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SLEEP BREATHING PHYSIOLOGY AND DISORDERS • ORIGINAL ARTICLE

Aortic root size is associated with nocturnal blood pressure in a population of hypertensive patients under treatment for obstructive sleep apnea

Pascal Delsart[1,2] · Jerome Soquet[1,3] · Elodie Drumez[4] · Francis Juthier[1,3] · Seenam Kutoati[1] · Thomas Nganou Koutouzi[1] · Stephanie Fry[1] · Anne Mallart[1] · David Montaigne[1,3] · Claire Mounier-Vehier[1,3]

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Abstract

Background Obstructive sleep apnea (OSA) is associated with an increased aortic root size. This association has never been studied in patients with hypertension undergoing continuous positive airway pressure (CPAP) treatment for OSA.

Methods The 24-h blood pressure (BP) monitoring of 142 hypertensive patients undergoing CPAP treatment for OSA was prospectively documented. Aortic root diameter was assessed by echocardiography.

Results The population included 33.8% women, with an overall mean age of 60.7 ± 10.5 years. The median body mass index was 32.7 [29. 5–36.3] kg/m^2. The median treatment score was 3 [2–4] anti-hypertensive drugs per day. The median 24-h systolic and diastolic BP were 130 [120–144] and 74.5 [69–82] mmHg, respectively. The night-time systolic and diastolic BP were 119.5 [108–136] and 67 [61–74] mmHg, respectively. The mean diameter of the aorta at the level of the Valsalva sinuses was 34.9 ± 4.4 mm and 20.4 ± 2.3 mm/m when adjusted for height. Patients underwent ventilation for a median duration of 3.8 [1. 7–7.5] years, with a median night-time duration of 6.6 [5. 5–7.5] h per night. The median residual apnea-hypopnea index under ventilation was 2 [1–4] events per hour. A multivariate analysis showed that aortic root size was associated with male gender ($p < 0.01$) and nocturnal diastolic BP ($p < 0.01$). When normalized for height, aortic root diameter was positively associated with age ($p < 0.01$) and nocturnal diastolic BP ($p < 0.01$).

Conclusion In OSA patients, the relationship between aortic root diameter and nocturnal BP persists on CPAP therapy.

Further studies that evaluate the potential protective effect of OSA treatment on aortic root dilatation should monitor nocturnal diastolic BP.

Keywords Aortic root · Nocturnal blood pressure · Obstructive sleep apnea · Ambulatory blood pressure monitoring · Continuous positive airway pressure

Introduction

Obstructive sleep apnea (OSA) is one of the main causes of poor blood pressure (BP) control. The association of higher BP level and OSA is independent of confounders including age and obesity [1, 2]. In cases of resistant hypertension, severe OSA is found in about 60% of patients [3].

The link between OSA and aortic diameter has been described at different levels of the aorta (ascending aorta and abdominal aorta) [4, 5]. A high prevalence of OSA in different aortic diseases has also been described and it can affect the prognosis during follow-up [6–8]. Different mechanisms are involved in the pathogenesis. Thus, an association between the aortic root size and a lower mean nocturnal oxygen saturation has been described [4]. A study by Baguet et al. showed that a higher diastolic BP was independently associated with an increased aortic root size [4]. In a longitudinal study, the aortic root diameter was positively correlated with BP level [9].

An increased BP is linked with intermittent hypoxemia and other mechanisms [10]. Continuous positive airway pressure

✉ Pascal Delsart
delsartpascal@yahoo.fr; Pascal.DELSART@chru-Lille.fr

1 CHU Lille, Institut Cœur Poumon, Bd Pr Leclercq, F-59000 Lille, France

2 Vascular Medicine and Hypertension Department, Institut-Coeur-Poumon, Boulevard Pr Leclercq, 59037 Lille Cedex, France

3 University of Lille, CHU Lille, F-59000 Lille, France

4 Department of Biostatistics, Univ. Lille, CHU Lille, EA 2694 - Santé publique : épidémiologie et qualité des soins, F-59000 Lille, France

(CPAP) improves nocturnal oxygen saturation and reduces BP even in cases of well-treated hypertension [11, 12]. CPAP can also correct different physiopathologic mechanisms caused by OSA as it reduces aortic stiffness and sympathetic activity [13]. It has been hypothesized that these parameters could be factors influencing cardiovascular prognosis.

