

IMPROVEMENTS IN THE DIAGNOSIS OF OBSTRUCTIVE SLEEP APNEA IN CHILDREN

Luminița Rădulescu¹, Cristian Mârțu^{1*}, Dan Mârțu¹, Horia Chiriac², Sorin Corodeanu²

1. University of Medicine and Pharmacy “Gr. T. Popa”, Iași, România;

2. National Institute of Research and Development for Technical Physics, Iași, România;

Abstract

Obstructive sleep apnea in children is due to adeno-tonsillar hypertrophy, in the majority of cases – a common condition in children that can be cured by the ENT surgeon by a relatively safe surgery. The final diagnosis of Obstructive sleep apnea is possible with polysomnography. This test is difficult to be performed in small children due to the contact sensors that are displaced during the agitated sleep of a child with sleep apnea. We propose a quasi noncontact sensor to be used to record the respiratory movements of children during sleep. The preliminary tests show that the method might be applicable in the clinical settings.

Keywords: *apnea, children, surgery, recording, sensor*

***Corresponding author:** *Cristian Mârțu, MD, MBA, AUD. Teaching Fellow*

ENT Department University of Medicine and Pharmacy “Gr. T. Popa” Iasi

Clinical Rehabilitation Hospital Iasi, Romania

E-mail: cristimartu@gmail.com

Introduction

In 1836 Charles Dickens in The Pickwick Paper made the first description of obstructive apnea during sleep to a character named Joe.

In 1889 Hill notes that snoring and restless sleep during the night are causes of retardation in the child’s neuropsychological development[1], but had to pass 100 years before the publication of a study on obstructive sleep apnea (OSA)[2].

It is now recognized that OSA is one of the most common respiratory disorders. If the prevalence of this condition was initially estimated at 1-3% of normal children[3-7], more recent studies indicate a prevalence of 5-6% [8,9] with a peak at ages of 2 to 8

years, when the lymphoid tissue hyperplasia in the pharynx reach a critical size.

Untreated OSA in children can lead to significant morbidity [10-14]. There is some evidence indicating that alteration of sleep architecture by repeated episodes of hypoxia followed by awakening may affect the child’s behavior leading to aggression, hyperactivity, decreased alertness, anxiety and impaired neurocognitive functions [15,16].

One of the most controversial issues is regarding the existence of snoring unaccompanied by significant changes in gas concentrations in the blood. Recent studies describing the presence of neurological and behavioral deficits in children indicate that

primitive snoring is not as harmless as much

as was initially thought [17].

Material and Method

The gold standard for OSA diagnosis is polisomnography.

Performing polisomnography in children might be extremely challenging because of the contact sensors that are thrown away during the agitated sleep of a child.

To overcome this drawback together with a team from the Institute of Technical Physics we imagined a quasi noncontact sensor to register the respiratory movement of children during sleep.

The first product was a mattress with a quasi noncontact sensor in the form of a

wire. The sensor was tested on a group of healthy people and the results were published[33].

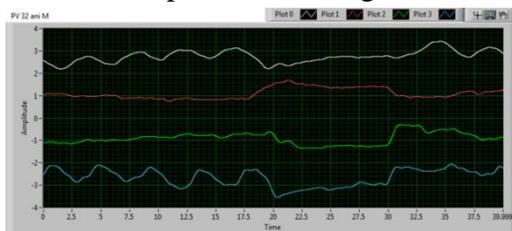
The problems regarding this sensor were related to the fact that children are moving a lot during the sleep and can lose the contact with a sensor that is fixed in the mattress. The solution was to improve the recording conditions using more than a sensor. We added three more sensors.

The new mattress with the four quasi noncontact sensors was tested on ten volunteer healthy subjects.

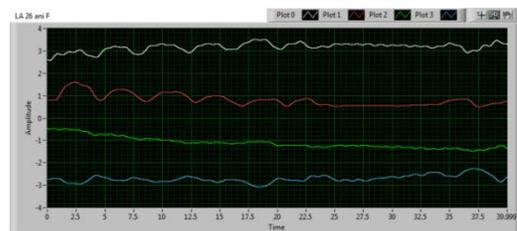
Results

We have obtained 10 graphics from which 2 were compromised during the

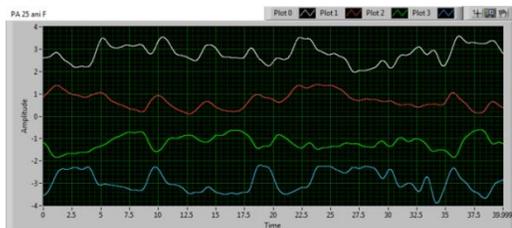
recording process. The eight remaining charts (fig. 1) were analysed.



