PERFORMANCE IN SELECTED COGNITIVE FUNCTIONS IN PEDIATRIC PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Výkon vo vybraných kognitívnych funkciách u detských pacientov s obštrukčným spánkovým apnoe

Michaela BABNICOVA, Anna DURDIKOVA, Stanislava SUROVIAKOVA, Dominika DVORSKA, Peter DURDIK, Peter BANOVCIN

Comenius University in Bratislava, Jessenius Faculty of Medicine in Martin, Paediatric department,
head prof. MUDr. P. Bánovčin, PhD.

Abstract
Objectives: The aim of this study was to use neuropsychological assessment to investigate the relationship between obesity and cognitive performance in patients with OSA.
Background: Obstructive sleep apnea (OSA) in children is a frequently occurring sleep- related breathing disorder, which can contribute to the deterioration of cognitive performance. Obesity is considered a risk factor for the development of this disease, and the incidence of OSA has been shown to be higher in obese patients than in those of normal weight.
Methods: The study included 41 probands who were divided into 2 groups: patients with OSA and an increased body mass index corresponding to obesity (23 probands) and a control group without chronic diseases (18 probands). All probands were administered a neuropsychological examination consisting of 5 tests to cover basic cognitive functions such as memory, attention and executive functions. We compared these results with a healthy control group.
Results: We found that the clinical group scored significantly lower than the control group in some memory tests. In the attention tests, except for two subtests, the probands of the clinical group scored equally poorly and achieved the same results in the measurement of executive functions.
Conclusion: Our results present a significant association between impaired cognitive performance and the presence of OSA in obese patients. Results of our work also point to the usefulness of testing cognitive functions in the pediatric patient population *(Tab. 4, Ref. 30). Text in PDF www.lekarsky.herba.sk.*
KEY WORDS: cognitive functions, memory, attention, executive functions, obstructive sleep apnea.
Lek Obz 2024, 73 (9): 312-317

Abstrakt
Cieľom tejto štúdie bolo využiť neuropsychologické vyšetrenie na skúmanie vzťahov medzi obezitou a kognitívnou výkonnosťou u pacientov s OSA.
Obštrukčné spánkové apnoe (OSA) u detí je často sa vyskytujúca porucha dýchania súvisiaca so spánkom, ktorá môže prispieť k zhoršeniu kognitívnej výkonnosti. Obezita sa považuje za rizikový faktor rozvoja tohto ochorenia a u obéznych pacientov sa ukázalo, že výskyt OSA je vyšší ako u pacientov s normálnou hmotnosťou.
Do štúdie bolo zaradených 41 probandov, ktorí boli rozdelení do 2 skupín: pacienti s OSA a zvýšeným indexom telesnej hmotnosti zodpovedajúcim obezite (23 probandov) a kontrolná skupina bez chronických ochorení (18 probandov). Všetkým probandom bolo aplikované neuropsychologické vyšetrenie pozostávajúce z batérie 5 testov na pokrytie základných kognitívnych funkcií, ako sú pamäť, pozornosť a exekutívne funkcie. Tieto výsledky sme porovnali so zdravou kontrolnou skupinou. Výsledky poukazujú na zistenia, že klinická skupina dosiahla v niektorých pamäťových testoch výrazne nižšie skóre ako kontrolná skupina. V testoch pozornosti, okrem dvoch subtestov, probandi klinickej skupiny skórovali rovnako zle a rovnaké výsledky dosiahli aj pri meraní exekutívnych funkcií.
Naše výsledky prezentujú významnú súvislosť medzi zhoršenou kognitívnou výkonnosťou a prítomnosťou OSA u obéznych pacientov. Výsledky našej práce poukazujú aj na užitočnosť testovania kognitívnych funkcií v populácii detských pacientov *(tab. 4, lit. 30). Text v PDF www.lekarsky.herba.sk.*
KĽÚČOVÉ SLOVÁ: kognitívne funkcie, pamäť, pozornosť, exekutívne funkcie, obštrukčné spánkové apnoe.
Lek Obz 2024, 73 (9): 312-317

Introduction

Sleep disorders in the pediatric population are associated with many problems, including somatic illnesses (1, 2), impaired academic performance (3, 4, 5, 6), impaired cognitive function (7, 8), and psychiatric problems (9, 10, 11). The relationship between sleep problems and emotional, behavioral, and somatic symptoms is often complicated and reciprocal (7). Awareness of these psychosocial and somatic connections is useful for understanding the importance of early diagnosis and the need for further intervention (12).

