions. Further research is needed because few studies have evaluated the effects of treatment using a CPAP device while the timeframe and the population that needs to be studied remain uncertain.

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INTRODUCTION

The obstructive sleep apnea syndrome (OSAS) is a disorder of breathing during sleep characterized by prolonged periods of full or partial obstruction of the upper airway[1], thus reducing oxygen saturation and causing brief awakenings[2]. Evidence from studies indicates that it affects at least 9 to 15% of middle aged people[3]. Compared to women the prevalence of OSAS among males is 1.5 to 3 times greater[4]. International literature indicates that patients with OSAS show deficits in most cognitive and executive brain

regions and that the severity of the syndrome correlates strongly with the scores of various neuropsychological tests[5-9].

The literature review of Andreou et al (2014) concludes that further research is needed to clarify which are the precise executive functions that have suffered damage in patients with OSAS[10].

The treatment of OSAS is performed by tonsillectomy, by mandibular treatment[11,12] and by the most prevalent therapy of granting a positive air pressure device (CPAP). CPAP therapy is based on restoration of the air flow on the upper airway during sleep, so that the air passes to the lungs freely[13]. Recent studies indicate that treatment using a CPAP device is associated with cognitive and executive improvements. The use of a CPAP device is effective in reducing the symptoms of drowsiness and in improving cognitive and executive function regions[10]. Patients with OSAS exhibit a poorer performance in certain cognitive functions and a reduced volume of gray matter in the hippocampus, in the parietal cortex and in the frontal regions of the brain. After CPAP treatment there have been improvements in memory, attention, executive functions and an increase of gray matter in the above areas[14].

Crawford-Achour et al (2015) observed that out of 126 patients aged 65 years and above, only 26% had proper treatment of more than 6 hours per night[15]. Finally Zimmerman et al (2006) found that memory decline was eight times more likely to be smoothed out with an average use of 6 hours of CPAP per night[16].

The purpose of this literature review is to study the effects of the obstructive sleep apnea syndrome (OSAS) in the executive functions of the brain in adult patients, before and after any treatment and whether the treatment may improve the decline that brain regions may have suffered.

METHOD

A literature search was conducted on the interval from 20/01/2017 to 20/04/2017 on the Pub Med database. The selection of the literature was done aiming at a comprehensive coverage of the topic. The keywords used were: **obstructive sleep apnea syndrome, executive functions, neurocognitive battery, neurocognitive functions**. The primary studies which have been used in this paper regard scientific research, qualitative and quantitative, which has been published in international journals.

Selected studies involve adult patients. Articles that involved patients with comorbidities, review articles and meta-analysis articles were excluded. Using the above keywords 126 articles were found in the PUBMED database.

From these 126 articles we excluded: 24 articles about children, 3 which were off topic, 16 articles that involved comorbidities and finally 36 reviews and meta-analysis articles. In conclusion, 49 articles were selected about OSAS relation to the executive functions, and 45 of them were used in this literature review because the remaining 4 were not written in English. Out of the 44 articles used, 30 articles concerned OSAS and the executive functions without offering treatment while the remaining 14 articles included treatment (Figure I).

On the basis of the above keywords we tried to retrace the chronological appearance of the articles. The first article mentioning OSAS and executive functions was from Bédard et al 1991 in the journal J clin Exp Neuropsycho[17] while the first article that included some treatment was from Montplaisir et al 1992 in the journal Sleep[18]. Finally, the journals with the most published articles on the relation of OSAS to the executive functions were the Sleep, the J Sleep Res and the Sleep Breath.

RESULTS

The severity of OSAS is significantly associated with neurological damage and blood cortisol levels, which account for 16% of the variance in the areas of learning, memory and working memory[19]. Also the reduction of the executive functions is directly linked to the severity of the syndrome and not to daytime drowsiness[20].

The study of Bédard et al (1991) found that, compared to the control group, moderate and severe OSAS presented differences in many cognitive functions and that both frequent awakenings and the nocturnal hypoxemia can contribute to cognitive and executive dysfunction[17]. The severity of OSAS is associated with the damage of brain tissues in the various cognitive areas; also the syndrome may increase the susceptibility of the brain resulting in an ageing population[21].

