Autonomic Cardiovascular Tests in Children with Obstructive Sleep Apnea Syndrome

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Study Objectives: The aim of our study was to investigate cardiovascular autonomic activity during wakefulness, using cardiovascular tests, in a population of children with OSAS.

Design: Prospective study.

Setting: Sleep unit of an academic center.

Participants: We included 25 children (mean age 10.2 ± 2.3 years) undergoing a diagnostic assessment for OSAS, and 25 age-matched healthy control subjects. All subjects underwent an overnight polysomnography and autonomic cardiovascular tests using parts of the Ewing test battery, which is a physiological test used for the assessment of autonomic function (head-up tilt test, Valsalva maneuver, deep breathing test).

Measurements and Results: Eighteen of 25 children with OSAS (11 males, mean age 9.4 ± 1.7 years) concluded the study. OSAS patients had higher systolic blood pressure, diastolic blood pressure, baseline heart rate, the 30:15 index (which represents the RR interval at the 15th and 30th beats during the head up tilt test), and delta diastolic and systolic blood pressure during the head-up tilt test, while the heart rate variability during the deep breathing test was lower, compared with controls. A positive correlation between systolic and diastolic blood pressure and the apnea-hypopnea index (AHI), and negative between AHI and both the 30:15 index and Valsalva ratio, were found. Stepwise linear regression analysis detected a negative correlation between AHI and the 30:15 index and Valsalva ratio, a positive correlation between overnight mean oxygen saturation and delta heart rate, and between AHI and delta systolic blood pressure.

Conclusions: Our data point to an increase in basal sympathetic activity during wakefulness and to an impaired reaction to several physiological stimuli, which is dependent on the severity of OSAS.

Keywords: Autonomic cardiovascular system, obstructive sleep apnea syndrome, children

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CHRONIC CARDIOVASCULAR AUTONOMIC ABNORMALITIES DURING BOTH SLEEP AND WAKEFULNESS HAVE BEEN DEMONSTRATED IN ADULTS WITH obstructive sleep apnea syndrome (OSAS), caused by sympathovagal balance alterations.[1-3] Persistent muscle sympathetic nerve activation and high levels of circulating norepinephrine increase the sympathetic drive in adults with OSAS.[4,5] At the same time, heart rate variability (HRV) during sleep is markedly reduced, which suggests impaired parasympathetic control.[6] The pathogenesis of this persistent sympathetic activation has yet to be fully understood. Repetitive episodes of apnea cause hypoxia and sleep fragmentation, inducing tonic activation of chemoreflex activity, and an increase in sympathetic nerve activity, as, demonstrated by muscle sympathetic microneurography.[7-11] Studies in children with OSAS have reported increased blood pressure,[12] changes in cardiac structure and function,[13,14] increased fasting insulin and lipid levels,[15,16] and endothelial dysfunction, as signs of cardiovascular damage.[17,18] However, the few studies that have evaluated autonomic dysfunction reported an increase in diastolic blood pressure, both during wakefulness and sleep,[19,20] as well as an increase in sympathetic activity, demonstrated by peripheral arterial tonometry,[21,22] and catecholamine concentration measurements in plasma and urine.[23] Yet other studies have documented an increase in HRV,[24] and a drop in blood pressure during wakefulness, as assessed by the head-up tilt test, in children with OSAS.[25] The head-up tilt test is part of the Ewing battery, which is a physiological test for the assessment of autonomic function.[26] It studies cardiovascular autonomic reflexes by analyzing changes in heart rate and arterial pressure induced by a range of physiological stimuli.

The aim of our study was to investigate the activity of the autonomic nervous system during wakefulness using the cardiovascular tests in a cohort of children with OSAS, in order to detect not only sympathetic, but also parasympathetic activity.

SUBJECTS AND METHODS

We included in our study 25 consecutively enrolled children (mean age 10.2 ± 2.3 years, range 6-16.6 years) who were undergoing a diagnostic assessment for OSAS in our pediatric sleep centre (Rome, Italy) because of habitual snoring, apnea or restless sleep, as witnessed by their parents. The diagnosis of OSAS was confirmed by laboratory PSG yielding an obstructive apnea/hypopnea index > 1, according to the criteria of the American Academy of Sleep Medicine.[27] Primary snoring was diagnosed in children with habitual snoring and an apnea-hypopnea index < 1 and with snoring detected by microphone.