The effect that treating OSA has on the prevention of aortic root enlargement has never been studied. In the present work, our aim was to analyze whether the association between the aortic root size on one hand and respiratory and BP parameters on the other hand, was still present in a population of hypertensive patients undergoing CPAP for OSA.

Material and methods

From December 2014 to February 2017, we prospectively screened consecutive hypertensive patients who were referred to our Vascular Medicine and Hypertension Department to perform 24-h BP monitoring. Our center mostly receives patients at high cardiovascular risk or for secondary prevention, including patients with diabetes mellitus and peripheral artery disease. We only enrolled subjects undergoing CPAP treatment. Exclusion criteria were respiratory disease requiring oxygen therapy, central sleep apnea, poor adherence to CPAP treatment, and defined by a duration of use less than 4 h per night.

BP evaluation

All patients had an oscillometric clinical BP measurement with an Accutor plus MINDRAY* device after 5 min of rest, according to European guidelines [14]. Twenty-four-hour BP monitoring was performed with a Spacelab 90207 device, with a BP measurement programmed every 15 min. The awake period was defined as 7 am to 10 pm, and the sleeping period as 10 pm to 7 am. The day-time BP used in the analyses was the mean of the BP measurements performed during the awake period, and the night-time BP was the mean of the BP measurements performed during the sleeping period. A nocturnal hypertension was defined as a systolic BP ≥ 120 mmHg and/or a diastolic BP ≥ 70 mmHg. The dipping status was determined as a fall of at least 10% of the mean BP values during night-time compared to the day-time values. The different classes of anti-hypertensive medications (beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin 2-receptor antagonists, diuretics including thiazides and spironolactone, calcium-channel blockers, alpha-blockers, central acting agents) were recorded to create a treatment score, defined as the sum of the number of the different anti-hypertensive agents taken by a patient each day. Resistant hypertension was defined by the intake of at least three anti-hypertensive drugs per day including a full dose of thiazide diuretic. Patients included in the study were referred to our unit for a 24-h BP recording after an appointment with a cardiologist of our institution. At the time of enrollment (time of the 24-h BP recording), demographic and anthropometric data, conventional risk factors, and previous cardiovascular diseases were collected from patients' medical record. A conventional resting electrocardiogram was performed in all patients. Electric left ventricular hypertrophy was defined by the Sokolow-Lyon formula (SV1 + RV5 or RV6 > 35 mm). All subjects had a plasma measurement of creatinine, fasting glucose, cholesterol, triglycerides, high-density cholesterol, and low-density cholesterol.

Imaging

All patients had a transthoracic echocardiography performed by experienced physicians blinded to the respiratory data. Aortic root diameter was measured in a parasternal long-axis view in end diastole and at the largest cut of the sinuses of Valsalva as described by Roman et al. [15]. The aortic root was normalized for height by dividing the diameter of the aortic root by the height of the patient.

Respiratory parameters

We collected the data of each patient's ventilation device to determine the actual time spent under ventilation per night and the residual apnea-hypopnea index (AHI) under treatment. As the patients were recruited at the time of the BP monitoring, we retrospectively collected the data of OSA at the time of diagnosis before the initiation of CPAP treatment. Day-time sleepiness was evaluated by the Epworth Sleepiness Scale.

The present study was approved by the French National Data Protection Commission (*Commission Nationale de l'Informatique et des Libertés; Référence DEC201* 5–*19*). Each patient received a written information about the right to withdraw permission for the subsequent use of his or her personal data.

Statistical analyses

Data are presented as count (percentage) for qualitative variables and mean ± standard deviation (SD) or median [interquartile range] for quantitative variables. Normality of distribution was verified graphically and by using the Shapiro-Wilk test. Associations between the size of aorta and the size of aorta normalized for height and clinical data, OSA, and BP parameters were analyzed using the Student's *t* test for qualitative variables and the Pearson or Spearman correlation

coefficient test for quantitative variables. Factors associated in bivariate analyses with a P value < 0.10 were considered as candidates for multivariable analysis of covariance. Since multiple factors were highly correlated, factors were considered as candidates based on clinical relevance. The full models were simplified with a backward selection procedure with a value of $P < 0.05$ used as the cutoff for retention in the model. Comparisons of clinical data, OSA, and BP parameters according to nocturnal BP control were performed using the chi-squared or the Fisher's exact test for qualitative variables and the Student's t test or the Mann-Whitney U test for quantitative variables. All statistical tests were performed at the two-tailed α level of 0.05. Data were analyzed with SAS software version 9.4 (SAS Institute Inc., Cary, NC).