Severe OSAS promotes the loss of neuro-axles and causes impairment of myelin, the white substance and the frontal lobe resulting in irreversible damage to the cognitive and executive fields[22]. The study of Hrubos-Strom et al (2012) noted that the problem of verbal memory and executive function was mild in adults at high risk of OSAS. The average oxygen saturation was the index of

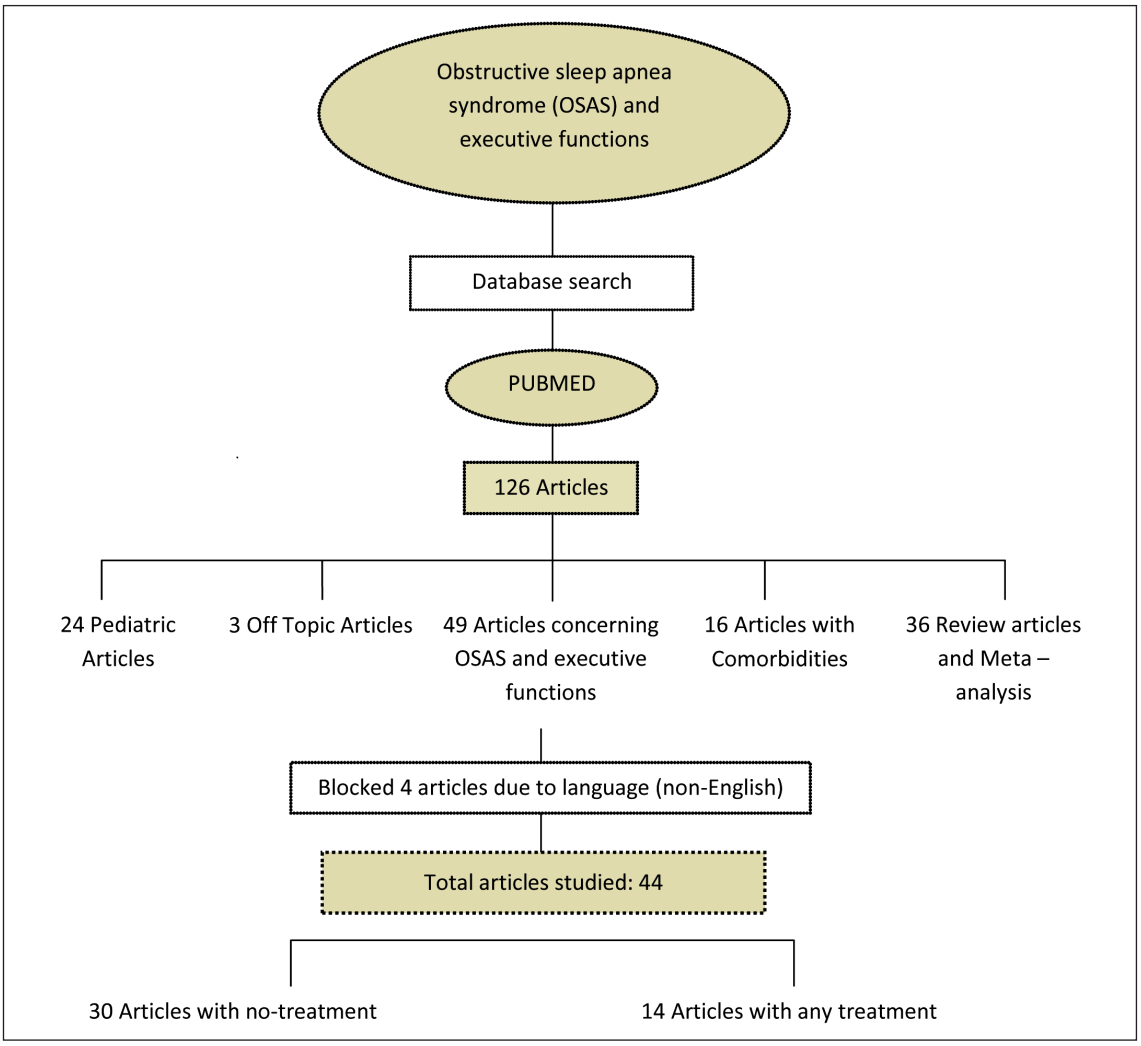

FIGURE 1. Schematic representation of the selected articles.

obstructive apnea and found to be significantly associated with cognitive function[23].

Patients with moderate and severe OSAS exhibit deficits in working memory and memory capacity[24] as well as reduced information processing rate, reduced working memory capacity and lack of attention[25]. Also the mild and moderate OSAS has an impact in the areas of attention, executive function, processing rate[26], decision making[27], visual-spatial and executive functions[28,29], attention[30], working memory[31,32], learning and also disorders in executive and neurocognitive functions as a whole[33].