Twenty-five healthy children, with no reported sleep disturbance, were recruited from a community-based survey to act as controls (mean age 10.8 ± 2.9 years, range 6-12.5 years). All the children were enrolled between May 2007 and May 2008.

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Upon recruitment, all the participants were asked in detail about their personal and family history and underwent a general clinical examination. Body mass index (BMI) was calculated as weight/height^2 (kg/m^2); obese children with a BMI value ≥ 95th percentile were excluded.[28,29] Exclusion criteria included the presence of genetic disorders, cerebral palsy, neuromuscular diseases, cardiac disease, renal disease, and any systemic diseases. Patients using medications that could potentially affect autonomic nervous system function and blood pressure (including β-agonists) were excluded. All the participants' parents provided written informed consent to the study. The study procedures were approved by the hospital ethics committee.

Polysomnographic Analysis

All the patients underwent overnight standard polysomnography performed in our sleep laboratory using a digital multichannel system (Model Heritage Grass Instruments; Quincy, MA) in a sound-proof room. Each recording started at around 21:00 and stopped when the children woke up spontaneously in the morning. None of the subjects was sedated or suffered from sleep deprivation. All the recordings included: standard scalp electroencephalographic (EEG) leads (C4-A1/C3-A2, O2-A1/O1-A2 according to the 10-20 electrode placement system); bilateral electro-oculogram; chin electromyogram; 2-lead ECG; nasal and oral thermistor airflow; thoracic and abdominal respiratory effort using a piezo-respiration effort sensor (Protech, Mukilteo, WA USA); overnight oxygen saturation (SpO_2) and pulse wave (PW), using finger pulse oximetry (Nelcor ENPCOR 290, Pleasanton, CA, USA).

Sleep was subdivided into 30-sec epochs, and sleep stages were scored according to the standard criteria by Rechtschaffen and Kales.[30] Central, obstructive, and mixed apnea events were counted according to the criteria established by the American Academy of Sleep Medicine.[31] The apnea-hypopnea index (AHI) was defined as the average number of apneas and hypopneas per hour of total sleep time (TST = time from sleep onset to the end of the final sleep epoch minus time awake); oxygen desaturation index (ODI) was defined as the average number of oxygen desaturations per hour of TST.

Autonomic Cardiovascular Tests

After undergoing the polysomnographic recording in the morning (08:00), the patients underwent the autonomic cardiovascular tests according to the test battery described by Ewing, and to the standard procedure described in the consensus statement, i.e., the heart rate response to Valsalva maneuver, deep breathing and standing and the blood pressure response to standing or tilting[26,32]

ECG monitoring was performed using a digital multi-channel system (Model Heritage Grass Instruments; Quincy, MA). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured manually with a Riva Rocci sphygmomanometer,[38] according to the standard method. Room temperature was 20-23°C. The results of these tests in the patient group were compared with the normal reference values obtained from the control group.

We selected the children who were most collaborative to the tests (5 of 25 OSAS children were excluded because they did not collaborate). Before testing, we explained the test to the child and mimicked it; in the Valsalva maneuver and deep breathing test, the operator helped the child count the seconds.

Head-up tilt test

The patient started the tilt test after 10 min of supine rest. Before testing began, systolic and diastolic blood pressure (SBP and DBP, mm Hg) were measured twice, i.e., every 5 min. After standing, blood pressure was measured once/min during the first 10 min, and then once/3 min until the end of the test, in order to obtain the maximum number of measurements (manually). We analyzed blood pressure according the standard criteria for sex and age, following international guidelines.[33]

The electrocardiogram (ECG) was performed in the supine position for 10 min and the heart rate (HR, beats/minute, bpm) recorded. The patient was then rapidly placed upright by means of a motorized table with footboard support for about 20 min. The start of the maneuver was marked on the tracing. The cardiac response to the manoeuvre was expressed as the ratio between the longest R-R interval recorded during the first 30 beats after orthostatism and the shortest R-R interval during the first 15 beats (maximum/minimum 30:15 ratio). This response is mediated by the vagus nerve (parasympathetic activity); rapid increase in heart rate occurs at about the 15th beat after standing, and relative bradycardia occurs at about the 30th beat.[34]

Arterial pressure measured with the subject lying down was measured again upright, maintaining the arm cuff at the level of the heart. Standing upright induces a physiological fall in blood pressure, which activates peripheral vasoconstriction mediated by sympathetic activity.[34]

The main parameters of the head-up tilt test considered were:

1. systolic and diastolic blood pressure changes during the first 3 min of the test (ΔSBP and ΔDBP, mm Hg). ΔSBP and ΔDBP are the differences between the highest and the lowest blood pressure values during the 3 min in the orthostatic position;
2. heart rate change during the first 30 sec in the orthostatic position (ΔHR, bpm). Highest and lowest HR changes during the first 30 sec in the orthostatic position;
3. the 30:15 index, which represents the RR interval at about the15th and 30th beats after tilt.