Studies have found that disrupted sleep can cause impairments in memory, decision-making and general learning ability. it is influenced by performance in tasks requiring long-term attention, in some components of memory, visuospatial abilities and in the field of executive functions (5, 8).

Obstructive sleep apnea (OSA) is characterized by episodes of partial or complete obstruction of the upper airways, while this phenomenon can be associated with hypoxia, hypercapnia and changes in sleep architecture (13, 14). The prevalence of OSA in the pediatric population is reported at 1-5.7%. OSA is a frequently occurring disease in preschool age precisely because of the growth of adenoid tissue, but it is not an exception even in older children (15).

The connection between OSA and cognitive or behavioral dysfunction can be explained by changes in sleep, hypercapnia and intermittent hypoxia, which can trigger stress at the cellular and biochemical level, leading to homeostatic imbalance and changes in the viability of neurons and glia in certain areas of the brain, especially in the prefrontal cortex. which is also confirmed by animal studies (16, 17). Another explanation for the impact of OSA on cognition and behavior is that OSA significantly disrupts sleep quality. Meta-analytic studies point to findings that shorter sleep time is associated with children's behavioral problems as well as poor academic performance. The literature dealing with the pathophysiology of OSA also points to the fact that these children showed more agitation, aggression and hyperactivity, lower scores on academic tests, as well as impaired attention, intelligence, memory, learning and behavior (18, 19).

In the literature, we can find several studies that also tested the global cognitive performance of patients suffering from OSA using the Differential Ability Scale (DAS), the Wechsler Intelligence Scale for Children-III (WISC -III), and the Stanford-Binet Scale (SBS). In the analysis of the results, Hunter and colleagues (20) point to a significant deterioration of the overall cognitive performance of children with OSA compared to a healthy control group, but still within the normal range, specifically in a more severe form of OSA [mean±SD, 92.40±12.4] and milder OSA [mean±SD, 98.02±15.30]) . However, Halbower et al. (21) showed that some children with OSA had IQ below normal.

The authors of a study focused on the reversibility of cognitive performance after adenotonsillectomy in patients with OSA found that neurocognitive capacity is impaired in all children suffering from OSA. However, most functions improve to the level of the control group, suggesting that impaired neurocognitive functions are mostly reversible, at least 3 to 10 months after adenotonsillectomy (22).

Some studies point to the presence of IQ impairment, while others suggest smaller differences in performance. Thus, we can assume that although OSA reduces overall cognitive performance, in most cases it remains within the normal range or at the lower limit of the norm (9, 18, 19).

Several studies by foreign authors dealt with the evaluation of memory performance using different methods focusing on short-term or working memory and long-term declarative memory. No deterioration in verbal short-term memory was demonstrated (22), while the study by Halbover et al. (21) reported impaired verbal working memory in children with OSA, but no deficit in visuospatial working memory or verbal learning ability (California Verbal Learning Test for Children). According to other researches, there was deterioration in visual and verbal declarative memory (9).

The OSA group also performed significantly lower on tests of executive function, including verbal working memory and verbal fluency. However, functions reported to be impaired in adult sleep apnea (ie, problem solving and planning, inhibitory control, sustained attention, and vigilance) were not impaired in children with severe obstructive sleep apnea (9).

The relationship between high BMI and cognitive performance is less clear in children and adolescents than in adults (23). In adults, lower weight is associated with better cognitive performance (24, 25). However, with a high risk of comorbid complications in obesity such as OSA, the risk of impaired cognition in children also increases (26, 27). Several studies suggest that obesity is a significant factor contributing to the deterioration of cognitive performance in patients with OSA (26, 27).

In studies looking at OSA and the comorbidity of obesity, the authors found that BMI correlated with reduced overall IQ. Specifically, research in children and adolescents with overweight/obesity and a comorbid sleep disorder has demonstrated impaired neurocognitive performance. Thus, overweight/obese children may be at increased risk for neurocognitive deficits when they develop symptoms of dg. OSA and other sleep disorders (16, 27, 28, 29).