Park et al (2016) in a recent study of 151 people noted that the OSAS group presented abnormalities in the parietal, frontal lobe and left hemisphere, regions responsible for proper cognitive function[34]. Patients with OSAS also exhibit reduced frontal activation[35].

Using electroencephalography (EEG) on OSAS patients, changes were observed during the REM stage not only in the frontal region of the brain but also across the entire range of neuropsychological functions[36], while using magnetic resonance imaging (MRI) it was found that grey matter density and the reduction of metabolic activity were changed even in patients without cognitive disorders, suggesting that the brain changes precede the onset of neurocognitive deficits[37].

According to Goya et al (2016) a group of patients with OSAS exhibited latency and procrastination during ongoing work[38]. Nemeth et al (2012) found that the memory performance was reduced in the OSAS group compared to the control group, but the learning process was similar in both groups[39].

Sharma et al (2010) reported in their survey that pa-

tients with OSAS had significantly reduced performance in tests of alertness, working memory, inhibition of responsiveness, problem-solving, executive function and reduced information processing rate resulting in reduced short-term memory[40].

Despite the fact that IQ testing is an important predictor of reduced attention and reduced executive function, no correlation with the above was found in the Olaithe et al (2015) study[41].

Shpirer et al (2012) noted a correlation of cognitive function in patients with OSAS to nightly hypoxemia[29]. Edwards et al (2014) after research concluded that cortisol levels are considered prognostic markers of cognitive functions[19]. Sales et al (2013) also demonstrated that Oxidative Stress is increased in patients with OSAS[42], also Borges et al (2013) note that OSAS without comorbidities does not lead to cognitive impairment[43]. Finally, another study of Chen et al (2011) of 394 people demonstrated that the MoCA method is a more sensitive tool for detecting cognitive impairment of patients with OSAS than the MMSE method[44].

In contrast to the above studies, the literature mentions items where no correlation between OSAS and the executive functions was found. In a study of 20 patients with OSAS, no deficits were found in executive functions[45], while the literature states that hypoxemia is an important cognitive deficit factor. Hoth et al (2013) found that the group with high hypoxemia displayed better levels of verbal memory in relation than the low hypoxemia group[46].

In a study of Rouleau et al (2002), there were no deficits in learning skills in patients with OSAS[47]. Finally, as noted by Shpirer et al (2012) the executive functions in certain patients, are not related to polysomnographic parameters[30] (Table I).

Treatment using a CPAP device

The study by Crawford - Achour et al (2015), after a 10-year study, demonstrated that CPAP treatment protects cognitive and executive performance, particularly in memory and normalizes the mental state of patients with OSAS[48]. Following a 3-month treatment with a CPAP device, Canessa et al (2011) observed significant improvement in memory, attention and executive functions. There was also an increase in the volume in the hippocampus and frontal regions[49].

Kushida et al (2012), after analysing the results, concluded that CPAP treatment leads to a transient enhancement of executive and frontal lobe function[50]. Also in the study of Lau et al (2010), where they tried to compare the state of 37 patients before and 3 months after treatment, there was an improvement in the quality of sleep and a reduction in daytime drowsiness in patients with OSAS[51].

According to Dalmases et al (2015) there was an improvement in brain function after 3 months of treatment, increasing the connectivity of the default mode network (DMN) and diluting the cerebral cortex[52]. It was also found that since the first month of treatment with CPAP, there was a complete reversal of white matter abnormalities, fractional imbalance, average diffusion and improved memory, attention and executive functions[53].

Finally, the study by Ferini-Ssrambi et al (2003) observed at the onset of treatment with CPAP, deficits in attention, learning, visual-spatial learning and executive functions. After 15 days of CPAP treatment improvement was observed and after 4 months CPAP treatment no further improvement was observed in the above areas, the results confirm the hypothesis of partial reversibility of cognitive dysfunction in patients with OSAS following treatment with CPAP[54].

In contrast to the above results, there are studies in which no improvement has been observed after treatment with a CPAP device in brain regions associated with cognitive functions. In a study by Berlowitz et al (2013), after treatment using a CPAP device for 6 months, there was no improvement in neurocognitive functions compared to virtual CPAP treatment[55]. The same conclusion is reached by Saunamaki et al (2010) in which no improvement in the performance of patients with OSAS was observed after 6 months of treatment[56].