Valsalva maneuver

The Valsalva maneuver reflects cardiac parasympathetic activity.[34] The patient performed the Valsalva maneuver in the sitting position. The child inhaled and exhaled forcibly into a mouthpiece connected to a modified sphygmomanometer, until the pressure value reached one-third of the subject's baseline arterial pressure (values around 30 mm Hg). The subject maintained that pressure for 15 s under continuous ECG monitoring. Blood pressure was measured once during testing and once after the mouthpiece had been removed. The Valsalva ratio[35] was expressed as the ratio between the longest R-R interval after the maneuver (rebound bradycardia) and the shortest R-R interval during respiratory effort (expression of tachycardia during the maneuver), determined from the ECG trace.

For this test, we slightly modified the Ewing battery, as we performed the Valsalva maneuver once, as opposed to 3 times, in order to increase the compliance of the child (the test thus

becomes more comfortable and quicker), according to the O'Brien battery.[36]

The parameters considered were:

1. the difference between the highest heart rate during the respiratory effort and the baseline heart rate (ΔHR max-min, bpm)
2. the Valsalva ratio, which represents the longest RR interval after manoeuvres and the shortest RR interval during manoeuvres.

Deep breathing test

The deep breathing test reflects parasympathetic activity.[34] The patient sits quietly and breathes deeply, taking 6 breaths per minute (5 sec in and 5 sec out) for one minute. Deep breathing at a rate of 6 breaths per minute is considered the most convenient and reproducible technique, as explained by Ewing et al.,[37] and is also used to reduce interferences between breathing and heart rate regulation.[38] An electrocardiogram is performed throughout the deep breathing and the onset of each inspiration and expiration is indicated. The RR interval during each cycle is expressed as the mean difference between the maximum and minimum heart rates for the 6 cycles measured (beats per minute). Heart rate variation is also measured as the ratio between the expiration and inspiration heart rates (called E:I ratio).

When the data analysis was performed, 2 main parameters were considered:

1. the E:I ratio, which represents the longest RR interval (expressed in msec) during the expiratory phase and the shortest RR interval (expressed in msec) during the inspiratory phase
2. the highest and lowest heart rate changes (ΔHR insp-exp, bpm), which represent the mean difference between the highest and lowest heart rate in each respiratory cycle (normal value > 15 beats/min).

Statistical Analysis

Data are expressed as the mean ± SD. Unpaired *t*-test or ANOVA with post hoc Scheffe test were used for comparisons between ≥ 2 subgroups when appropriate. Pearson correlation coefficients between variables were calculated. A multiple linear regression analysis (stepwise method) was performed to explore any correlations between age, sex, BMI percentile, SBP percentile, DBP percentile, HR, AHI, and SpO_2%, ODI and mean oxygen desaturation, and the autonomic test results. Data were considered to be statistically significant at P values of less than 0.05. Statistical analysis was performed using a commercial software package (SPSS, version 11; SPSS; Chicago, IL).

RESULTS

Two OSAS children were excluded after they received β-agonists drugs for an asthma exacerbation, and a further 5 were lost as a result of poor compliance with the autonomic test procedure (they refused to undergo Valsalva maneuver and deep breathing test). The final sample was thus composed of 18 children with OSAS (11 males, mean age 9.4 ± 1.7 y) and 25 healthy subjects (14 males, mean age 10.8 ± 2.9 y). There were no significant differences in the anthropometric parameters between the 2 groups (Table 1). The polysomnographic parameters revealed a significantly higher AHI, ODI, and a significantly lower minimal oxygen desaturation in children with OSAS than in controls (Table 1).