Childhood is an important period for brain development and maturation, and the hippocampus is a key subcortical structure involved in memory consolidation during wakefulness and sleep. Several studies concluded that OSA in children was associated with different brain structure assessed by magnetic resonance imaging (MRI) (28, 29) . For example, results from children with OSA showed less gray matter volume in areas related to cognitive control (i.e., frontal and prefrontal cortices, parietal cortices, temporal lobes, and brainstem) than their peers without any sleep disorder (28, 29). As for children with obesity, they have been shown to be more prone to OSA than children of normal weight and often have impaired development of gray and white matter (17, 27, 28, 29).

However, several studies point to findings that symptoms of sleep-disordered breathing can impair academic performance in children beyond a diagnosis of obesity alone. Studies of OSA and sleep disturbances, elevated BMI, and cognitive performance have found multidirectional relationships, with any of these factors serving as a precursor, mediator, or outcome (16, 17, 27, 28).

Materials and methods

The aim of our work was to evaluate and compare the performance in tests of cognitive functions in the

clinical and control group. The clinical group consisted of pediatric patients who were diagnosed with OSA based on a standard all-night polysomnographic examination performed in the Children's Sleep Laboratory of the University Hospital in Martin. The examination was performed for patients with a history of suspected apnoeic pauses, difficulty breathing and snoring. At the same time, the comorbidity of obesity is present in these patients, which was detected by determining their body mass index (BMI) in relation to age and gender. The control group consisted of healthy individuals in whom the presence of acute as well as chronic diseases (cardiovascular, endocrinological, neurological, respiratory, metabolic, immunodeficiency, presence of genetic syndrome or sleep disorders, or psychiatric comorbidities) was ruled out and had a BMI within the norm. Data collection took place at the Paediatric department of Jessenius Faculty of Medicine and University Hospital in Martin in the years 2019 – 2023. All patients had informed consent signed by their legal representative. The study was approved by the Ethical Committee of Jessenius Faculty of Medicine.

The study included 41 probands (20 girls and 21 boys) who were divided into 2 groups: patients suffering from OSA and with an increased BMI index corresponding to obesity (23 probands) and a healthy control group (18 probands) with a normal BMI. The clinical group consisted of 23 patients (53%), of which 11 were female and 12 were male, with an average age of 11.8 years. The control group consisted of 18 participants (43%), of which 9 were female and 9 were male.

All probands were administered a neuropsychological examination by a psychologist consisting of a battery of five tests compiled for the purposes of covering the spectrum of basic cognitive functions such as memory, attention and executive functions. The methods used were Slovak versions of Rey-Osterrieth Complex Figure (Parts: Copy and Reproduction, abbr. ROCF C and ROCF R), Trail Making Test (part A and B, abbr. TMT A and B), Verbal Learning Memory Test (total score and delayed reproduction, abbr. AVLT TS and AVLT DR), Digit Span Test (total score, abbr. DST TS) and Numerical square (sten values M, M1/M2 and average time of M,M1,M2 in seconds, abbr. NS M sten, M1/M2 sten, M, M1/M2 time/s). The measured results of the neuropsychological examination were evaluated according timeto the manual of the individual tests and available standards. The individual results were then compared between the clinical groups and the results of the control group of probands. These data were statistically processed and evaluated.

Results

Based on the results of the statistical analysis, we can state that in the tests ROCF Reproduction ($t=-3.46$; $p<0.01$), AVLT TS ($t=-4.24$; $p<0.001$) and PTU ODR ($t=-7.29$; $p<0.001$) that mapped the performance in the area of memory, the probands of the control group achieved a significantly higher average score and therefore a better result than the probands of the clinical group. In the Digit span test, statistically significant differences between the memory performance of the monitored groups were not confirmed ($t=-0.44$; $p>0.05$) (Tab. 1).

Table 1. Performance in memory tests. t-test: clinical group with OSA and control group.

