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Cardiovascular capacity in adults with obstructive sleep apnoea

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SUMMARY

BACKGROUND: Obstructive sleep apnoea (OSA) patients have a decreased exercise capacity. Potential cardiovascular capacity in patients with OSA with different severity without known cardiovascular disease has not been described.

OBJECTIVE: To evaluate impaired potential exercise cardiovascular capacity during cardiopulmonary exercise testing (CPET) in OSA patients and to compare cardiovascular performance reaction with different severity during graded exercise stages.

DESIGN: All participants were accompanied without cardiovascular disease, especially hypertension and arrhythmia. Parameters of different stages were compared between subjects with and without OSA, and among OSA patients with varied severity.

RESULTS: Despite having significantly higher peak exercise diastolic blood pressure (DBP) and ventilatory equivalent for CO_2 ($EQCO_2$), patients with OSA had a lower peak oxygen uptake (VO_2), heart rate (HR), heart rate recovery (HRR) and respiratory reserve (BR) than normal subjects. Furthermore, significant correlations were found between VO_2, DBP, $EQCO_2$, HRR, BR and the apnoea-hypopnea index. In severe OSA, there was a greater difference in HR and HRR during the anaerobic threshold stages.

CONCLUSIONS: OSA patients demonstrate reduced potential cardiovascular capacity, even without documented cardiovascular disease. Patients with severe OSA develop impaired exercise capacity at early stage during exercise. These data point to exaggerated haemodynamic response to graded exercise and delayed post-exercise cardiovascular response recovery in OSA patients. CPET can be a supplement for assessment of OSA severity.

KEY WORDS: OSA; DBP; SBP; VO_2; cardiopulmonary exercise testing

Obstructive sleep apnoea (OSA) is characterised by repetitive nocturnal upper airway collapse, subsequent hypoxic episodes and micro-awakenings, interrelating with high cardiovascular morbidity and mortality.[1] OSA patients are particularly prone to experiencing an elevated risk of several adverse cardiovascular consequences, including congestive heart failure, hypertension, arrhythmia, and coronary artery disease.[2] The precise mechanisms involving cardiovascular dysfunction and the severity of OSA have not been thoroughly determined, but chronic repetitive sleep apnoea can cause hypoxia, carbon dioxide (CO_2) retention, autonomic and hormonal imbalance, endothelial dysfunction and oxidative stress.[3,4] These physiology disturbances can result in uncontrolled variations in blood pressure and heart rate.

Cardiovascular disturbance is considered as one of the most severe comorbidities of OSA.[5] Substantial evidences have shown that sleep apnoea significantly increase the risk of ischaemic heart disease.[3] Although the apnoea-hypopnea index (AHI) by overnight laboratory polysomnography (PSG) is always regarded as the gold standard in diagnosing OSA, the limitation of availability and substantial cost of PSG contributes to decreasing detection and the growing number of undiagnosed individuals.[6] Due to limited specificity and sensitivity, these approaches have recently attracted low acceptance. For example, echocardiography can detect cardiac dysfunction,[7] but it cannot evaluate cardiac function in different exercise loads and latent cardiopulmonary insufficiency.

Despite the ample evidence of cardiovascular risk in OSA patients, little is known about the cardiopulmonary capacity to exercise in these patients. Cardiopulmonary exercise testing (CPET) provides an integrative non-invasive assessment to evaluate cardiopulmonary, neuropsychological, haematopoietic and muscular systems functions, which can provide a valuable cardiovascular instrument for risk stratification and prognosis assessment. It has been proved that individuals with OSA have altered cardiopulmonary and metabolic capacity, which can potentially

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impair their ventilatory and metabolic reactivity to graded exercise.[8,9] Many epidemiological studies revealed that cardiopulmonary response to graded exercise is a critical endpoint measure, especially blood pressure and heart rate response (HRR).[10,11] Hypertensive response to graded exercise is not only independently correlated with increased risk of subsequent arterial hypertension, but also used to determine hypertension risk.[12]

Although OSA is associated with hypertension, references regarding cardiovascular response of normotensive OSA patients with different severity during exercise are rare. The aim of this study was 1) to evaluate impaired potential exercise cardiovascular capacity during CPET in OSA patients, and 2) to compare cardiovascular performance reaction of OSA patients with different severity during the various graded exercise stage.