Table 1—Anthropometric characteristics and polysomnographic parameters in children with obstructive sleep apnea syndrome (OSAS group) and in healthy children (control group)

	OSAS group (n = 18)	Control group (n = 25)	P
Anthropometric characteristics			
Age (y)	9.4 ± 1.7	10.8 ± 2.9	NS
Males (n)	11	14	NS
BMI (kg/m^2)	18.3 ± 1.9	18.6 ± 3.2	NS
BMI centile	70.3 ± 19.2	61.6 ± 29.2	NS
Polysomnographic parameters			
TST (min)	430.95 ± 37.9	466.00 ± 31.5	NS
S1 %	4.1 ± 3.1	4.0 ± 3.9	NS
S2 %	49.8 ± 9.9	43.9 ± 4.1	NS
SWS %	32.9 ± 6.4	29.9 ± 5.3	NS
REM %	20.6 ± 8.5	19.1 ± 5.4	NS
SE %	83.6 ± 7.6	86.6 ± 5.8	NS
AHI (/h)	5.3 ± 4.8	0.3 ± 0.2	0.004
SpO_2 (%)	97.2 ± 1.7	97.7 ± 1.5	NS
ODI (/h)	1.6 ± 2.0	0.1 ± 0.2	0.03
Minimal SpO_2 (%)	93.3 ± 2.4	96.2 ± 2.6	0.01
Mean oxygen desaturation (%)	95.5 ± 2.3	96.9 ± 1.7	NS

TST refers to total sleep time; SE, sleep efficiency; S1, sleep stage 1; S2, sleep stage 2; SWS, slow-wave sleep; AHI, apnea-hypopnea index (events/h); SpO_2%, average overnight arterial oxygen saturation; ODI, oxygen desaturation index (events/hour).

Autonomic Cardiovascular Tests

Children with OSAS had significantly higher values than controls in the baseline SBP, DBP, HR, as well as in ΔSBP and ΔDBP, during the head-up tilt test, and significantly lower values in the 30:15 index (Table 2). AHI correlated positively with SBP (r = 0.451, P = 0.01; Figure 1) and DBP (r = 0.324, P = 0.04; Figure 2), while DBP correlated negatively with mean overnight oxygen saturation (SpO_2%) (r = −0.430, P = 0.02; Figure 3). We also found a significant positive correlation between ΔHR and SpO_2% (r = 0.387, P = 0.04; Figure 4), as well as between ΔSBP and AHI (r = 0.485, P = 0.009; Figure 5), and a significant negative correlation between the 30:15 index and AHI (r = −0.454, P = 0.01; Figure 6).

No significant differences were found between the groups in the Valsalva parameters (Table 2). A significant negative correlation was found between the Valsalva ratio and AHI (r = −0.490, P = 0.008; Figure 7); ΔHR during the deep breathing test was significantly lower in OSAS children than in controls (Table 2).

Stepwise linear multiple-regression analysis identified AHI as the only variable that was significantly correlated with the 30:15 index and Valsalva ratio; SpO_2% was the only variable

Table 2—Autonomic cardiovascular tests parameters in children with obstructive sleep apnea syndrome (OSAS group) and in healthy children (control group)

	OSAS group (n = 18, 41.8%)	Control group (n = 25, 58.2%)	P
Cardiorespiratory parameters			
SBP (mm Hg)	111.66 ± 7.9	100.5 ± 8.5	0.001
SBP centile	81.61 ± 17.7	52.8 ± 11.2	0.001
DBP (mm Hg)	64.7 ± 9.1	57.7 ± 11.1	0.03
DBP centile	66.3 ± 20.3	54.9 ± 13.8	0.05
HR (bpm)	77.0 ± 9.0	67.5 ± 7.3	0.001
Autonomic cardiovascular tests			
Head-up tilt test			
ΔHR (bpm)	21.1 ± 12.2	20.6 ± 14.3	NS
30:15 index	1.16 ± 0.13	1.25 ± 0.13	0.03
ΔSBP (mm Hg)	12.8 ± 9.2	7.8 ± 7.7	0.05
ΔDBP (mm Hg)	17.1 ± 10.3	9.0 ± 5.9	0.002
Valsalva maneuver			
Valsalva ratio	1.5 ± 0.3	1.6 ± 0.3	NS
ΔHR (max-min, bpm)	17.6 ± 9.6	13.7 ± 9.3	NS
Deep breathing test			
E:I ratio	1.38 ± 0.2	1.5 ± 0.2	NS
ΔHR (insp-exp, bpm)	9.0 ± 3.4	13.4 ± 5.9	0.006