	Mean ± SD OSA (n=23)	Mean±SD Control group (n=18)	p
ROCF R TS	16.826±6.102	23.444±6.041	0.0013
AVLT TS	40.608±4.988	47.388±5.203	0.0001
AVLT DR	6.652±1.584	10.388±1.685	0.0000
DST TS	12.304±3.308	12.777±3.540	0.6616

SD – standard deviation, n – number, p – level of statistical significance, ROCF R HS – Rey-Osterrieth of the complex figure, part Reproduction, total score, AVLT TS – Verbal Learning Memory Test, total score, AVLT DR – Verbal Memory learning test, delayed reproduction, DST TS – Digit span test, total score

In the case of examining attention, the results showed statistically significant performance differences in all monitored tests except the TMT B and time/s in part M1 of Numerical square, in which the results of the probands of both groups did not differ significantly ($p>0.05$). The statistically largest differences (statistically highly significant differences) were observed in the tests of NS M sten ($U=57.50$; $p<0.001$) and NS M1/M2 sten ($t=-3.66$; $p<0.001$). Highly significant differences were confirmed in the TKF R ($t=-3.46$; $p<0.01$) and TKF K ($t=-2.99$; $p<0.01$) tests. In the case of the other tests, there were differences at the 5% level – TMT part A ($U=130.50$; $p<0.05$), CS M time/s ($U=127.00$; $p<0.05$) and CS M2 time/s ($U=111.00$; $p<0.05$). In all the mentioned tests, the probands of the control group achieved significantly better results (Tabs 2, 3).

Table 2. Performance in attention tests: t-test: clinical group with OSA and control group.

	Mean±SD OSA (n=23)	Mean±SD Control group (n=18)	p
ROCF R HS	16.826±6102	23444±6041	00013
NS M1/M2 sten	4739±2416	7055±1304	00007

SD – standard deviation, n – number, p – level of statistical significance, ROCF R TS – Complex figure test, part Reproduction, total score, NS M1/M2 sten – value of sten in the Numerical square

The results showed that in the TMT test part A, probands of the control group achieved a significantly better result (significantly better time) compared to the clinical group ($U=130.5$; $p<0.05$). In the second part of the test – TMT part B, statistically significant differences between the performance of the monitored groups in the area of executive functions were not confirmed ($U=135.00$; $p>0.05$) (Tab. 4).

Table 3. Performance in attention tests: Mann-Whitney U test: clinical group with OSA and control group.

	Rank Sum OSA (n=23)	Rank Sum Control (n=18)	U	Z	p
ROCF TS	369.000	492.000	93.000	**–2.991**	**0.0027**
TMT A time/s	559.500	301.500	130.500	**2.002**	**0.0452**
TMT B time/s	555.000	306.000	135.000	1.880	0.0599
NS M sten	333.500	527.500	57.500	**–3.992**	**0.0000**
NS M time/s	563.000	298.000	127.000	**2.093**	**0.0362**
NS M1 time/s	539.500	321.500	150.500	1.472	0.1408
NS M2 time/s	579.000	282.000	111.000	**2.512**	**0.0119**

U, Z – test statistic, p – level of statistical significance, n – number, ROCF K TS – Rey-Osterrieth Complex Figure, part Copy, total score, TMT A – Trail Making test, part A, time in seconds , TMT B – Trail Making test, part B, time in seconds, NS M sten – sten value of Numerical square part M, NS M, M1, M2 time/seconds – average time of M,M1,M2 parts in seconds

Table 4. Performance in tests of Executive functions: Mann–Whitney U test: clinical group with OSA and control group.

	Rank Sum OSA (n=23)	Rank Sum Control (n=18)	U	Z	p
TMT A time/s	559.500	301.500	130.500	**2.002**	**0.0452**
TMT B time/s	555.000	306.000	135.000	1.880	0.0599

U, Z – test statistic, p – level of statistical significance, n – number, TMT A time/s – Trail making test, part A, time in seconds, TMT B time/s – Trail making test, part B, time in seconds

Discussion

Obstructive sleep apnea in children is a multifactorial disease caused by anatomical and functional pathophysiological and, last but not least, genetic mechanisms. Secondary consequences of OSA negatively affect the cardiovascular and metabolic systems, as well as neurocognitive functions, which are responsible for cognitive and behavioral difficulties.