Results

Global characteristics

From the 303 patients screened at the time of the BP monitoring, 142 met the full inclusion criteria. The flow chart of the study population is presented in Fig. 1. The mean age of the population was 60.7 ± 10.5 years, with a proportion of women of 33.8%. The median body mass index (BMI) was 32.7 [29.5–36.3] kg/m^2, and the mean abdominal circumference was 117.3 ± 16.1 cm. Patients with a BMI ≥ 30.0 kg/m^2 accounted for 70.4% of the cohort. The prevalence of diabetes mellitus was 40.8%. A prior history of atrial fibrillation was noted in 9.9% of the population, a history of stroke in 12.7%, a

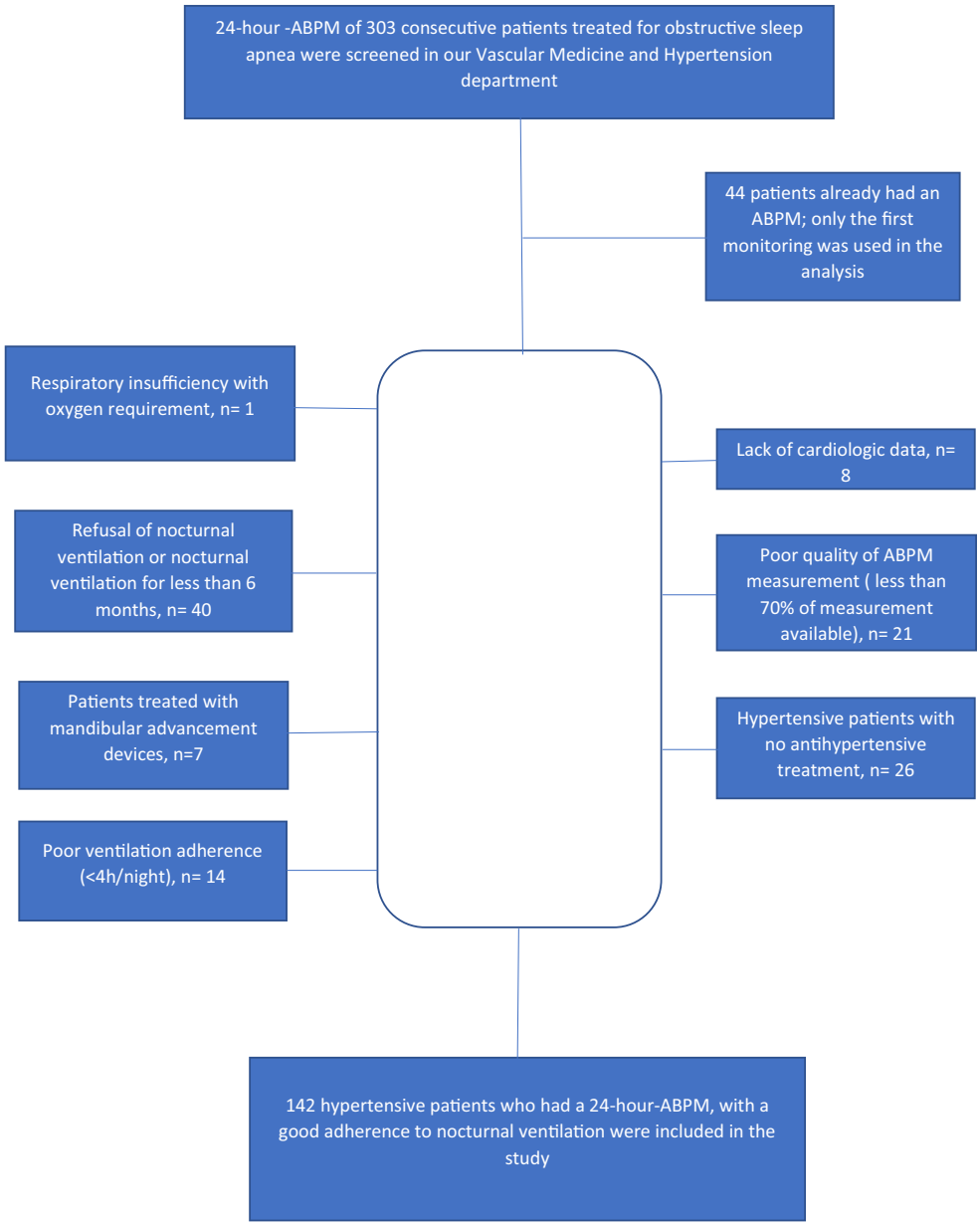

Fig. 1 Flow chart of the study population. ABPM ambulatory blood pressure monitoring