SBP, systolic blood pressure (mm Hg); DBP, diastolic blood pressure (mm Hg); HR, heart rate (bpm); ΔHR, highest and lowest heart rate changes during first 30 sec in orthostatic position (bpm); 30:15 index, RR interval at 15th and at 30th beats after tilt (normal value > 1.04); ΔSBP and ΔDBP (mm Hg), diastolic and systolic blood pressure changes during first 3 min of test; Valsalva ratio, the longest RR interval after manoeuvres and the shortest RR interval during manoeuvres (normal value more than 1.21); ΔHR (max-min, bpm), the greatest heart rate difference from baseline during Valsalva maneuver; E:I ratio, the longer RR interval during expiratory phase and the shorter RR interval during inspiratory phase (normal value more than 1.10); ΔHR (insp-exp, bpm), the mean difference between highest and lowest heart rate in each respiratory cycle (normal value > 15 bpm).

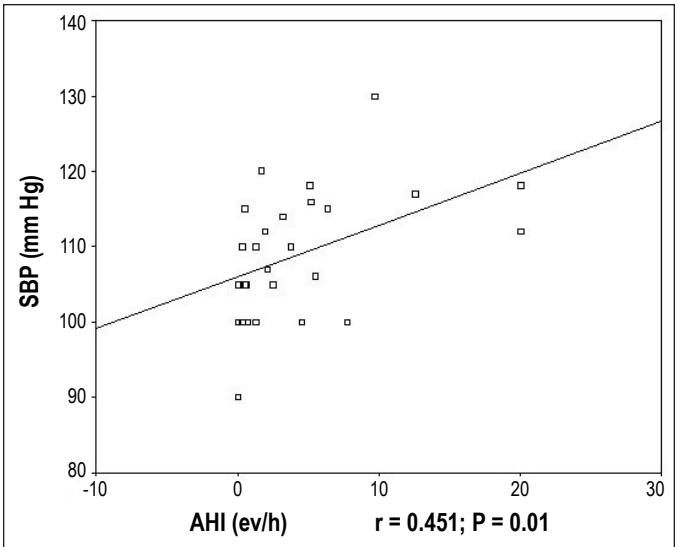

Figure 1—Correlation between basal blood pressure values SBP (mm Hg) and apnea-hypopnea index (AHI, events/hour)

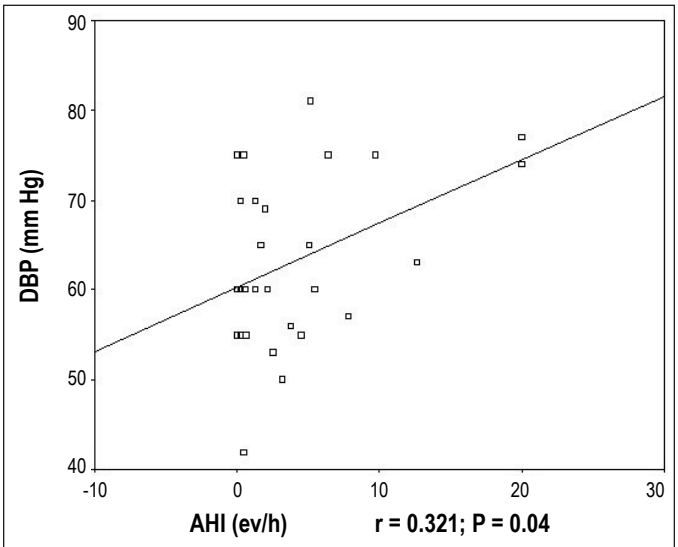

Figure 2—Correlation between basal blood pressure values DBP (mm Hg) and apnea-hypopnea index (AHI, events/hour)

significantly correlated with ΔHR; AHI, SpO_2%, and SBP were significantly correlated with ΔSBP (Table 3).

DISCUSSION

This study provides data about autonomic cardiovascular activity during wakefulness, by means of a functional test, in a population of children with OSAS. We observed an increase in the baseline SBP, DBP, and HR of children with OSAS, whereas the autonomic cardiovascular tests revealed a lower 30:15 index, greater SBP and DBP variability during the supine-to-orthostatic posture change, as well as less HRV during deep breathing in OSAS patients than in controls. Indeed, we observed reduced HRV after 30 sec of deep breathing in OSAS patients, whereas control subjects displayed increased HRV. Our results point to a diurnal sympathetic imbalance. We believe that this imbalance may reflect an increase in sympathetic activity or a decrease in parasympathetic activity. The excessive drop in orthostatic blood pressure observed in this study might be considered another sign of increased sympathetic tone, while parasympathetic nervous system hypoactivity was found during the deep breathing test. However, the fact that we did not find any difference between OSAS children and controls in the Valsalva maneuvers suggests that the extent of the damage in the former is mild.