Behavioral and neurocognitive changes are frequent, especially in school-aged children with OSA. The mechanism by which OSA leads to cognitive impairment is probably multifactorial and based primarily on dysfunction of the prefrontal cortex, which is related to chronic nocturnal hypoxia, sleep fragmentation, and sleep deprivation. It is described that the occurrence of these disorders in school-age children with OSA is up to 3 times higher compared to the healthy population of children. These symptoms in pediatric patients with OSA are similar to those in children with chronic sleep deprivation (12).

Obese pediatric patients belong to a high-risk group for the possible development of serious sleep-disordered breathing. Obesity, the percentage of which is constantly increasing worldwide, but also in Slovakia in the child population, not only causes but also worsens sleep-disordered breathing. their consequence is poor quality, interrupted sleep, ventilation disorders during sleep, changes in respiratory gases, which subsequently lead to disorders of cell regeneration, metabolism or cardiovascular diseases, and these again lead to obesity. Currently, obesity is also considered one of the causal factors in the development of OSA in the pediatric population.

There are assumptions that high BMI contributes to impairment of cognitive functions, but these findings are less consistent in the population of children and adolescents (23, 24, 25). However, in connection with the occurrence of OSA, we find findings that the risk of impaired cognition is more significant (26, 27). The treatment of childhood obesity is very important both as a prevention of the occurrence of comorbid problems, but also to improve the clinical condition of patients with OSA and, last but not least, to preserve optimal cognitive development in childhood (30).

Neuropsychological examination is a very necessary and useful part of the differential diagnosis of psychiatric and neurological diseases, but it also has a wider use in pediatric clinical practice. Such an examination provides both general and specific information about the current state of the patient's cognitive functions as well as the extent of any cognitive deficit. The aim of the neuropsychological examination is mainly a detailed description of the current behavioral and functional state of the child, specification of dysfunctions and their depth, determination of strengths as a starting point for rehabilitation.

In our research groups, we examined memory performance using Rey-Osterreith complex figure tests, the results of which tell us about the performance of non-verbal memory and visuospatial memory. We hypothesized that there is a statistically significant negative effect of OSA and the presence of obesity according to BMI on memory performance and thus the results will be worse in this test than in the control group. In the TKF test part of reproduction ($t=-3.46$; $p<0.01$), probands of the control group achieved a significantly higher average score, i.e. significantly better result in non-verbal and visual-spatial memory compared to probands diagnosed with OSA and obesity. Subsequently, we tested memory performance with the Verbal Memory Learning Test, which focuses on performance in the area of short-term and long-term verbal memory, and in this test, our assumptions, on the basis of which we once again expected worse performance in our research sample, were confirmed. In the individual parts of the verbal learning memory test – AVLT TS ($t=-4.24$; $p<0.001$) and AVLT DR ($t=-7.29$; $p<0.001$), the probands of the control group achieved a significantly higher average score (i.e. a significantly better result in the area of short-term and long-term verbal memory). In the last test, the Digit span test, which focuses primarily on short-term verbal memory and working memory, statistically significant differences between the performance of these two groups were not confirmed ($t=-0.44$; $p>0.05$). Although our findings are different and the differences were not confirmed in all memory tests used, our findings largely agree with foreign authors and point to the possibility of a memory deficit in patients with this type of disease.

As the second cognitive domain, we tested attention by comparing the results in the Rey-Osterreith complex figure tests, the Trail making test and its two parts, and the numerical square. The results of the probands showed statistically significant performance differences in all monitored tests, except for the TMT part B and NS M1 tests, in which the results of the probands of both groups did not differ significantly ($p>0.05$). Statistically, the biggest differences were found in the tests Numerical square M sten ($U=57.50$; $p<0.001$) and Numerical square M1/M2 ($t=-3.66$; $p<0.001$). Highly significant differences were confirmed in the ROCF R ($t=-3.46$; $p<0.01$) and ROCF C ($t=-2.99$; $p<0.01$) tests. In the case of the other tests, there were differences at the 5% level – TMT part A ($U=130.50$; $p<0.05$), NS M time/s ($U=127.00$; $p<0.05$) and CS M2 time/s ($U=111.00$; $p<0.05$). In all the mentioned tests, the probands of the control group achieved significantly better results, which indicates the possibility of the presence of weakened performance in the area of attention.