METHODS

Study design and participants

Sixty-one eligible patients with an AHI score of >5 events/h from the First Affiliated Hospital of Soochow University, Suzhou, China, were included. Twenty patients with AHI <5 events/h were also invited. All patients were excluded from the study if they had 1) obstructive or restrictive lung disease confirmed using spirometry with forced expiratory volume in 1 sec percentage predicted (FEV_1 %pred) <70%, performed few hours before CPET; 2) known hypertension; 3) diabetes or a fasting blood glucose >6.1 mmol/L; 4) known valvular heart disease; 5) known neuromuscular disease that could affect their exercise capacity; 6) abnormal thyroid function. All participants underwent a heart ultrasound, blood pressure, heart rate and simple spirometric. All participants provided written informed consent. The project was approved by the Clinical Institutional Ethics Committee of Soochow University, Suzhou, China.

Data collection

Polysomnography

Nocturnal polysomnography was performed according to the standard instrumentation and procedures.[13] Obstructive apnoea was defined as the disappearance of airflow for more than 10 sec in the presence of continued respiratory effort. Hypopnoea was defined as a reduction of respiratory airflow in more than 50% of the airflow, accompanied by a decrease in blood oxygen saturation of more than 4%. The AHI is defined as the average number of episodes of apnoea and hypopnoea episodes per hour of night time sleep according to the consensus guidelines. Based on AHI results, OSA severity was classified as normal (<5 events/h), mild and moderate (5–30 events/h) and severe (>30 events/h). The Epworth Sleeping Scale (ESS) was also recorded for each patient.

Cardiopulmonary exercise testing

Maximum, symptom-limited, incremental CPET using a Masterscreen CPX System (Jaeger Corp, Hoechberg, Germany) was employed for ventilatory expired gas analysis.[14] Graded exercise protocols were performed according to subjects itself with different circumstances. Exercise capacity was gradually increased using a ramp protocol (10–25 W/min) after a 3-min reference warm-up. Gas exchange data on oxygen (O_2) and CO_2 were measured using the breath-by-breath technique with the online system. Anaerobic threshold was determined using the V-slope method. This provided data on the respiratory gas exchange, including oxygen uptake (VO_2), ventilatory equivalent for CO_2 ($EQCO_2$), respiratory reserve (BR) and heart rate recovery (HRR). The VO_2 is the most important parameter during CPET.[15,16] The CPET was conducted under continuous monitoring of heart rate (HR), 12-lead electrocardiogram, and pulse oxygen saturation (SpO_2), while systolic (SBP) and diastolic (DBP) blood pressure was recorded every 2 min with a cuff sphygmomanometer. Exercise subjects were encouraged to try their best to exhaustion. All study participants completed the test without early termination.

The whole cardiopulmonary exercise is divided into the reference stage, anaerobic threshold stage, peak exercise stage and the 3-min recovery stage. Indications for exercise termination included myocardial ischaemia, extreme fatigue, Grade 2 or Grade 3 atrio-ventricular block, complex ventricular premature beats, SpO_2 < 80%, a sudden drop in BP levels by more than 20 mmHg, increased SBP >220 mmHg or DBP > 120 mmHg, dizziness, confusion and sudden pallor.

Pulmonary function tests

Lung function was tested using a computerised spirometer (Jaeger Corp), following standard procedures recommended by the American Thoracic Society to exclude pulmonary disease. FEV_1 %pred, FVC %pred and FEV_1/FVC ratio were measured in every subject before CPET.

Echocardiography

Comprehensive echocardiography of all subjects was performed using commercially available ultrasound machines before CPET. Left ventricular ejection fraction (LVEF) was estimated using Simpson's rule. The end-systolic and left ventricle diameters were measured from the short-axis views, and the ejection fraction (EF) was derived from these measurements.