A mild increase in blood pressure, that does not however reach the pathological threshold, has previously been reported in pediatric OSAS patients during both sleep and wakefulness.[16,17,24] A chronic increase in sympathetic tone or impaired parasympathetic control, has previously been postulated in adults.[6,33,34] Indeed, it has been hypothesized that cardiovascular morbidity in OSAS adult patients might be caused by increased sympathetic activity,[37,38] in response to intermittent hypoxia and arousals during sleep.[39,40] The enhanced sympathetic drive observed in OSAS adults may even be related to reduced daytime baroreflex sensitivity.[41-43] However, the long term significance of these changes in children need to be fur-

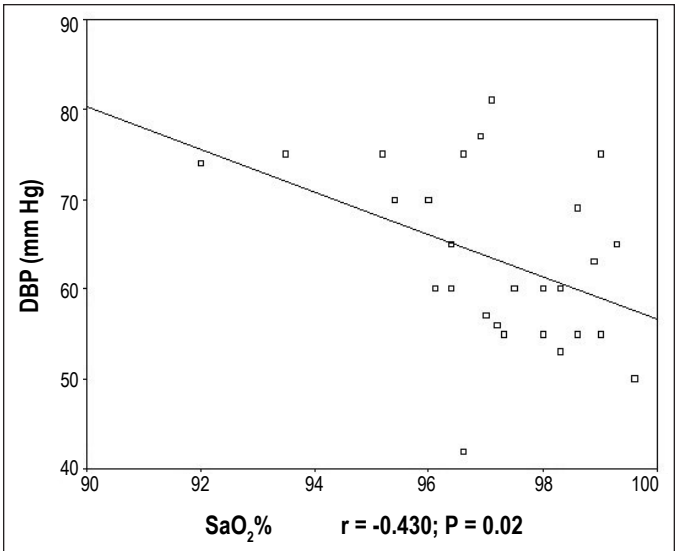

Figure 3—Correlation between DBP (mm Hg) and overnight arterial oxygen saturation (SpO_2%)

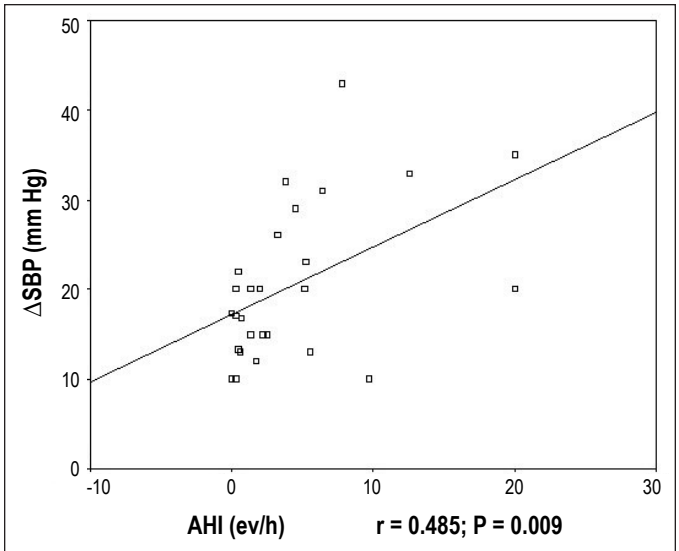

Figure 5—Correlation between systolic blood pressure changes (ΔSBP, mm Hg) during head-up tilt test and apnea-hypopnea index (AHI events/h)