Also in a study conducted before CPAP treatment there was a slightly disturbed mental state, deficits in executive functions and learning while after 6 months of treatment no improvement was found in the above categories[57]. Naegele et al (1998) found that of short-term memory impairment was evident in patients with OSAS despite CPAP treatment of 4 and 6 months[58].

Damage resoration by surgical intervention

In a study conducted in 51 patients with OSAS improvement in learning and memory was seen after tonsillectomy (UPPP) as well as improvement in executive functions due to better oxygenation[12].

Teqelberg et al (2012) concluded that after 6 months of mandibular treatment an improvement was observed concerning the awakenings, attention and executive function in patients with severe OSAS. The findings suggest that mandibular treatment can be an important treatment for OSAS[12] (Table II).

TABLE I. Study data without treatment

Study	Study Group	Age (years)	Methods	Neuropsychological Tests
Yilmaz et al (2016)	**Total n=43** Patient n=28 Control n=15	18 – 60 years	PSG, ESS, MRI	WCST, SCWT
Park et al (2016)	**Total n=151** Patient n=69 Control n=82	Patient = 48.3±9.2 years Control = 47.6±9.1 years	PSG, ESS, MRI	BDI-II, BAI, PSQI
Goya et al (2016)	**Total n=35** Patient n=20 Control n=15	40-65 years	PSG, ESS	MMSE, WASI, BCI-II, SCWT
Delazer et al (2016)	**Total n= 50** Patient n=30 Control n= 20	Mean age = 51.0	PSG, IED, SSS	HADS, VFT, TMT, WMS, VLMT, IGT
Olaithe et al (2015)	**Total n= 150** Patient n=134 Control n= 16	18 years and above	PSG, ESS	COWA, TMT, NART
Chen et al (2015)	**Total n= 241** Patient n= 201 1. Primary snoring n= 49 2. Mild OSAS n=51 3. Moderate OSAS n=50 4. Severe OSAS n= 51 5. Control n= 40	1= 32.53±10.20 2= 33.33±7.97 3= 35.40±10.43 4= 32.80±9.94 5 = 34.53±9.95	PSG, ESS	MoCA, MMSE
Gelir et al (2014)	**Total n=30** Patient n=15 Control n=15	Patient = 41.5±2.50 Control =35.9±2.5	PSG, ESS	TMT, N-Back task, MDT
Edwards et al (2014)	**Total n= 55** Patient n=55 Control n= 0	Patient = 29-65 years	PSG	WAIS, DSST, BVMT, HVLT, TMT, DVT, SCWT, TWFT
Tulek et al (2013)	**Total n= 27** Patient n=14 Control n= 13	Patient =36.0± 6.1 Control = 37.2 ±6.9	PSG, ESS	TPT, BDI, DSST, WCST, RL
Sales et al (2013)	**Total n= 38** Patient Moderate-Severe OSAS n= 24 Control n= 14	Patient = 48.1 Control = 49.6	PSG, ESS	BDI-II, SCWT, TAI, MMTE
Hoth et al (2013)	**Total n= 40** Patient High Hypoxemia n= 20 Control Low Hypoxemia=20	Patient = 55.6 Control= 53.5	PSG, ESS	BDI, AMNART, PASAT, TMT, COWAT, LNST, HVLT-R, GP
Borges et al (2013)	**Total n=44** Patient n=22 Control n=22	Patient n 52.1 Control=50.7	ESS	BAI, BDI, SCWT, RPM, VAMS, VFT, TMT, DS, CORSI, CPT
Nemeth et al (2012)	**Total n=40** Patient n=20 Control n=20	Patient = 52.70 Control = 52.40	ESS	ASRT, LST
Hrubos-Strom et al (2012)	**Patient n= 290**	Mean age = 48.2	PSG, ESS	BQ, SCWT, RAVLT
Shpirer et al (2012)	**Total n=** 40 patients Mild OSAS n=11 Moderate OSAS n=15 Severe OSAS n=14	Mean age 53.3	PSG, EDS	CPT, TMT, BDI, STAI, ADHD, SRS, SRS, ASRS, MMSE, WAIS, TOL, WCST, VFT, DS