myocardial revascularization in 13.4, and 14.8% had a peripheral vascular disease. Patients' characteristics are presented in Table 1. None of the patients had a history of connective tissue disease, a bicuspid aortic valve, nor a mild or severe aortic insufficiency. The median duration of CPAP treatment was 3.8 years [1. 7–7.5]. The median duration of ventilation was 6.6 h/night [5.5–7.5]. The median anti-hypertensive treatment score was 3 [2–4]. The rates of use of different anti-hypertensive drugs were 44.4% for angiotensin-converting enzyme inhibitors, 30.3% for angiotensin 2 receptor antagonists, 54.9% for beta-blockers, 74.6% for calcium channel blockers, and 64.1% for diuretics. The median 24-h systolic and diastolic BP were 130 mmHg [120–144] and 74.5 mmHg [69–82], respectively. The median nocturnal systolic and diastolic BP were 119.5 mmHg [108–136] and 67 mmHg [61–74], respectively.

Table 1 Global characteristics of the study population ($n = 142$)

	Values
Patient's characteristics	
Age (years)	60.7 ± 10.5
Women	48 (33.8)
Weight (kg)	97 [86 to 110]
Height (cm)	171.9 ± 9.5
Body mass index (kg/m^2)	32.7 [29.5 to 36.3]
Abdominal circumference (cm)	117.3 ± 16.1
Cardiovascular risk factors	
Diabetes mellitus	58 (40.8)
Obesity	100 (70.4)
Current smoker	29 (20.4)
Dyslipidemia	90 (63.6)
Familial history of cardiovascular disease	25 (17.6)
Clinical blood pressure	
Systolic blood pressure (mmHg)	142.5 [133.5 to 153.5]
Diastolic blood pressure (mmHg)	81.5 [74.5 to 89.0]
Heart rate (beat per minute)	72 [62 to 85]
Echocardiographic data	
Aortic root diameter (mm)	34.9 ± 4.4
Aortic root diameter corrected by height (mm/m)	20.4 ± 2.3
Aortic root diameter > 40 mm	19 (13.4)
Biological data	
Plasma creatinine (mg/L)	10 [8 to 11]
LDL-cholesterol (mmolL)	2.8 ± 1.0
OSA parameters at the time of diagnosis	
Epworth Scale Score	8 [6 to 13]
AHI (event/h)	40.2 [31.0 to 56.0]
Whole-night nocturnal oxygen saturation	93.0 [92.0 to 94.3]
OSA parameters under CPAP	
Duration of CPAP (years)	3.8 [1.7 to 7.5]
CPAP nightly adherence (h/night)	6.6 [5.5 to 7.5]
Residual AHI (event/h)	2 [1 to 4]

Values are count (percentage), mean ± SD or median [interquartile range]

AHI apnea-hypopnea index, CPAP continuous positive airway pressure, LDL low-density lipoprotein, OSA obstructive sleep apnea, SD standard deviation

Correlation with the aortic root size

There was no correlation between aortic diameter and respiratory parameters at the time of OSA diagnosis (AHI, nocturnal oxygen saturation). There was a nonsignificant association between aortic root size normalized for height and OSA parameters on CPAP treatment ($p = 0.07$ for the duration of ventilation and $p = 0.60$ for nocturnal adherence). An increased aortic root size was significantly associated with male gender (36.2 mm ± 4.3 for men vs 32.6 mm ± 3.7 for women, $p < 0.01$), 24-h diastolic BP ($r = 0.21$, $p = 0.01$), nocturnal systolic BP ($r = 0.22$, $p = 0.01$), and nocturnal diastolic BP ($r = 0.33$, $p < 0.01$) (Table 2). Multivariate analysis showed that gender (β ± SEM for men using women as reference, 2.67 ± 0.85; $p < 0.01$) and diastolic nocturnal BP (β ± SEM, 0.14 ± 0.04; $p < 0.01$) were independently and positively associated with an increased aortic root diameter. Systolic and diastolic nocturnal BP were associated with aortic diameter normalized for height ($r = 0.021$, $p = 0.02$ for the systolic BP and $r = 0.25$, $p < 0.01$ for the diastolic BP) (Table 3, available in the online supplement). In multivariate analysis, two parameters were independently associated with the aortic root size normalized for height: age (β ± SEM, 0.06 ± 0.02; $p < 0.01$) and nocturnal diastolic BP (β ± SEM, 0.07 ± 0.02; $p < 0.01$).