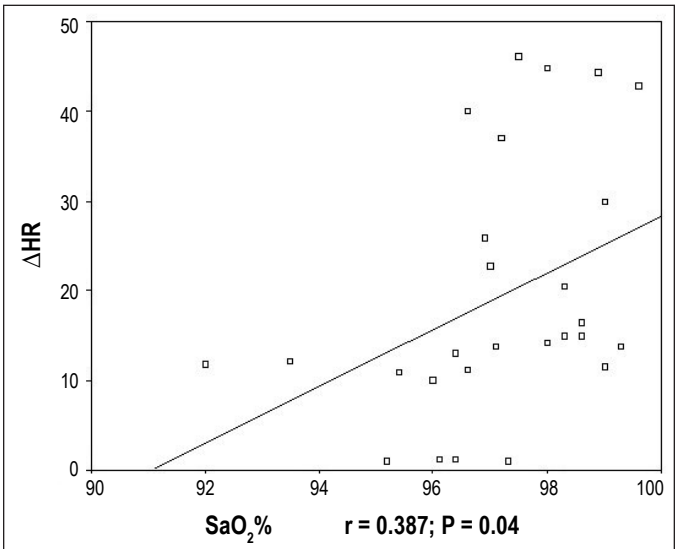

Figure 4—Correlation between heart rate change (ΔHR, bpm) during the head-up test and the overnight arterial oxygen saturation (SpO_2%)

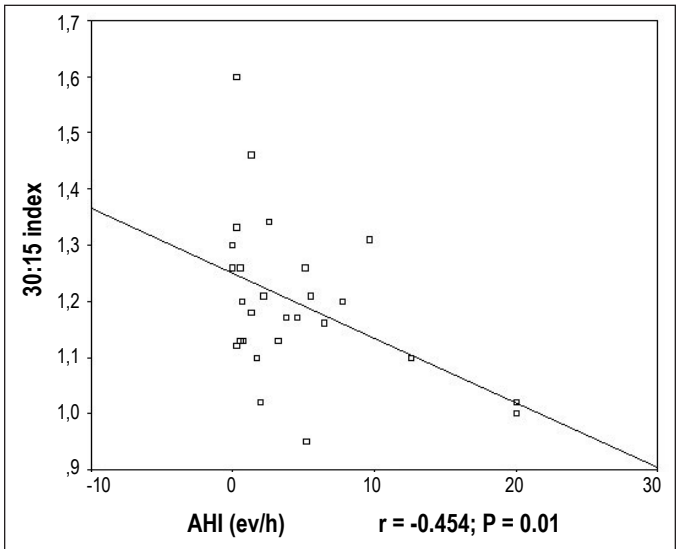

Figure 6—Correlation between 30:15 index and apnea-hypopnea index (AHI events/hour)

ther investigated, in order to find out a continuum with the autonomic changes found in adults.

In addition, our results confirm previous data reported in studies on children. One study examined HRV in a small group of children with SDB. Analysis of moment-to-moment changes in the RR intervals revealed increases in sympathetic activity, as well as vagal discharge during respiratory events.[32] Some studies based on urine catecholamines or peripheral arterial tonometry have revealed an increase in sympathetic tone in children with OSAS,[22,23] while other studies have reported sympathovagal balance alterations during sleep, as demonstrated by reduced HRV.[35,36] Unfortunately, we did not calculate HRV during sleep in our subjects. We have previously assessed cardiovascular autonomic function by means of Ewing battery and we also calculated HRV during sleep, in a group of children affected by diabetes mellitus.[44] In this study, patients did not show differences in cardiovascular activity compared with controls, during either wake or sleep. We suggest that these two measures of cardiovascular activity (Ewing battery and HRV) may be able to detect similar autonomic cardiovascular changes.[44]

We also found a positive correlation between blood pressure and sleep respiratory indexes (systolic pressure correlated with AHI, while diastolic pressure correlated with SpO_2). Linear regression analysis indicated AHI and SpO_2% as possible causative factors in the cardiovascular alterations. These results support the hypothesis that recurrent hypoxia during sleep and sleep respiratory events may acutely stimulate chemoreceptors. It has been demonstrated that in adults sleep respiratory events may acutely stimulate chemoreceptors thereby inducing cyclic sympathetic overactivity,[45] and raising the 'set point' of baroreflex control.[46] However, these results can not be confirmed in children because of the paucity of studies evaluating long-term changes in autonomic activity in pediatric OSAS.

One study investigated the head-up tilt test performed in children with OSAS.[25] The authors of that study, who found increased SBP and DBP variability, suggested that arterial BP is predominantly modulated by parasympathetic tone during the daytime, hypothesizing that resistive breathing reduces BP, increases respiratory drive during inspiration and reduces muscle sympathetic nerve activity. In our study, we performed autonomic cardiovascular tests adding Valsalva maneuver and the deep breathing test to the head-up tilt test. Although our results confirm those reported in that previous study,[25] we suggest that these findings might reflect an increase in baseline sympathetic activity, associated with an imbalance of parasympathetic response to acute stimuli.