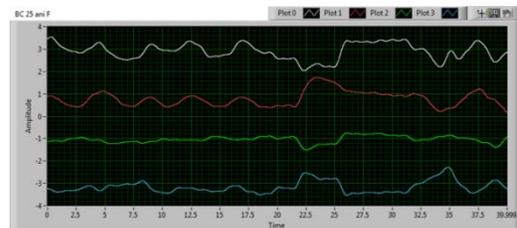
Patient 1



Patient 2



Patient 3



Patient 4

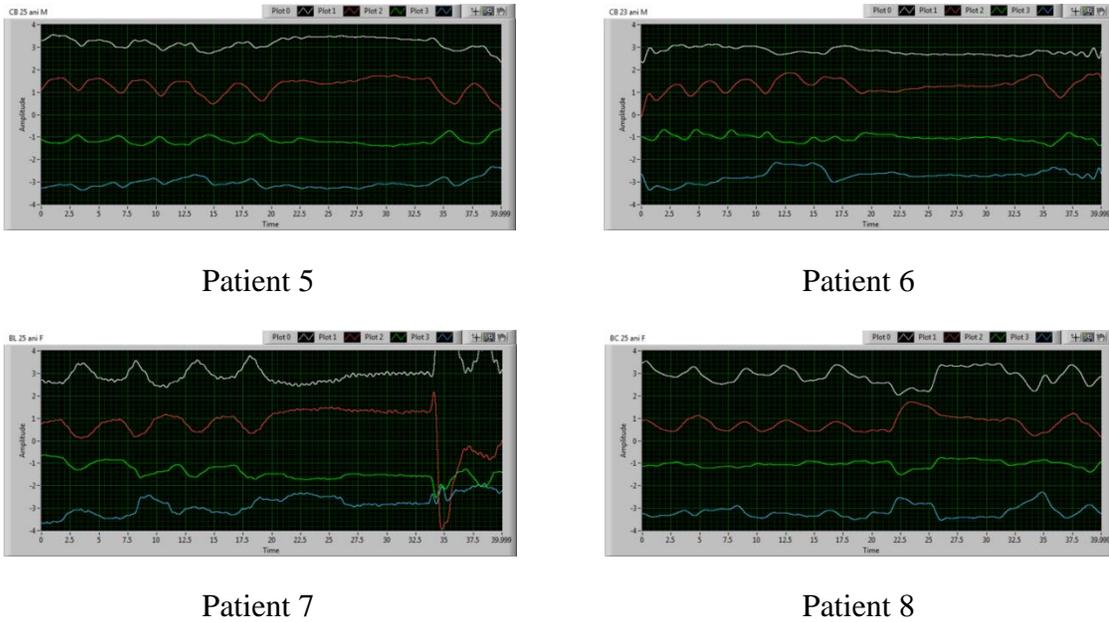


Fig. 1 Normal respiratory movements recorded with the mattress with four wires sensor

One from the registered line was identical with the one obtained after using just one sensor placed in the fifth intercostal

space (fig.2) and so the charts could be interpreted using the same parameters[33].

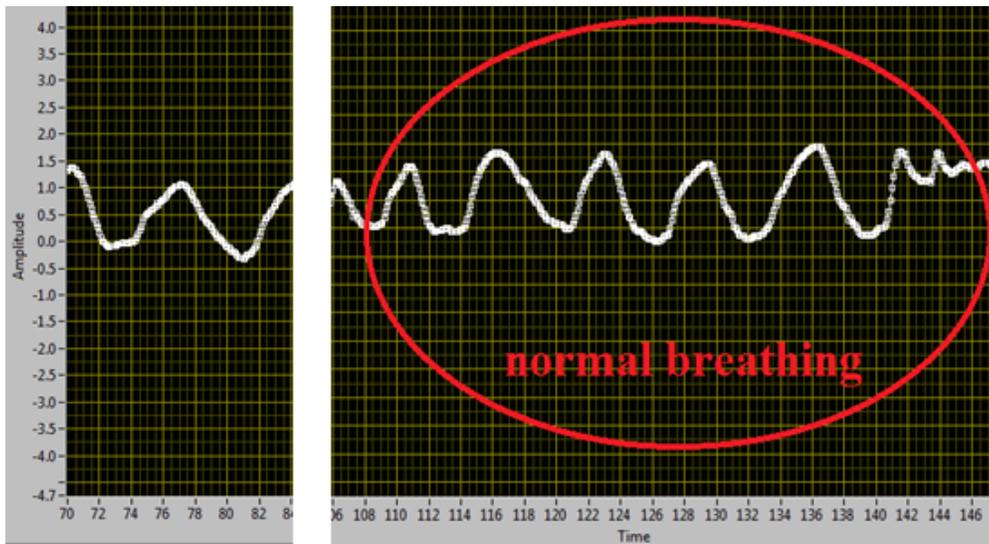


Fig. 2 Normal respiratory movements recorded with the mattress with one wire sensor

Discutions

During sleep the upper airway (UAW) resistance increases [18] with the narrowing of the air tract caused by the decrease in tone of pharyngeal dilators [19]. The pressure in UAW during inspiration becomes negative under normal conditions.