Association with a higher nocturnal BP

Eighty-two subjects (58.8%) had a nocturnal systolic BP ≥ 120 mmHg and/or a nocturnal diastolic BP ≥ 70 mmHg. We noted a higher proportion of women in the controlled nocturnal BP group vs uncontrolled nocturnal BP group ($p < 0.01$). The size of the aorta was higher when BP was uncontrolled during the night ($p < 0.01$). The number of patients with an aorta ≥ 40 mm was also higher in this group ($p = 0.01$). The differences between both groups (controlled vs uncontrolled nocturnal BP) are presented in Table 4.

Discussion

This work is, to our knowledge, the first one to show that nocturnal BP is a significant modulator for aortic root size in a population of patients treated by CPAP for OSA. The main strength of the study is the good adherence of our population to nocturnal ventilation.

The pathophysiologic role of BP on the aortic enlargement is still unclear. Previous studies as the one by Meuleman et al.

Table 2 Variables associated with aortic root size, influence of nocturnal blood pressure

Variables	Values	*p*
Clinical characteristics		
Age (years)	0.05	0.53
BMI (kg/m^2)		0.62
OSA parameters at diagnosis		
Epworth Scale Score	0.23	0.049[a]
AHI (event/h)	0.05	0.60
Whole-night nocturnal oxygen saturation (%)	0.02	0.85
OSA parameters under CPAP		
Duration of ventilation (years)	0.09	0.35
CPAP nightly adherence (h/night)	− 0.02	0.81
Residual AHI (event/h)	− 0.01	0.90
Clinical blood pressure		
Systolic blood pressure (mmHg)	0.14	0.11
Diastolic blood pressure (mmHg)	0.15	0.08
24-h blood pressure monitoring		
24-h systolic blood pressure (mmHg)	0.15	0.092
24-h diastolic blood pressure (mmHg)	0.21	0.01
Day-time systolic blood pressure (mmHg)	0.08	0.34
Day-time diastolic blood pressure (mmHg)	0.14	0.11
Night-time systolic blood pressure (mmHg)	0.22	0.01[a]
Night-time diastolic blood pressure (mmHg)	0.33	< 0.01[a]
Continuous factors	Correlation coefficient	$p < 0.01$[a]
Gender		
Women	32.6 ± 3.7	
Men	36.2 ± 4.3	
Dipping status		0.15
Yes	34.5 ± 4.5	
No	35.6 ± 4.3	

AHI apnea-hypopnea index, BMI body mass index, CPAP continuous positive airway pressure, OSA obstructive sleep apnea

[a] Selected factors included in multivariable analysis

Table 3 Variables associated with aortic root size corrected by height, influence of nocturnal blood pressure

	Values	*p*
Clinical characteristics		
Age (years)	0.21	0.01*
Gender		
Men	19.9 ± 2.3	0.13
Women	20.6 ± 2.3	
BMI (kg/m^2)	0.11	0.21
OSA parameters at diagnosis		
Epworth scale score	0.17	0.16
AHI (event/h)	0.05	0.59
Whole-night nocturnal oxygen saturation (%)	0.02	0.86
OSA parameters under CPAP		
Duration of ventilation (years)	0.17	0.07*
CPAP nightly adherence (h/night)	− 0.05	0.06
Residual AHI (event/h)	0.04	0.69
Clinical blood pressure		
Systolic blood pressure (mmHg)	0.12	0.18
Diastolic blood pressure (mmHg)	0.09	0.30
24-h blood pressure monitoring		
24-h systolic blood pressure (mmHg)	0.14	0.11
24-h diastolic blood pressure (mmHg)	0.11	0.21
Day-time systolic blood pressure (mmHg)	0.08	0.36
Day-time diastolic blood pressure (mmHg)	0.03	0.71
Night-time systolic blood pressure (mmHg)	0.21	0.02*
Night-time diastolic blood pressure (mmHg)	0.25	< 0.01*
Dipping status		0.051*
Yes	20.0 ± 2.3	
No	20.9 ± 2.3	
Categorical factors	Mean aortic root size corrected by height	*p*
Gender		0.13
Women	19.9 ± 2.3	
Men	20.6 ± 2.3	
Dipping status		0.051*
Yes	20.0 ± 2.3	
No	20.9 ± 2.3	