Statistical analyses

Statistical Product and Service Solutions v19.0 data analysis software system (IBM Corp, Armonk, NY,

USA) was employed for statistical analysis. Subject characteristics are given as means ± standard deviations (SDs), unless stated otherwise. Samples *t*-test was used to compare continuous variables, while a χ^2 test was performed with categorical variables. Pearson's correlation coefficient was used to perform correlation analysis. $P < 0.05$ was considered statistically significant.

RESULTS

The baseline characteristics of the two study groups are given in Table 1. No significant differences were found in age, BMI, LVEF, SBP and DBP at rest between two groups. The patients' pulmonary function testing (PFT) revealed normal results without any abnormalities. Significant differences were noted in AHI, the Mallampati oropharyngeal score, nasal obstructive disease and ESS.

Exercise capacity was significantly reduced and blunted in the OSA group, with lower HRR and HR %predicted at peak exercise response and recovery response ($P < 0.05$) (Table 2). At the peak exercise stage, individuals with OSA exhibited significantly lower values of VO_2 max, DBP and BR, along with higher $EQCO_2$, than the control group ($P < 0.05$). However, there was no significant difference in SBP between the OSA group and the control group overall ($P > 0.05$).

At the peak exercise stage, VO_2 max was significantly correlated with AHI ($r = -0.542$; $P < 0.01$); peak DBP and $EQCO_2$ had a positive relationship with AHI ($r = 0.572$, $r = 0.420$; $P < 0.05$), while BR and HRR were negatively related to AHI ($r = -0.420$, $r = -0.509$; $P < 0.05$). HR% predicted was negatively related to AHI ($r = -0.420$, $r = -0.406$; $P < 0.05$) (Table 3). These CPET indicators had no statistically significant relationship with mean SpO_2, lowest SpO_2 and ESS.

Table 4 gives a summary of different OSA severity during CPET. Mild-moderate OSA had a significantly higher DBP, lower HRR and lower HR% predicted than participants without OSA during the peak exercise and recovery stages ($P < 0.05$). More importantly, significant differences in HR %predicted and HRR were noted in severe OSA compared to mild-moderate OSA and in controls in the anaerobic threshold stage ($P < 0.05$). However, SBP, DBP were not different in participants with severe OSA at the anaerobic threshold from controls and those with mild-moderate OSA ($P > 0.05$).

DISCUSSION

The main findings of this study are 1) untreated OSA patients without documented cardiovascular disease demonstrate decreased VO_2 max, DBP responses, chronotropic impairment and a prolonged HRR during symptom-limited exercise testing compared to controls. 2) Furthermore, the reduced VO_2 max in patients with OSA was associated with lower peak HR, lower BR, slightly higher peak DBP, higher $EQCO_2$ and impaired HRR compared to controls. 3) In cases of severe OSA, notable differences in HRR

Table 1 Baseline characteristics and polysomnography results of patients with obstructive sleep apnoea and controls

Characteristics	Controls ($n = 20$) mean ± SD	OSA ($n = 61$) mean ± SD	*P* value
Age, years	43.15 ± 9.42	46.03 ± 12.06	0.33
Sex, male:female	17:3	54:7	—
BMI, kg/m^2	27.09 ± 3.65	28.20 ± 3.68	0.24
LVEF, %	65.10 ± 4.63	64.93 ± 4.03	0.89
AHI, events/h	2.5 ± 1.48	39.31 ± 22.18	<0.05
Mean SpO_2, %	97.05 ± 1.27	92.76 ± 5.45	<0.05
Lowest SpO_2, %	93.25 ± 1.29	75.19 ± 11.79	<0.05
Neck circumference, cm	37.80 ± 5.10	38.69 ± 3.41	0.37
Abdominal circumference, cm	95.55 ± 12.81	95.32 ± 12.13	0.94
Mallampati oropharyngeal score 1–4	2.3 ± 0.9	3.8 ± 0.6	<0.05
Nasal obstructive disease, *n* (%)	4 (20)	29 (48)	0.03
Allergic rhinitis	2 (10)	21 (34)	0.04
Nasal septal deviation	1 (5)	17 (28)	0.03
Nasal polyp	1 (5)	8 (13)	0.44
SBP, mmHg	124.0 ± 15.50	126.70 ± 20.25	0.58
DBP, mmHg	73.75 ± 11.41	77.33 ± 11.25	0.22
HR, bpm	86.0 ± 15.0	85 ± 14.8	0.31
Smoke, %	9 (45.00)	29 (47.54)	0.67
Epworth Sleeping Scale score	3.80 ± 2.95	13.16 ± 5.78	<0.05
FVC, %predicted	99.14 ± 6.79	96.76 ± 8.63	0.26
FEV_1, %predicted	94.57 ± 7.31	93.59 ± 9.57	0.67
FEV_1/FVC	81.48 ± 4.63	81.68 ± 5.51	0.88