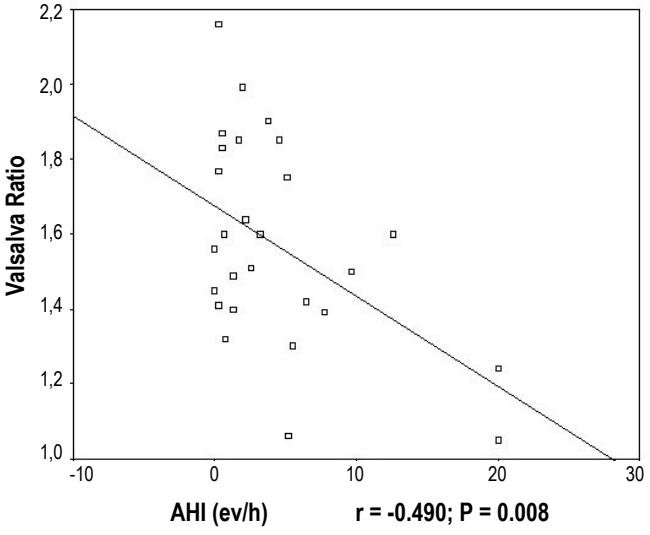

Figure 7—Correlation between Valsalva ratio and apnea-hypopnea index (AHI events/hour)

Table 3—Stepwise multiple linear regression analysis

Regression analysis results				
Dependent variable	**Significant variable**	**r^2**	**Standardized β coefficients**	**P**
ΔHR (bpm)	SpO_2%	0.15	0.387	0.042
30:15 index	AHI (/h)	0.21	−0.454	0.015
	AHI (/h)	0.23	0.804	< 0.001
ΔSBP (mm Hg)	SBP centile	0.39	0.408	0.020
	SpO_2%	0.55	−0.361	0.044
Valsalva ratio	AHI (/h)	0.24	−0.490	0.008

ΔHR (bpm), Highest and lowest heart rate changes during first 30 sec of tilt test; 30:15 index, RR interval at 15th and at 30th beats after tilt (normal value > 1.04); ΔSBP (mm Hg), systolic blood pressure changes during first 3 min of tilt test; Valsalva Ratio, the longest RR interval after maneuver and the shortest RR interval during manoeuvres (normal value more than 1.21); AHI, apnea-hypopnea index (events/hour); SpO_2%, overnight arterial oxygen saturation.
Model 1 (ΔHR) excluded variables: age (years), sex, BMI centile, AHI (/h), basal HR (bpm), SBP centile and DBP centile, ODI oxygen desaturation index (events/h), mean oxygen desaturation (%)
Model 2 (30:15 index) excluded variables: age (y), sex, BMI centile, SpO_2 (%), basal HR (bpm), SBP centile and DBP centile, ODI oxygen desaturation index (events/h), mean oxygen desaturation (%)
Model 3 (ΔSBP) excluded variables: age (y), sex, BMI centile, basal HR (bpm) and DBP centile, ODI oxygen desaturation index (/h), mean oxygen desaturation (%)
Model 4 (Valsalva ratio) excluded variables: age (y), sex, BMI centile, SpO_2 (%), basal HR (bpm), SBP centile and DBP centile, ODI oxygen desaturation index (/h), mean oxygen desaturation (%).

Lastly, we should bear in mind that the autonomic cardiovascular tests requires the subject's cooperation and that inter- and intra-subject variability may consequently affect the results.[44,47]

Furthermore, computer-assisted systems for the collection and analysis of heart rate and blood pressure values are preferable to the manual collection of data. We collected the heart rate data using a computerized system, though we did not measure beat-to-beat heart rate or blood pressure variability, thus missing the opportunity to obtain considerably more information. However, despite the limited number of subjects investigated and the limitations of our methods, the results of our study appear to confirm the presence of autonomic cardiovascular anomalies in pediatric OSAS patients. Indeed, the fact that the children we enrolled had relatively mild forms of OSAS suggests that cardiovascular system alterations affect all OSAS patients, regardless of the severity of disease. Further follow-up studies on larger samples of children with OSAS are warranted to confirm these findings.

DISCLOSURE STATEMENT

This was not an industry supported study. The authors have indicated no financial conflicts of interest.

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