Asterix represent the varaibles included int he multivariable analysis

AHI apnea-hypopnea index, BMI body mass index, CPAP continuous positive airway pressure, OSA obstructive sleep apnea, SD standard deviation

one did not find any relationship between aortic size and hypertension [16]. In the latter work, the authors excluded patients with a previous cardiovascular disease, whereas patients had a high cardiovascular risk in our study. The relationship between aortic root size and different cardiovascular risk factors is known. Thus, a report of the Cardiovascular Health Study showed that the presence of an aortic root dilatation in a population of patients older than 65 years was associated with different cardiovascular risk factors, in particular a high diastolic BP [17]. Likewise, in the longitudinal community study of Framingham, an increased BP was associated with aortic root remodeling during follow-up [9]. The harmful effect of BP on the aortic size is probably increased by the association of different cardiovascular risk factors. Aortic root enlargement is the reflection of atherosclerosis and can be considered as a target organ damage of hypertension associated with other

Table 4 Comparisons of clinical, respiratory, and blood pressure parameters with nocturnal blood pressure control

Variables	Uncontrolled nocturnal BP group, ≥ 120 mmHg for SBP and/or ≥ 70 mmHg for DBP ($n = 82$)	Controlled nocturnal BP group, < 120 mmHg for SBP and < 70 mmHg for DBP ($n = 60$)	p
Clinical data			
Age (years)	62 (54, 68)	62 (55, 68)	0.85
Women	19 (23.2)	29 (48.3)	0.01
Weight (kg)	98.0 (89.0, 118.0)	94.0 (84.0, 105.0)	0.063
Height (cm)	173.6 ± 8.6	169.6 ± 10.2	0.01
Body mass index (kg/m^2)	32.8 (29.7, 38.7)	32.2 (29.0, 35.9)	0.44
Abdominal circumference (cm)	120.1 ± 16.7	113.7 ± 14.7	0.03
Cardiovascular risk factor			
Diabetes mellitus	38 (46.3)	20 (33.3)	0.12
Obesity	60 (73.2)	40 (66.7)	0.40
Current smoker	17 (20.7)	12 (20.0)	0.91
Dyslipidemia	51 (62.2)	39 (65.0)	0.73
Prior cardiovascular event			
Stroke	11 (13.4)	7 (11.7)	0.76
Coronary artery disease	16 (19.5)	8 (13.3)	0.33
Atrial fibrillation	10 (12.2)	4 (6.7)	0.28
OSA parameters at diagnosis			
AHI (event/h)	40.9 (30.0, 57.0)	38.8 (32.0, 56.0)	0.92
Nocturnal oxygen saturation (%)	93.0 (92.0, 94.0)	93.0 (92.5, 95.0)	0.31
OSA parameters under CPAP			
Residual AHI (events/h)	2.0 (1.0, 3.8)	2.0 (1.2, 4.7)	0.78
CPAP nightly adherence (h/night)	6.5 (5.5, 7.5)	7.0 (5.5, 7.5)	0.69
Duration of ventilation (years)	4.4 (2.4, 7.5)	2.8 (1.4, 7.5)	0.12
Biological data			
GFR, CKD-EPI (mL/min)	82.2 (56.6, 93.7)	79.2 (72.1, 87.3)	0.78
LDL cholesterol (g/L)	2.6 (2.0, 3.3)	3.1 (2.3, 3.6)	0.27
Echocardiography data			
Aorta diameter (mm)	35.8 ± 4.6	33.7 ± 3.8	0.01
Aorta diameter normalized for height (mm/m)	20.6 ± 2.4	20.0 ± 2.2	0.10
Aortic root diameter ≥ 40 mm	16 (19.5)	3 (5.0)	0.01

Values are presented as count (percentage), mean ± SD or median (interquartile range)

AHI apnea-hypopnea index, BMI body mass index, BP blood pressure, CPAP continuous positive airway pressure, DBP diastolic blood pressure, GFR glomerular filtration rate, LDL low-density lipoprotein, OSA obstructive sleep apnea, SD standard deviation, SBP systolic blood pressure

cardiovascular risk factors but not with diabetes [18]. The understanding of the different mechanisms that influence aortic root enlargement is crucial because aortic dilatation is associated with major adverse cardiovascular events and mortality.