SD = standard deviation; OSA = obstructive sleep apnoea; BMI = body mass index; LVEF = left ventricular ejection fraction; AHI = apnoea-hypopnea index; SpO_2 = oxyhaemoglobin saturation; SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; FVC = forced vital capacity; FEV_1 = forced expired volume in 1 sec.

Table 2 Comparison of cardiopulmonary indicators of groups

Variable	Controls ($n = 20$) mean ± SD	OSA ($n = 61$) mean ± SD	*P* value
Anaerobic threshold			
VO_2, %predicted	58.75 ± 9.30	56.38 ± 12.50	0.43
SBP, mmHg	156.70 ± 12.22	158.40 ± 19.19	0.69
DBP, mmHg	73.73 ± 11.41	75.59 ± 11.15	0.58
HRR, bpm	30.45 ± 7.65	28.78 ± 10.56	0.51
HR, %predicted	72.75 ± 8.54	69.57 ± 9.20	0.17
$EQCO_2$	29.59 ± 3.04	31.04 ± 3.17	0.07
BR	65.25 ± 10.73	66.28 ± 11.20	0.72
Peak exercise			
VO_2 max, %predicted	99.10 ± 17.35	91.72 ± 13.35	<0.05
SBP, mmHg	188.90 ± 7.40	186.70 ± 3.61	0.54
DBP, mmHg	79.80 ± 2.30	85.79 ± 1.90	<0.05
HRR, bpm	25.34 ± 9.76	20.98 ± 7.87	<0.05
HR, %predicted	97.80 ± 6.13	90.8 ± 5.60	<0.05
$EQCO_2$	25.75 ± 3.24	32.68 ± 4.56	<0.01
BR	36.70 ± 7.15	29.16 ± 7.27	<0.01
Recovery			
SBP, mmHg	149.00 ± 17.08	150.60 ± 16.38	0.57
DBP, mmHg	75.65 ± 9.49	76.77 ± 9.35	0.64
HRR, bpm	46.08 ± 9.56	38.80 ± 1.80	<0.05
$EQCO_2$	29.73 ± 2.32	29.05 ± 2.76	0.32
BR	34.55 ± 5.84	31.15 ± 7.92	0.08

SD = standard deviation; OSA = obstructive sleep apnoea; VO_2, %predicted = peak oxygen consumption percentage of predicted; SBP = systolic blood pressure; DBP = diastolic blood pressure; HRR = heart rate recovery; HR = heart rate; $EQCO_2$ = ventilatory equivalent for CO_2; BR = respiratory reserve.

and HR during the anaerobic threshold stage can be observed. Patients with severe OSA develop impaired exercise cardiovascular capacity at an early stage during exercise.

Impaired cardiovascular function of OSA was observed in our study, which was consistent with previous studies.[17–19] Normally, decreased sympathetic and increased parasympathetic activity helps lower BP and HR.[20] However, intermittent hypoxia in OSA leads to four cardiovascular consequences: 1) intermittent hypoxemia-reoxygenation fluctuations, resulting in oxidative stress and inflammation;[20] 2) excessive arousals resulting in decreased parasympathetic activity and increase sympathetic activation;[21] 3) large negative intrathoracic pressure swings;[2,22,23] and 4) intermittent hypoxemia, resulting in oxidative stress and increased inflammatory mediators and cytokines, which contribute to endothelial dysfunction,[20] decreased parasympathetic activity and increase sympathetic activity. These are the main probable causes of potential cardiovascular insufficiency in OSA patients.