TABLE I. (continued) Study data without treatment

Study	Study Group	Age (years)	Methods	Neuropsychological Tests
Torelli et al (2011)	**Total n= 30** Patient Moderate-Severe n=16 Control n= 14	Patient = 55.8±6.7 Control =57.0±5.2	ESS, MRI	MMSE, RAVL, DS, VMT, ROCF, SVFT, SCWT
Greneche et al (2011)	**Total n= 22** Patient n=12 Control n= 10	Patient = 51.8 Control= 49.6	PSG, ESS	MMSE, WMS, WCST, VAS, DS, PF, ST
Zhang et al (2011)	**Total n = 18** Patient n=9 Control n= 9	Patient =38.4 Control =37.9	PSG, ESS, MRI	
Chen et al (2011)	**Total n= 394** Primary snoring n = 46 Mild OSAS n = 92 Moderate OSAS n = 70 Severe OSAS= 186	Primary snoring= 44.5±10.2 Mild OSAS = 46.2 ± 12.1 Moderate OSAS = 47.2±10.9 Severe OSAS = 46.0±11.0	PSG, ESS, EDS	MoCA, MMSE
Sharma et al (2010)	**Total n= 75** Patient Severe osa n=50 Control n= 25	Patient = 43±7.5 Control = 45.6±6.2	PSG, ESS	WAIS, DSST, WCST, DS, SCWT
Yaouhi et al (2009)	**Total n= 30** Patient n=16 Control n= 14	Patient = 54.75±5.71 Control =52.71±7.01	PSG, ESS, MRI	SAQLI, MMSE, BDI-II, WMS
Lis et al (2008)	**Total n= 30** Patient n=20 Control n= 10	Patient =57.9 Control =53.6	PSG	SRT, SDT, CRT, CRT-vig, N-back tasks
Sagaspe et al (2007)	**Total n= 35** Patient OSAS n=20 Control INSOMNIA n= 10	Patient OSAS =45.7±9.5 Control INSOMNIA = 47.4±12.4	PSG, ESS	RT, SSRT
Quan et al (2006)	**Total n= 141** Patient n=67 Control n= 74	Patient mean age = 54.4 Control mean age = 57.4	PSG, ESS	GP, DSST, WAIS, SCWT, TMT, LNS, DS
Alchanatis et al (2004)	**Total n= 46** Patient OSAS n= 22 OSAS Subgroup n=14 Control n= 10	OSAS= 49±9.7 OSAS Subgroup = 48±10.1 Control = 42.9±10.5	MRS, PROBE, T1, FRAIR	
Vestraeten et al (2004)	**Total n= 65** Patient n=33 Control n= 32	Mean age = 48.3	PSG	TMT, SDMT, SCWT, WAIS, FPT, RT, DS, VVS
Rouleau et al (2002)	**Total n= 46** Patient n=28 Control n= 18	Patient = **47.4** Control n = **47.2**	PSG, MSLT, FCRTT,	**D2T, TMT, WCST, VFT, WMS, RAVLT, WAIS, MTT, DS, RPT**
Salorio et al (2002)	**Total n= 52** Patient n=28 Mild OSAS n = 10 Moderate OSAS n = 6 Severe OSAS= 14 Control n= 24	28-60 years	PSG	WCST, VFT, CVLT,
Morisson et al (1998)	**Total n= 31** Patient n=21 Control n= 10	Mean age = 44	EEG, MSLT, EDS	
Bédard et al (1991)	**Total n= 30** Control n=10 Moderate n=10 Severe n=10	Control =50.0 Moderate =53.2 Severe = 51.5	PSG, EDS, MSLT	WAIS, RAVL, WMS, VFT, TMT, VMT, FCRTT, WISC

