Sleep Medicine 15 (2014) 76–82

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

Long-term changes in heart rate variability in elementary school–aged children with sleep-disordered breathing

Anna Vlahandonis [a], Stephanie R. Yiallourou [a], Scott A. Sands [a,b], Gillian M. Nixon [a,c], Margot J. Davey [a,c], Lisa M. Walter [a,1], Rosemary S.C. Horne [a,*,1]

[a] The Ritchie Centre, Monash Institute of Medical Research, Monash University, Melbourne, Australia
[b] Division of Sleep Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA
[c] Melbourne Children's Sleep Centre, Monash Children's Programme, Monash Medical Centre, Melbourne, Australia

ARTICLE INFO

Article history:
Received 26 April 2013
Received in revised form 21 June 2013
Accepted 24 June 2013
Available online 14 October 2013

Keywords:
Sleep disordered breathing
Pediatric
Heart rate variability
Autonomic dysfunction
Follow-up
Treatment

ABSTRACT

Objective: Sleep-disordered breathing (SDB) in adults and children has been associated with reduced heart rate variability (HRV) indicative of autonomic dysfunction, which in turn is associated with an increased risk for cardiovascular morbidity. However, the long-term effects of pediatric SDB that has either resolved or remains unresolved on HRV are unknown.

Methods: Forty Children with previously diagnosed SDB and 20 non snoring controls underwent repeat overnight polysomnography (PSG) four years after the original diagnosis. At follow-up, children aged 11 to 16 years were categorized into resolved (absence of snoring and obstructive apnea hypopnea index [OAHI] $\leqslant 1$) or unresolved (continued to snore or had an OAHI > 1) groups. HRV was assessed using power spectral analysis for each sleep stage.

Results: There were no group differences in age, sex or body mass index (BMI) *z* score. Both the resolved and unresolved SDB groups showed significant improvement in OAHI. The control, resolved, and unresolved groups all showed a significant reduction in total power ($P < .001$), low-frequency (LF) power ($P < .05$), high-frequency (HF) power ($P < .001$), and an increase in the LF/HF ratio ($P < .001$) from baseline to follow-up in all sleep stages.

Conclusions: HRV did not differ between non snoring children and children with resolved and unresolved SDB four years after initial diagnosis, concomitant with a significant reduction in OAHI in both SDB groups. All groups demonstrated a decrease in HRV from baseline to follow-up which may reflect an age-related phenomenon in these children.

Crown Copyright © 2013 Published by Elsevier B.V. All rights reserved.

1. Introduction

Sleep-disordered breathing (SDB) is a common condition in childhood. While all children with SDB snore, this disorder exhibits a spectrum of severity. Between 1% and 5% [1] of children have the most severe form, obstructive sleep apnea (OSA), which is characterized by repetitive and prolonged partial and/or complete episodes of airway obstruction during sleep, concomitant with intermittent hypoxia or sleep fragmentation. However, many more children (up to 35%) [2] are affected by primary snoring (PS), which is not associated with any gas exchange abnormalities or significant sleep disruption and is considered to be the mildest form of SDB. Unlike adults, the primary cause of childhood SDB is adenotonsillar hypertrophy, and surgical removal of the tonsils and adenoids is the first-line treatment.

The termination of an obstructive respiratory event during sleep is accompanied by marked surges in blood pressure (BP) and heart rate (HR), which has been reported in both adults [3] and children [4]. These surges occur due to an increase in sympathetic activity in response to hypoxemia or arousal from sleep [5]. However, this elevation in sympathetic activity is not isolated to the time of the event but can persist during periods without upper airway obstruction [5] and also into wakefulness [5,6]. Furthermore, studies in normotensive adults with OSA have suggested that increased sympathetic activity may precede the development of hypertension [7,8], a condition that is prevalent in adults with OSA and increases cardiovascular morbidity.

Heart rate variability (HRV), the analysis of fluctuations in heart rate, has become an increasingly popular and non invasive tool to assess the functioning of the autonomic nervous system [9]. It can be assessed using either time or frequency (spectral analysis) domain methods, though frequency domain measures are preferred

* Corresponding author. Address: The Ritchie Centre, Level 5, Monash Medical Centre, 246 Clayton Road, PO Box 5418, Clayton, Victoria 3168, Australia. Tel.: +61 3 95945100; fax: +61 3 95946811.
E-mail address: rosemary.horne@monash.edu (R.S.C. Horne).
[1] Denotes senior co-authorship.

1389-9457/$ - see front matter Crown Copyright © 2013 Published by Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.sleep.2013.06.023

for short-term recordings [9]. Spectral analysis of HRV separates the electrocardiogram (ECG) signal into its high-frequency (HF) and low-frequency (LF) components. HF power occurs at the respiratory frequency and is widely accepted as a measure of parasympathetic activity, while LF power is reflective of both parasympathetic and sympathetic activities [9–12]. Adults with OSA have been reported to show a reduction in vagal modulation of HRV together with an elevation in sympathetic tone [13,14]. Overall, adult studies have shown that SDB is significantly associated with lower HRV [15–17], which is concerning, as low HRV also has been associated with the development of hypertension [18], coronary artery disease [19–21] and a higher risk for all-cause mortality in survivors of myocardial infarction [22]. Furthermore, a study in adults has demonstrated that HRV also is altered in habitual snorers (without OSA) compared to healthy controls [8]. Studies assessing HRV in children with SDB also show evidence of autonomic dysfunction [23–27] demonstrating elevated LF power [25] and reduced HF power [28].