As the most comprehensive indicator of pulmonary-cardiovascular-musculoskeletal-haematological function, a reduced peak VO_2 in patients suffering from OSA has been observed in previous studies.[11,15,23] Other indicators of CPET assist us in determining cardiovascular, pulmonary and metabolic capacity.[4] Significant lower VO_2 max is not only confirmed in patients with OSA, but is also positively correlated with AHI severity at peak exercise stage in our study. Meanwhile, AHI is associated with lower peak HR, lower peak BR, slightly higher peak DBP, higher $EQCO_2$ and impaired HRR.[4,22,24,25] Chronic nocturnal hypoxemia and hypercapnia lead to decreased sensitivity of central and peripheral chemoreceptors in OSA patients, resulting in decreased vascular endothelial function, increased angiotensin activity and decreased species of reactive oxygen species, thus leading to changes in cardiopulmonary function after strenuous exercise.[20,26]

Our study focused on individuals with OSA who did not exhibit symptoms of cardiovascular disease

Table 3 Correlations of cardiopulmonary capacity and the severity of OSA

Variable	VO_2 max	DBP peak	BR peak	$EQCO_2$ peak	HRR peak	HR peak, %predicted
AHI	−0.542*	0.572†	−0.420†	0.412†	−0.509†	−0.406†
Mean SpO_2	0.161	−0.265	−0.240	0.36	−0.131	0.137
Lowest SpO_2	0.099	−0.185	0.295	−0.11	−0.251	0.171
Epworth Sleeping Scale score	−0.137	−0.016	−0.249	0.03	−0.212	−0.172

* $P < 0.01$.
† $P < 0.05$.
OSA = obstructive sleep apnoea; VO_2, %predicted = peak oxygen consumption percentage of predicted; DBP = diastolic blood pressure; BR = respiratory reserve; $EQCO_2$ = ventilatory equivalent for CO_2; HRR = heart rate recovery; HR = heart rate; AHI = apnoea-hypopnea Index; SpO_2 = oxyhaemoglobin saturation.

Table 4 Contrast of cardiovascular indicators among groups with different severity

Variable	Control* ($n = 20$) mean ± SD	Mild–moderate OSA ($n = 28$) mean ± SD	Severe OSA ($n = 33$) mean ± SD
Anaerobic threshold			
SBP, mmHg	156.70 ± 12.22	157.10 ± 9.89	158.80 ± 12.62
DBP, mmHg	73.73 ± 11.41	74.13 ± 12.17	75.55 ± 11.33
HRR, bpm	30.45 ± 7.65	28.58 ± 7.65	24.12 ± 8.90†‡
HR, % predicted	72.75 ± 8.54	71.38 ± 11.24	66.21 ± 8.66†‡
Peak exercise			
SBP, mmHg	188.90 ± 7.40	193.3 ± 9.48	194.30 ± 11.55
DBP, mmHg	79.80 ± 2.30	86.10 ± 13.11†	91.8 ± 8.79†‡
HRR, bpm	25.34 ± 9.76	19.71 ± 8.98†	14.89 ± 9.15†‡
HR, %predicted	97.80 ± 9.13	88.13 ± 10.02†	80.42 ± 8.84†‡
Recovery			
SBP, mmHg	149.06 ± 17.08	148.40 ± 17.87	150.30 ± 14.15
DBP, mmHg	75.65 ± 9.49	75.13 ± 6.93	77.18 ± 9.90
HRR, bpm	46.18 ± 9.56	41.11 ± 7.56†	34.76 ± 7.13†‡
HR, %predicted	69.09 ± 7.18	64.80 ± 6.64†	60.55 ± 7.17†‡