TABLE II. Study data with treatment

Study	Study group	Age (years)	Therapy	Follow ups	Methods	Neuropsychological Tests
Crawford-Achour et al (2015)	**Total n=126** Patient n=93 (χωρίς CPAP) Control n=33 (με CPAP)	Patient = 74.7±1.1 Control =75.0±1.1	**CPAP**	10 Years	EPSG, ESS	MMSE, WAIS, VAS, PDS, GB, BT, SCWT, VFT
Dalmases et al (2015)	**Total n=66** Patient n=33 Cpap n= 17 CC n = 16	Patient = 71.30 ± 5.51 CPAP= 70.76±5.13 CC = 71.88±6.00	**CPAP**	3 Months	PSG, ESS, MRI, QSQ	HANDS, RAVLT, MMSE, TMT, VFT, DIGIT SPAN
Castronovo et al (2014)	**Total n=32** Patient n=17 Control n=15	Patient = 43.23 Control= 42.15	**CPAP**	3-12 Months	PSG, MRI	MMSE, DS, CORSI, RL, TMS, SCWT, PASAT, RPM
Berlowitz et al (2013)	**Total n=1098** Patient active n=556 Control sham n=542	Mean age= 52	**CPAP**	6 Months	PSG, ESS	PNTTT, BSRT-SR, SWMT-OMD
Kushida et al (2012)	**Total n=1098** Patient n=556 Control n=542	Patient = 52.2 Control= 50.8	**CPAP**	2-6 Months	PSG, ESS	PFN-TOTL, BSRT-SR, SWMT-OMD
Teqelberg et al (2012)	**Total n=45** Patient - Severe n= 30 Control - Moderate n=15	Mean age = 20-65	**Θεραπεία κάτω γνάθου**	6 Months	PSG, ESS	WMT, CPT-IP, TMT
Canessa et al (2011)	**Total n=32** Patient n=17 Control n=15	Patient mean age = 44 Control mean age = 42.15	**CPAP**	3 Months	PSG, ESS, MRI	SF-36, BDI, DS, SCWT, RL, MMSE, PASAT, WSRT, CORSI, TMT
Saunamaki et al (2010)	**Total n=37** Patient n=20 Control n=17	Mean age = 20-65	**CPAP**	6 Months	PSG, ESS	ROCFT, TMT, WAIS, DS, DSS, BDS, TWFT, IEDSS, CANTAB
Lan et al (2010)	**Total n=64** Patient n=37 Control n=27	Patient = 57.9 Control = 56.7	**CPAP**	3 Months	PSG, ESS	PSQI, DS, VMST , WMS, N-back task, WAIS, WCST, WISC, TMT, WMS, SCWT
Saunamaki et al (2009)	**Total n=60** Patient n=40 Control n=20	Patient = 47.2±7.8 Control = 42.9±10.3	**CPAP**	6 Months	PSG, ESS	IEDSS, WAIS, CANTAB, ROCFT , WCST, DSST, BDS, TMT, TWFT
Ferini-Ssrambi et al (2003)	**Total n=46** Patient severe OSAS n=23 Control n=23	Mean age = 56.5 ± 6.13	**CPAP**	15 Days 4 Months	PSG, ESS	BDI, DS, CORSI, TMT, SCWT TR2, PPT, ROCF, VFT, RPM
Dahlöf et al (2002)	**Total n=104** Patient n=51 Control n=53	Mean age= 50	**Αμυγδα-λεκτομή**	6 Month	PSG	WCST, BVRT, SRB, CDT, WAIS
Naegele et al (1998)	**Total n=34** Patient n=17 Control n=17	Patient = 44,80 Control = 45.30	**CPAP**	4-6 Months	PSG	TMT, DBL-CANC, SCWT, CORSI, DS, DC, WISC, VER-L, VIS-L, VFT
Montplaisir et al (1992)	**Total n=30** Patient n=20 Control n=10	Mean age = 35-65	**CPAP**	6 Months	EDS, MSLT	WMS, TWFT, FCRTT, WAIS, ROCF, TMT, BDS

ABBREVIATIONS

A ADHD = Attention-Deficit Hyperactivity Disorder,
AMNART = American National Adult Reading Test,
Apfg = Anterior Preffontal Guri,
ASRT = Alternating Serialreaction Time

B BAI = The Beck Anxiety Inventory,
BDI = The Beck Depression Inventory,
BD = Block Design,
BQ = Berlin Questionnaire,
BSRT-SR = Buschke Selective Reminding Test-Sum Recall,
BT = Buschle Test,
BVMT = Brief Visuospatial Memory Test-Revised,
BVRT = Benton visual retention test

C CASI = Cognitive Ability Screening Instrument,
CANTAB = Cambridge Neuropsychological Test Automated Battery,
CBF = Cereblar Blood Flow,
CD = The Claeson–Dahl test,
CORSI = Visuo-spatial short-term memory,
COWAT = Controlled Oral Word Association task,
CRT = Choice Reaction Task,
CPT = Continuous Performance Test,
CPT-IP = Continuous, Performance Test, Identical Pairs Version,
CRT-vig = Vigilance Version of the CRT,
CVLT = California Verbal Learning Test

D DBL-CANC = Digit-Cancellation Task,
DC-SPAN = Double-Encoding Task,
D2T = Test of Attention,
DS = Digit-span Forward and Backward,
DSST = Digit Symbol Substitution Test,
DVT = Digit Vigilance Test

E EDS = Exxcessive Daytime Sleepiness,
ESS = Epworth Sleepiness Scale

F FC = Regional Functional Connectivity,
FCRTT = Four-Choice Reaction Time Test,
FPT = Flexibility Point Test