Longitudinal studies of HRV in children with SDB and the effects of treatment are few—only two have been performed thus far and these have been short-term studies (6 months–1 year) [29,30] and only included children treated for moderate/severe OSA. One additional study by Constantin et al. [31] assessed pulse rate and pulse rate variability after treatment, but again this was a short-term study (<1 year). To date, the long-term effects of childhood SDB on HRV are unknown. In addition, the HRV outcomes in children with milder disease such as PS, who often are not treated, also remain to be elucidated. Hence the aim of our study was to assess HRV in children with either resolved or unresolved SDB and non-snoring controls four years after initial diagnosis. We hypothesized that children with resolved SDB would show increased HRV indicated by increased parasympathetic activity, whereas children with unresolved SDB would exhibit the reverse.

2. Methods

Participation in our study was voluntary and no monetary incentive was offered. Written informed consent was obtained from parents and verbal assent from the children on the night of the study. Approval was granted by Southern Health and Monash University Human Research Ethics Committees.

2.1. Subjects and study design

Our study was a prospective follow-up study of children who were originally studied between July 2004 and December 2008 when they were aged 7 to 13 years. At baseline 155 children were studied, 116 of whom had been referred for assessment of suspected SDB and 39 of whom were non snoring control children recruited from the community. Our study began in 2009 and aimed to follow-up children after a time frame of four years. Therefore, children originally studied in 2004 were excluded, leaving 135 children ($n = 99$ SDB; $n = 36$ control) eligible to participate. Of those 135 children, 16 were not able to be contacted. The remaining 119 children were invited to participate in our study and those who agreed to participate underwent repeat overnight polysomnography (PSG) together with completion of questionnaires to assess their quality of life and sleep habits and quality. The sleep and respiratory outcomes from the follow-up study have been published [32].

2.2. Protocol

Prior to the PSG study, anthropometric measures including height and weight were recorded and body mass index (BMI) was calculated. BMI was converted to BMI z scores [33]. Participants underwent routine overnight pediatric PSG in the Melbourne Children's Sleep Centre using a commercially available PSG system (either Series S or Series E Sleep System, Compumedics, Melbourne, Australia at baseline, and Series E Sleep System at follow-up). The following signals were recorded: electroencephalography (Cz, C4-A1, C3-A2, O2-A1, O1-A2), left and right electrooculogram, submental electromyogram (EMG), left and right anterior tibialis muscle EMG, and ECG. Oxygen saturation was measured using Masimo Radical®SET (Masimo Corp., CA, USA) at baseline and the Bitmos GmbH, (Dusseldorf, Germany) at follow-up, both of which use Masimo signal extraction technology for signal processing. All oximeters were set to a 2-s averaging time. Transcutaneous carbon dioxide was measured at baseline using a TCM4/40 (Radiometer, Copenhagen, Denmark) and at follow-up using a TINA TCM3 (Radiometer, Copenhagen, Denmark). Respiratory parameters recorded included thoracic and abdominal respiratory inductance plethysmography (Pro-Tech zRIP™ Effort Sensor, Pro-Tech Services Inc., Mukilteo, WA, USA), nasal pressure (Salter Style®, Salter Labs, Arvin, CA, USA) and oronasal airflow (Sandman®BreathSensor™, Child Airflow Thermistor, Tyco Healthcare, UK). The children went to bed once lead application was complete and were awakened at 6:00 am.

In addition to standard PSG, BP was measured continuously and non-invasively via finger arterial photoplethysmography using a Finometer™ (Finapres Medical Systems, Amsterdam, Netherlands). However, BP data are not reported here.

2.3. PSG data analysis and groups

All PSG studies were sleep-staged in 30-s epochs by experienced pediatric sleep technologists, with respiratory and sleep parameters being scored and calculated using the same protocol as previously published for the baseline study [34]. Studies were excluded if <4 h of sleep were recorded. Because the initial PSG studies were conducted before the implementation of the American Academy of Sleep Medicine new scoring rules [35], all PSG studies were sleep-staged using Rechtshaffen and Kales criteria [36] into wake after sleep onset, non rapid eye movement sleep stages 1, 2, 3 and 4 (NREM1-4), and rapid eye movement (REM) sleep. Sleep technologists were blinded to previous SDB severity. Respiratory events were scored if they were $\geqslant$2 respiratory cycles in duration, and the classification of apneas and hypopneas was based on the American Academy of Sleep Medicine criteria [37] with minor modifications in accordance to clinical practice at the time of the baseline study. Arousals were scored as either cortical arousals as defined by the American Sleep Disorders Association [38] or as sub cortical activations [37] Sub cortical activations were scored when $\geqslant$2 of the following were present: an increase in EMG, an increase in HR, or a body movement. Respiratory events were only analyzed during periods without body movement. An obstructive apnea was defined as a decrease in flow to <10% of baseline in flow signal in the presence of continued or increased respiratory effort. An obstructive hypopnea was scored when a clear reduction from baseline in flow signal occurred in the presence of respiratory effort (with paradox or phase shift) and was associated with snoring or noisy breathing at event termination in conjunction with an arousal, awakening, or $\geqslant$3% oxygen desaturation. An obstructive apnea index was calculated as the total number of obstructive apneas, hypopneas and mixed apneas per hour of total sleep time (TST), and 3% oxygen desaturation index (ODI) as the ODI $\geqslant$3% per hour of TST.

SDB severity was categorized using the obstructive apnea–hypopnea index (OAHI) into primary snoring (PS, OAHI $\leqslant$1 event/h), mild OSA (OAHI between >1 and $\leqslant$5 events/h), and moderate/severe OSA (MS, OAHI >5 events/h). At follow-up, children were grouped into unresolved if they had an OAHI $\geqslant$1 or snored on

Download English Version:

https://daneshyari.com/en/article/6061316

Download Persian Version:

https://daneshyari.com/article/6061316

Daneshyari.com