The relationship between aortic root size and sleep breathing disorders has already been described. In a population of patients without cardiovascular risk factor, Baguet et al. showed that the size of the aortic root measured by ultrasound was independently influenced by nocturnal diastolic BP [4]. Nocturnal BP is associated with a higher target organ damage in hypertension. OSA influences the BP level by increasing the sympathetic activity and peripheral vascular resistances. The influence of nocturnal BP on target organ damage is not fully understood. Nocturnal BP may increase the level of BP over the nychthemeron. A higher BP level could also cause a higher endothelial dysfunction which affects the aortic wall. A higher level of nocturnal BP pressure is also related to a higher level of central BP and higher aortic stiffness.

Treatment of OSA by CPAP has shown a modest but beneficial effect on BP. The BP decrease is more important when the OSA is severe or when the BP level is high. The HIPARCO trial, performed in a cohort of patients with resistant hypertension, showed an improvement of the nocturnal BP pattern and of diastolic BP on CPAP [19]. The decrease in diastolic BP is probably influenced by the correction of intermittent hypoxia [20]. The reduction of BP is associated with the duration of

nocturnal CPAP. In our work, we chose to focus on a population with a high level of CPAP adherence [21]. We showed, like Baguet et al., that aortic root size was still influenced by nocturnal diastolic BP despite a satisfactory correction of nocturnal respiratory events [4]. As our population had a high level of CPAP adherence and residual AHI was low, we assume that most of the involved mechanisms (nocturnal hypoxemia, sympathetic activity) were corrected. The regular use of BP monitoring is necessary to assess optimal BP control in this specific population. The evaluation of nocturnal BP is difficult because of a higher nocturnal BP variability in OSA patients [22]. An interval of 15 min between each BP measurement is probably too long to accurately evaluate the real level of nocturnal BP. Moreover, the nocturnal BP variability in a population treated by CPAP for a long time needs to be evaluated.

The influence of nocturnal BP on aortic dilatation was not demonstrated in this study since there was no follow-up. However, the association between nocturnal BP and aortic root size should be integrated in the design of further studies testing the potential benefit of CPAP therapy on aortic dilatation. This point is of utmost importance because OSA has shown its influence on the aortic size [4, 5]. Previous reports have demonstrated the association between OSA and aortic diseases [6–8]. The link between aortic dissection and OSA is not completely clear, but the harmful influence of BP swings, of higher BP levels, and of a sympathetic overactivity are relevant explanations provided by the authors. Aortic dissection is a rare disease but with a poor prognosis during follow-up. We have recently shown that OSA affects the expansion rate of the residual false lumen after aortic dissection [23]. Likewise, we found that an increased nocturnal BP was a strong predictor of aortic event after type B aortic dissection [24]. Further studies, with a closely monitored nocturnal BP, should evaluate the potential protective effect of CPAP therapy on the different aortic diseases. We can assume that CPAP treatment of OSA patients suffering from aortic disease has a real implication. An association between aortic expansion and OSA has been described in severe OSA [25]. As CPAP is very effective to lower the BP in case of severe OSA, we can hypothesize a positive effect on slowing the progression of aortic disease by decreasing BP. CPAP corrects other mechanisms involved in aortic disease as BP swings and negative intrathoracic pressure.

This work needs to be interpreted with its limits: (1) the monocentric design and a relatively modest sample of population, (2) the diagnosis of OSA was made in different sleep laboratories, and (3) there was no prospective evaluation of the aortic root dilatation.

Conclusion

Aortic root diameter is associated with an elevated BP and nocturnal hypoxemia. The factors that influence aortic root dilatation in case of non-treated OSA are numerous. In our population with a high degree of nocturnal CPAP adherence, nocturnal diastolic BP was still associated with the aortic root size. This finding should be integrated in further studies that evaluate the potential benefit of nocturnal ventilation of OSA in various aortic diseases.

Compliance with ethical standards

Conflicts of interest The authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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Comment

This is an excellent study which demonstrates that aortic root size is correlated with nocturnal BP in patients with treated hypertension and OSA. Interpretation of previous studies has been limited by the influence of under treatment. This study implies that clinicians treating hypertension in this population should consider using night-time BP as a treatment target to potentially mitigate the effect of progressive aortic dilatation.

Ian Wilcox
NSW, Australia