In this sample of patients, we also focused on performance in the area of executive functions, where information about their performance was provided by the Trail making test and its two versions, but also partially by the Verbal Memory Learning Test, which also tests the ability of working memory. Probands achieved a significantly better time in the Trail making test only in part A, in part B statistically significant differences between the performance of the monitored groups in the area of executive functions were not confirmed ($U=135.00$; $p>0.05$). Part B, which tests planning ability and cognitive flexibility, is mainly focused on performance in the area of executive functions. In the verbal learning memory test, we can observe significantly better results in the control group, and these results do not only provide us with information about memory performance, but partially also about performance in the area of executive functions.

Conclusion

In our study, we analyzed performance in cognitive domains – memory, attention and executive functions in pediatric patients with OSA and obesity. At the same time, we also tested these cognitive domains in a healthy control group and then compared the results. Our results point to the significant risk of neurocognitive deficits in OSA in childhood. In addition to the proper management and treatment of these patients in the children's sleep laboratory, an examination of cognitive functions is also a suitable supplement to comprehensive care. The examination is not economically demanding and can provide a lot of valuable data at the beginning of treatment, in complex patient care or in rehabilitation.*

*__Compliance with Ethics Requirements:__ The authors declare no conflict of interest regarding this article. The authors declare, that all the procedures and experiments of this research respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008 (5), as well as the national law.

__Conflict of interest:__ The authors declare no conflict of interest.

__Informed consent:__ Informed consent was obtained from all individual participans included in the study.

References

1. SPRUYT K. Neurocognitive Effects of Sleep Disruption in Children a Adolescents. Child and adolescent psychiatric clinics of North America, 2021, 30 (1), 27–45.
2. TORRES-LOPEZ LV, Cadenas-Sanchez C, Migueles JH, et al. Does sleep-disordered breathing add to impairments in academic performance and brain structure usually observed in children with overweight/obesity? Eur J Pediatr 2022, 181: 2055–2065.
3. JOHNSON EO, CHILCOAT HD, BRESLAU N. Trouble sleeping and anxiety/depression in childhood. Psychiatry Res 2000, 94 (2): 93–102.
4. OKANO K, KACZMARZYK, JR., DAVE N, et al. Sleep quality, duration, and consistency are associated with better academic performance in college students. Npj Sci Learn 2019, 4: 16.
5. ORCHARD F, GREGORY AM, GRADISAR M, REYNOLDS S. Self-reported sleep patterns and quality amongst adolescents: cross-sectional and prospective associations with anxiety and depression. J Child Psychol Psychiatry 2020, 61 (10, 11): 26–37.
6. SADEH A, GRUBER R, RAVIV A. The effects of sleep restriction and extension on school-age children: what a difference an hour makes. Child Dev 2003, 44–55.
7. STORES G. Aspects of sleep disorders in children and adolescents. Dialogues Clin Neurosci 2009, 11: 81–90.
8. BUCKS RS, OLAITHE M, EASTWOOD P. Neurocognitive function in obstructive sleep apnoea: A meta-review. Respirology 2013, 18: 61–70.
9. DA SILVA GUSMÃO CARDOSO T, et al. Cognitive and behavioral effects of obstructive sleep apnea syndrome in children: a systematic literature review. Sleep medicine 2018, 46: 46–55.
10. GREGORY AM, VAN DEN ENDE J, WILLIS TA, VERHULST FC. Parent-reported sleep problems during development predicts self-reported anxiety/depression, attention problems and aggression later in life. Arch Ped Adolesc Med 2008, 162 (4): 330–335.
11. GREGORY S. Aspects of sleep disorders in children and adolescents, Dialogues in Clinical Neuroscience 2009, 11 (1): 81–90.
12. VITELLI O, et al. Impact of obesity on cognitive outcome in children with sleep-disordered breathing. Sleep Medicine 2015, 16 (5): 625–630.
13. SUROVIAKOVÁ S, ŠUJANSKÁ A, ĎURDÍK P, BÁNOVČIN P. Centrálne spánkové apnoe u detí: Central sleep apnea in children. Lekársky obzor 2016, 65 (11): 433–437.
14. ŠUJANSKÁ A. Vplyv obštrukcie horných dýchacích ciest na spánok u detí. Dizertačná práca, Klinika detí a dorastu, Jesseniova lekárska fakulta, Univerzita Komenského v Bratislave. Martin 2014, 62–63.
15. DAYYAT E, et al. Obstructive sleep apnea in children: relative contributions of body mass index and adenotonsillar hypertrophy. Chest 2009, 136 (1): 137–144.
16. BEEBE DW, et al. The association between sleep disordered breathing, academic grades, and cognitive and behavioral functioning among overweight subjects during middle to late childhood. Sleep 2010, 33 (11): 1447–1456.