G GP = Grooved Pegboard

H HVLT = Hopkins Verbal Learning Test-Revised

I IEDSS = Intra-Extra Dimensional Set Shifting test

L LNST = Letter-Number Sequencing Test

M MMSE = Mini-Mental- State Examination,
MoCA = Montreal Cognitive Assessment,
MRI = Magnetic Resonance Imaging,
MSLT= Multiple Sleep Latency Test,
MSNA = Muscle Sympathetic Nerne Activity,
MTT = Mirror Tracing Task

N NART = The National Adult Reading Test ,
N-Back Tasks = were used with two versions of WM load

P PASAT = Paced Auditory Serial Addition,
PDS = Pichot Depression Scale,
PF = Pichot scale of fatigue,
PFN-TOTL = Pathfinder Number Test-Total Time,
PNTTT = Pathfinder Number Test-Total Time,
PPT = Purdue Pegboard test,
PSG = Polysomnography,
PSQI = Pittsburg Sleep Quality Inventory

Q QAQ = Quebec Sleep Questionnaire

R RAVLT = Rey Auditory Verbal Listening Test,
RL= Rey list learning, recall, and recognition verbal long-term memory,
ROCF = Rey-Osterreith Complex Figure,
RPM = Raven Progressive Matrices,
RPT = Rotary Pursuit Task,
RST = Reading Span Task,
RT= Reaction Time

S SAS = Supervisory Attentional System,
SAQLI = Calgary Sleep Apnea Quality of Life Index,
SCWT = Stroop Color Word Test,
SDMT = Symbol Digit Modalities Test,
SDT = Stimulus Discrimination Task,
SF-36 = The Short Form (36) Health Survey ,
SOC = Stockings of Cambridge,
SPECT = Single Pfoton Emission Computed,
SRB = Synonyms Reasoning and Block design test,
SRS =Self-Report Scale,
SRT = Simple Reaction Task,
SSRT = Stop Signal Reaction Time,
SSS = Stanford Drowsiness Scale,
STAI = State-Trait Anxiety Inventory,
ST = Stenberg Tasks,
SVFT = Semantic Verbal Fluency Task,
SWMT = Sustained Working Memory Test-Overall Mid-Day Index,
SWMT-OMD =Sustained Working Memory Test-Overall Mid-Day Index

T TAI = Trait Anxiety Inventory,
TOL = Tower of London test,
TMS = Trail Making Test,
TMT = Trail Making Test
TR2 = Reaction Time Test,
TPT = Toulouse Pieron Test,
TWFT = Thurstone Word Fluency Test

V VAMS = Visual Analogue Mood Scales,
VAS= Visual Analogue Scale,
VER-L= Verbal Learning Tests for Long-Term Memory,
VFT = Verdal Fluency Test,
VIS-L= Visual learning tests for long-term memory,
VLMT = Verbal Learning Memory Test,
VMST = Verbal memory scanning task,
VMT = Visual memory test,
VVS = Visual Vigilance Test

W WAIS = Wechsler Adult Intelligence Scale,
WCST = Wisconsin Card Sorting Test,
WMT = Working memory test,
WMS = Wechsler Memory Scale,
WMS-R = Wechsler Memory Scale-Revised,
WSRT = Wilcoxon Signed Rank Test

DISCUSSION

OSAS inarguably causes neurocognitive deficiency and that the severity of the syndrome is responsible for the extent of neurocognitive dysfunction. Patients with OSAS exhibit depression, anxiety, drowsiness, reduced daily functionality and decreased quality of life. The treatment in adult patients with OSAS is: 1) treatment with continuous positive air pressure (CPAP) which is considered to be the most prevalent form of treatment, 2) mandible treatment and 3) tonsillectomy. The above methods act protectively against the maintenance and improvement of neurocognitive deficits.

The literature review of Gagnon et al (2014) is in agreement with the above data. The authors indicate that OSAS is directly linked to the cognitive and executive dysfunction and the possibility of permanent brain damage as well as the presence of dementia in elderly patients[59].

Vaessen et al (2015) observed that cognitive deficiency appears to be partly related to daytime somnolence and that the impact of CPAP treatment on cognitive functions requires further study and investigation, conclusions which agree with our review[60].

The study by Decary et al (2000) also agrees that cognitive performance test batteries vary even if they evaluate the same cognitive or executive function[61].