The negative pressure that appears during inhale in the UAW is counterbalanced by the activity of the dilator muscles of the pharynx [20], given that, due to pharyngeal lymphoid tissue hyperplasia (a condition that is very common in children) the resistance in the UAW is raised so that the UAW can collapse [21].

Pharyngeal lymphoid tissue grows in size faster than the bones in children of 3 to 5 years of age leading to a consequent narrowing of the respiratory tract [22]. MRI imaging studies have shown that children with OSA have pharyngeal lymphoid tissue larger than children without OSA [23].

Also it was demonstrated that there is a linear relationship between the tonsil's size and the severity of the OSA [23]. Many children have tonsillar hypertrophy but relatively few have OSA in the same time. The pathophysiology of OSA in children results from the complex interplay between the physiological factors (ie. neuromuscular control) and anatomical factors (structure of UAW and size of the tonsils and of adenoids). There are no known reasons for overgrowth of the tonsils in some children leading to the occurrence of respiratory disorders during sleep. On the other hand, in the child there were not yet been established normal respiratory parameters in sleep and also there was not yet achieved a correlation

between the apnea-hypopnea index and the emergence of pathological events. In adults even mild OSA is associated with daytime sleepiness[24] and with an increased cardiovascular risk [25].

It is possible that due to immature nervous system, the impact of sleep disorder breathing in children is even more important. Most cases of OSA in children are effectively treated by adenotonsillectomy leading to restoration of normal breathing, improving growth, sleep and behavior [26].

The risk of respiratory complications after adenotonsillectomy in the pediatric population is 1% [27,28]. In children in whom the SaO₂ drops below 80% the risk of complications may increase to 50% [29].

Using masks with continuous positive pressure in children is required in those that had not the expected results after adenotonsillectomy or in those in which the OSA is due to other causes [30].

Depending on each case may be recommended other treatment methods like nasal steroids in children with allergic rhinitis [31], craniofacial surgery in children with craniofacial malformations [32].

After adenotonsillectomy, children with OSA show a significant overall improvement in quality of life parameters.

Given the fact that the diagnosis of OSA is difficult in children, identification of the criteria for the diagnosis of obstructive sleep apnea in children is important. Furthermore, the exploration polysomnography is available for children only in some medical centers.

Conclusions

Considering that the child is moving all the time during sleep, the new mattress with the four wires quasi noncontact sensors will cover all the area on which the child will lay down during sleep. By analyzing the recording of monitoring through more sensors, the breathing pattern will be more

accurate and the data interpretation more reliable in terms of prediction. The mattress opens new diagnostic possibilities in the children's sleep pathology and might be a very useful tool in the early prediction of respiratory failure.

Author contribution: All authors made equal contribution to the paper, to that of first authors.

Bibliography:

1. Nixon GM, Brouillette RT.: Sleep Apnoea & Paediatric obstructive sleep. *Thorax* 2005; 60: 511-516
2. Brouillette RT., Fernbach SK, Hunt EC. Infants and obstructive sleep apnea in children. *J Pediatr* 1982; 100: 31-40.
3. Lu LR., Peat JK., Sullivan EC.. Snoring in preschool children: prevalence and association with nocturnal cough and asthma. *Chest* 2003; 124: 587-593.
4. Shin C., Joo S., Kim J., Kim T. prevalence and correlates of habitual snoring in high school students. *Chest* 2003; 124: 1709-1715.
5. Urschitz MS, Guenther A, Eltner S et al. Risk factors and natural history of habitual snoring. *Chest* 2004; 126: 790-800.
6. Castronovo V., Zucconi M., Nosetti L. et al. Prevalence of habitual snoring and sleep disordered breathing in preschool-aged children in year Italian community. *J Pediatr*. 2003; 142: 377-382.
7. Rosen C., Larkin E, Kirshner H, et al. Prevalence and risk factors for sleep disordered breathing in 8 to 11 year old children: association with race and prematurity. *J Pediatr*. 2003; 142: 383-389.
8. Guilleminault C., Lee JH, A. Chan Pediatric obstructive sleep apnea syndrome. *Arch Pediatr Adolesc Med* 2005; 159: 775-785.
9. Kennedy JD, Waters KA. Investigation and upper-airway obstruction and treatment: I. Childhood Sleep Disorders *MJA* 2005; 182 (8): 419-423.
10. Gozal D., Pope DW Jr. Snoring During early childhood and academic performance at ages thirteen to Fourteen years. *Pediatrics* 2001; 107: 1394-1399.
11. Gozal D., Morbidity if obstructive apnea in children: Facts and theory. *Sleep breath*.2001; 5: 35-42.
12. Amin RS, Kimball TR, Bean JA, et al. Left ventricular hypertrophy and abnormal ventricular geometry in children and adolescents with obstructive sleep apnea. *Am J Respir Crit Care Med*. 2002 ", 165: 1395-1399.
13. Amin RS., Carroll JR, Jeffries JL, et al. Twenty-four-hour ambulatory blood pressure in children with sleep-breathing Disorders. *Am J Respir Crit Care Med*. 2004; 169: 950-956.
14. Amin RS, Kimball TR, Kalra M et al. Left ventricular function in children with sleep-breathing disorders. *Am J Cardiol*. 2005; 95: 801-804.
15. Kennedy JD., S Blunden, Hirte C, et al. Reduced neurocognition in children snore. *Pediatr Who Pulmonol* 2004; 37: 15-16.
16. Gozal D., O'Brien LM. Snoring and Sleep Apnoea in children obstructive: Why shouldn't we treat? *Paediatr Respir Rev* 2004; 5: S371-376.
17. Urschitz MS, Guenther A, Eggebrecht E, et al. Snoring, intermittent hypoxia and academic performance in primary school children. *Am J Resp Crit Care Med* 2003; 168: 464-468.
18. Wiegand DA, Zwillich CW, White DP. Collapsibility of the human upper airway During normal sleep. *J Appl Physiol* 1989; 66: 1800-8.
19. Wiegand DA, Latz B Zwillich CW, et al. Geniohyoid muscle activity in normal men During wakefulness and sleep. *J Appl Physiol* 1990: 69: 1262-9.
20. Remmers JE, WJ DeGroot, Sauerland EK, et al. Pathogenesis of upper airway occlusion During sleep. *J Appl Physiol* 1978; 44: 931-8.
21. Pagella F, De Amici M, Pusateri A, Tinelli G, Matti E, Benazzo M, Licari A, Nigrisoli S, Quaglini S, Ciprandi G, Marseglia GL. Adenoids and clinical symptoms: Epidemiology of a cohort of 795 pediatric patients. *Int J Pediatr Otorhinolaryngol*. 2015 Dec;79(12):2137-41. doi: 10.1016/j.ijporl.2015.09.035. Epub 2015 Oct 8.
22. Jeans WD, Fernando DC, Maw AR, et al. A longitudinal study of the growth of the nasopharynx and Its contents in *Br J Pediatr* normal children 1998; 54: 117-21.

23. Fernbach SK., Brouillette RT, Riggs TW, et al. Radiologic evaluation of tonsils and adenoids in children with obstructive sleep apnea: plain films and fluoroscopy. *Pediatr Radiol* 1983; 13: 258-65.
24. Young T, Palta, M, J Dempsey et al. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993; 328: 1230-5.
25. Peppard PE, Young T, Palta M, et al. Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med* 2000; 342: 1378-1384.
26. Scholle S, Zwack G. arousal and obstructive sleep apnea syndrome in obstructive breathing abnormalities in Infants and children. *J Appl Physiol* 1996; 81: 2651-7.
27. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a Population health perspective. *Am J Crit Care Med*.
28. Rosen GM, Muckle RP, Mahowald MW, et al. Postoperative respiratory compromised in children with obstructive sleep apnea syndrome: can it be anticipated? *Peiatrics* 1994; 93: 784-8.
29. Wilson K., Lakheeran I., Morielli A., et al. Can assessment for obstructive sleep apnea help predict postadenotonsilectomy Respiratory Complications? *Anesthesiology* 2001; 96: 313-22.
30. McNamara F., Sullivan EC. Obstructive sleep apnea in Infants and Its nasal continuous positive airway management with pressure. *Chest* 1999; 116: 10-6.
31. Scadding GK. Cortisteroids in the treatment of pediatric allergic rhinitis. *J Allergy Clin Immunol* 2001; 108: S59-64.
32. Reuveni H., Simon T., Tal et al. Health care services utilization in children with obstructive sleep apnea syndrome. *Pediatrics* 2002; 110: 68-72.
33. Martu C., Cozma S., Corodeanu S., Chiriac H., Leata R., Radulescu L. Amorphous Ferromagnetic Materials used as Sensor in Monitoring Respiratory Movements. *Materiale Plastice* 52 (4), 612-614