* Control (AHI <5 events/h); mild–moderate OSA (AHI 5–30 events/h); severe OSA (AHI >30 events/h).
† Differs from controls, $P < 0.05$.
‡ Differs from mild–moderate OSA, $P < 0.05$.
SD = standard deviation; OSA = obstructive sleep apnoea; SBP = systolic blood pressure; DBP = diastolic blood pressure; HRR = heart rate recovery; HR = heart rate; AHI = apnoea-hypopnea Index.

and had no respiratory disorders, as confirmed by pulmonary function tests conducted prior to CPET. We discovered that patients with severe OSA demonstrated reduced HRR and a lower percentage of predicted heart rate (HR%) at the anaerobic threshold stage during exercise. These findings suggest that impaired functioning of skeletal muscles and pulmonary vascular beds could be contributing factors to the early decline in exercise capacity observed in individuals with severe OSA.[27–29] CPET can not only detect potential impaired cardiovascular function, but can also help compensate for the shortcomings of using AHI alone to define OSA severity. Moreover, early exercise damage helps patients with severe OSA avoid the side effects and risks caused by the strong increase of exercise load during graded exercise.

Our study had several limitations. Other PSG measures, such as nadir SpO_2, micro-arousal, hypoxic burden or hypercapnia reflecting OSA severity were not measured. Whether these indices are potent prognostic markers in patients with OSA need to be confirmed by larger prospective studies. Future studies aiming to explore the specific relationship of cardiac impairment at earlier stage of OSA patients might also be an interesting field of research.

CONCLUSION

OSA patients demonstrate reduced potential cardiovascular capacity, even without documented cardiovascular disease. Patients with severe OSA develop impaired exercise capacity at an earlier stage during exercise. These data point to exaggerated haemodynamic response to graded exercise and delayed post-exercise cardiovascular response recovery in OSA patients. CPET can be used as additional tool for the assessment of OSA severity.

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Conflicts of interest: none declared.

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RÉSUMÉ

CONTEXTE : Les patients souffrant d'apnée obstructive du sommeil (OSA) ont une capacité d'exercice réduite. La capacité cardiovasculaire potentielle des patients atteints de OSA de gravité différente et ne souffrant pas de maladie cardiovasculaire connue n'a pas été décrite.

OBJECTIF : Évaluer l'altération de la capacité cardiovasculaire potentielle à l'exercice pendant le test d'effort cardio-pulmonaire (CPET) chez les patients souffrant de OSA et comparer la réaction de la performance cardiovasculaire avec différentes sévérités pendant des étapes d'exercice graduées.

MÉTHODE : Tous les participants ne présentaient pas de maladie cardiovasculaire, en particulier d'hypertension et d'arythmie. Les paramètres des différentes étapes ont été comparés entre les sujets avec et sans OSA, et entre les patients atteints d'OSA de gravité variée.

RÉSULTATS : Bien que la pression artérielle diastolique (DBP) et l'équivalent ventilatoire du CO_2 ($EQCO_2$) soient significativement plus élevés à l'effort, les patients souffrant d'OSA avaient une absorption maximale d'oxygène (VO_2), une fréquence cardiaque (HR), une récupération de la fréquence cardiaque (HRR) et une réserve respiratoire (BR) inférieures à celles des sujets normaux. En outre, des corrélations significatives ont été trouvées entre VO_2, DBP, $EQCO_2$, HRR, BR et l'indice d'apnée-hypopnée. Dans l'OSA sévère, il y avait une plus grande différence dans la HR et la HRR pendant les étapes du seuil anaérobique.

CONCLUSIONS : Les patients souffrant d'OSA présentent une capacité cardiovasculaire potentielle réduite, même en l'absence de maladie cardiovasculaire documentée. Les patients souffrant d'un OSA sévère développent une capacité d'exercice réduite à un stade précoce de l'exercice. Ces données indiquent une réponse hémodynamique exagérée à l'exercice graduel et une récupération retardée de la réponse cardiovasculaire après l'exercice chez les patients souffrant d'OSA. Le CPET peut être un complément pour l'évaluation de la gravité de l'OSA.