17. BEEBE DW, GOZAL D. Obstructive sleep apnea and the prefrontal cortex: towards a comprehensive model linking nocturnal upper airway obstruction to daytime cognitive and behavioral deficits. Journal of Sleep Research 2002, 11: 1–16.
18. BLUNDEN S, et al. Cognitive and behavioural performance in children with sleep-related obstructive breathing disorders. Sleep medicine reviews 2001, 5 (6): 447–446.
19. O'BRIEN LM, et al. Neurobehavioral implications of habitual snoring in children. Pediatrics 2004, 114 (1): 44–49.
20. HUNTER SJ, GOZAL D, SMITH DL, et al. Effect of sleep-disordered breathing severity on cognitive performance measures in a large community cohort of young school-aged children. Am J Respir Crit Care Med 2016, 194 (6): 739–747.
21. HALBOWER AC, et al. Childhood Obstructive Sleep Apnea Associates with Neuropsychological Deficits and Neuronal Brain Injury. PLoS Med 2006, 3 (8).
22. FRIEDMAN BC, HENDELES-AMITAI A, KOZMINSKY, E, et al. Adeno- tonsillectomy improves neurocognitive function in children with obstructive sleep apnea syndrome. Sleep 2003, 26 (8): 999–1005.
23. ANDERSON YC, KIRKPATRICK K, DOLAN GMS, et al. Do changes in weight status affect cognitive function in children and adolescents with obesity? A secondary analysis of a clinical trial. BMJ Open 2019, 9: 021586.
24. VERONESE N, FACCHINI S, STUBBS B, et al. Weight loss is associated with improvements in cognitive function among overweight and obese people: A systematic review and meta-analysis. Neurosci Biobehav Rev 2017, 72: 87–94.
25. ZB Y, HAN SP, CAO XG, et al. Intelligence in relation to obesity: a systematic review and meta-analysis. Obes Rev 2010, 11: 656–670.
26. XU J, WANG X, QIAN C, HAN W, LI X, DING Y. Exploration of the Association Between Obesity and Cognitive Impairment in Chinese Children with Mild or Moderate Obstructive Sleep Apnea-Hypopnea Syndrome. J Pediatr Nurs 2020, 53: 36–40.
27. KOHLER M, KENNEDY D, MARTIN J, et al. The influence of body mass on long-term cognitive performance of children treated for sleep-disordered breathing. Sleep Medicine 2018, 51: 1–6.
28. ŠUJANSKÁ A, ĎURDÍK P, BÁNOVČIN P. Spánok a dieťa. Pediatria 2015, 10 (2): 77–80.
29. ŠUJANSKÁ A, ĎURDÍK P, RABASCO J, et al. Surgical and non -surgical therapy of obstructive sleep apnea syndrome in children. Acta Med 2014, 57 (4): 135–141.
30. ŠUTVAJOVÁ D, IGAZ M, KVAŠŠAYOVÁ J, et al. Liečba spánkových porúch dýchania neinvazívnym pozitívnym pretlakom (NIV) - naše skúsenosti. Ces-slov Pediat 2022, 77 (6): 345–352.

Do redakcie došlo 28. 5. 2024.

Address for correspondence:
Anna Durdikova MD, PhD.
Jessenius Faculty of Medicine CU
Paediatric department
Kollarova 2
036 01 Martin
E-mail: *annasujanska@gmail.com*