The same conclusion is reached by Gurubhagavatula (2010) that OSAS causes a multitude of medical consequences caused by the fall of hemoglobin saturation and the repeated awakening during sleep. It also provides new data about OSAS being responsible for the risk of increased motor and occupational accidents and that treatment, besides the improvement offered in the above deficits and the quality of life, also helps to reduce the enormous financial burden on the state in patients diagnosed with OSAS[62].

Grigg-Damberger et al (2012) observed that snoring increases the risk of neurocongnitive damage and that middle-aged people with severe OSAS are at a greater risk for cognitive impairment than young adults with apnea of equal severity[63].

Also, Weaver et al (2007) report data consistent with this review that most articles in the international literature support the use of a CPAP device. Following treatment, elderly patients with OSAS have improved alertness, memory and executive function and the disruption of sleep from nocturia was also reduced. Finally, a positive effect of the treatment was observed in the factors that affect heart function[64].

Regarding the limitations of this literature review the search took place only on the Pub Med databases. The articles that were used included the keywords: Sleep Apnea Syndrome, Executive Functions, Neurocognitive Battery, Neurocognitive Functions. The search was age-targeted to adult patients. Articles that included comorbidities, literature review articles as well as articles of meta-analysis were excluded. Finally, only articles in English were used.

In conclusion, OSAS arguably causes neurocognitive deficiency and reduced activation of brain regions responsible for cognitive function. The severity of the syndrome is significantly related to the scores of various neuropsychological batteries – tests. The most prevalent form of treatment is the use of a CPAP device which acts as a protective factor in neurocognitive function, nevertheless further research is required because few studies have evaluated the effect of treatments with a CPAP device while the time period and the population that needs to be studied remain uncertain.

ΠΕΡΙΛΗΨΗ

Η επίδραση του Συνδρόμου της Αποφρακτικής Άπνοιας Ύπνου (ΣΑΑΥ) πριν και μετά τη θεραπεία στις εκτελεστικές λειτουργίες του εγκεφάλου

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Σκοπός: *Να συνοψίσει τις πληροφορίες των δημοσιευμένων μελετών σχετικά με την επίδραση του ΣΑΑΥ στις εκτελεστικές λειτουργίες του εγκεφάλου σε ενήλικες ασθενείς πριν και μετά την οποιαδήποτε θεραπεία.* ***Μέθοδος:*** *Πραγματοποιήθηκε εκτεταμένη αναζήτηση βιβλιογραφίας στις βάσεις δεδομένων Pub*

Med. ***Αποτελέσματα:*** *Το ΣΑΑΥ αναμφισβήτητα προκαλεί νευρογνωστική ελλειμματικότητα και μειωμένη ενεργοποίηση περιοχών του εγκεφάλου που είναι υπεύθυνες για τη γνωστική λειτουργία. Οι τρόποι θεραπείας είναι με συσκευή θετικής πίεσης αέρα (CPAP), θεραπεία κάτω γνάθου και αμυγδαλεκτομή, οι παραπάνω τρόποι θεραπείας προσφέρουν κάποιου είδους προστασία στις γνωστικές περιοχές του εγκεφάλου. Η σοβαρότητα του συνδρόμου σχετίζεται έντονα με τις βαθμολογίες των διαφόρων νευροψυχολογικών μπαταριών, οι μπαταρίες αυτές ποικίλλουν ακόμη και εάν αξιολογούν την ίδια λειτουργία.* ***Συμπεράσματα:*** *Η σοβαρότητα του Συνδρόμου είναι υπεύθυνη για τον βαθμό νευρογνωστικής δυσλειτουργίας. Η ποιο διαδεδομένη θεραπευτική αντιμετώπιση είναι η χρήση συσκευής με θετική πίεση αέρα CPAP η οποία δρα και ως προστατευτικός παράγοντας στη νευρωγνωστική λειτουργία. Απαιτείται περαιτέρω έρευνα διότι λίγες μελέτες έχουν αξιολογήσει την επίδραση της θεραπείες με συσκευή CPAP και είναι ακόμη ασαφή το χρονικό διάστημα και ο πληθυσμός που πρέπει να απευθύνεται.*

Πνεύμων 2017, 30(4):243-254.

Λέξεις - Κλειδιά: *Σύνδρομο Αποφρακτικής Άπνοιας Ύπνου, Εκτελεστικές λειτουργίες, Νευρογνωστικές συστοιχίες, Νευρογνωστικές λειτουργίες*

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