Benefits of supervised community physical activity in obstructive sleep apnoea

To the Editor:

Obstructive sleep apnoea (OSA) is a chronic disorder characterised by repetitive episodes of partial or complete airway obstruction occurring during sleep [1]. Recent data suggest that moderate-to-severe OSA is highly prevalent, affecting up to 50% of males and 25% of females [2]. OSA is associated with frequent cardiovascular and cerebrovascular events [3]. Although continuous positive airway pressure (CPAP) is the gold standard treatment, long-term adherence remains poor, particularly in moderate and in non-sleepy patients [4], while the cardiovascular risk remains high.

Exercise has attracted attention by improving apnoea–hypopnoea index (AHI), daytime sleepiness and quality of life [5–8]. However, previous studies had several drawbacks including small sample size, too-specific populations, lack of randomisation and short-term follow-up, while OSA patients need long-term management.

Therefore, the aim of the present randomised controlled trial was to assess the effectiveness of a 9-month exercise programme undertaken outside hospital as a treatment for moderate OSA, defined by reaching an AHI <15 events·h^{-1} at follow-up.

Between June 2015 and September 2017, we screened 595 participants from the general population as well as 30 from the sleep laboratory of the University Hospital of Saint-Etienne (France). Eligible criteria were AHI in the range 15–30 events·h^{-1} and age 40–80 years. Exclusion criteria were current treatment for OSA; cardiovascular or respiratory comorbidities and/or Epworth Sleepiness Scale (ESS) score >10 justifying immediate initiation of CPAP; respiratory or heart disease contraindicating exercise discovered during stress testing; and Parkinson's disease. The study was approved by an institutional review board (CPP Sud-Est 1 #1508033, France) and registered with ClinicalTrials.gov (NCT02463890).

Eligible patients were assigned randomly to the exercise or to the control group after obtaining written informed consent. Patients randomised to the exercise group were enrolled in an exercise programme (NeuroGyV) performed in clubs of a non-competitive sports federation, the French Federation for Physical Education and Voluntary Gymnastics (FFEPGV). The programme combined three different supervised sessions of exercise per week during the 9 months of the FFEPGV annual activity: nordic walking, aquagym and gymnastics. Each session lasted 60 min, including 10 min of warm-up, 40 min of combined resistance and aerobic exercises at the anaerobic threshold and 10 min of cool-down, complying with public health recommendations. Control group participants attended two group educational sessions about healthy diet and physical activity recommendations.

All patients underwent a polygraphic recording (Nox T3; Nox Medical, Reykjavik, Iceland) at baseline and 9 months. Examinations were manually scored twice according to the 2012 American Academy of Sleep Medicine criteria [9], by a single scorer blinded to treatment assignment and OSA severity was defined as mild (5⩽AHI<15 events·h^{-1}), moderate (15⩽AHI<30 events·h^{-1}) or severe (AHI ⩾30 events·h^{-1}). A maximal cardiopulmonary exercise test (CPET) was performed on a cycloergometer including breath-by-breath respiratory gas exchange measurements (Jaeger Vyntus CPX; CareFusion, Hoechberg, Germany). Medical history was recorded and anthropometric data measured. Daytime sleepiness was assessed using the ESS and subjective sleep quality using the Pittsburgh Sleep Quality Index (PSQI); daily physical activity was evaluated using the Population Physical Activity Questionnaire [10].

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Long-term physical activity programmes outside of hospitals may help improve moderate obstructive sleep apnoea, particularly in obese patients http://ow.ly/11jg30lOJYS

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With 80% power and α=0.05, 48 participants per group were required to detect a 30% treatment efficacy in the exercise group and 5% in the control group, assuming a 10% attrition rate. All analyses were conducted in intention-to-treat (ITT) using IBM-SPPS Statistics 24.0 (IBM, Armonk, NY, USA). Data are presented as mean±SD and frequencies.

We included 96 patients (mean age 63±7 years, mean body mass index (BMI) 28.5±4.2 kg·m^{-2}, 61% male, 71% hypertensive and 19% diabetic); 88 patients completed follow-up visits at 9 months. No significant differences were observed between groups for any of the baseline characteristics. No adverse events occurred. The average exercise attendance was 61 out of the 88 prescribed sessions (median 76%, interquartile range 63–88%).

A significantly higher proportion of patients in the exercise group (25 out of 43; 58%, 95% CI 43–74%) reached an AHI <15 events·h^{-1} after 9 months compared to the control group (nine out of 45; 20%, 95% CI 8–32%; Chi-squared p<0.0001). The significant effect of exercise remained after adjustment for age, sex, baseline BMI, AHI and moderate-to-vigorous physical activity (MVPA) (p<0.0001). None of the patients reached an AHI<5 events·h^{-1}.

Compared to controls, the exercise group demonstrated a significant decrease in AHI (−18% *versus* +6%; p=0.007). In addition, other parameters of OSA improved in the exercise group while oxygen saturation (S_{pO_2}) parameters worsened in the control group (table 1). Supine time during sleep did not change in any group (p=0.39). Clinically, the exercise group showed a significant drop in ESS and PSQI scores compared to the control, group in which sleep quality worsened. Interestingly, the intervention effect was significantly greater in obese patients compared to patients with a BMI <30 kg·m^{-2} after adjustment for initial AHI (p=0.037).

Peak oxygen consumption increased in the exercise group compared to controls on CPET, along with anaerobic threshold and peak oxygen pulse (table 1). Both MVPA and total daily energy expenditure increased in the exercise group.

TABLE 1 Group comparisons of sleep, exercise and anthropometrics parameters at baseline and change at 9 months

	Control		Exercise		Between-group difference	p-value
	Baseline	Change at 9 months	Baseline	Change at 9 months		
Subjects n	45		43			
Sleep parameters						
AHI events·h^{-1}	20.7±6.1	1.2±8.0	23.1±8.0	−4.1±9.7**	−5.3±8.8	**0.007**
Obstructive events·h^{-1}	5.8±4.2	1.4±5.5	7.8±6.4	−1.4±6.4*	−2.8±5.9	**0.016**
Central events·h^{-1}	1.1±1.9	0.1±1.9	1.2±2.4	0.2±1.9	0.1±1.9	0.990
Hypopnoea events·h^{-1}	13.9±5.3	−0.3±5.5	13.9±4.3	−2.7±6.3**	−2.4±5.9	0.064
Supine time % TST	33.8±26.4	−3.2±24.2	37.2±25.9	−8.2±26.1	−5.0±25.1	0.389
ODI events·h^{-1}	21.6±7.3	0.8±8.2	22.7±8.4	−2.2±9.1*	−3.0±8.6	0.116
Mean S_{pO_2} %	92.4±1.7	−0.6±1.2**	92.1±1.3	−0.3±1.2	0.3±1.2	0.342
Percentage of time S_{pO_2} <90%	11.6±17.5	4.5±18.4**	10.5±11.4	1.0±14.5	−3.5±16.5	**0.036**
ESS score	7.4±4.3	0.1±3.2	8.4±4.4	−1.8±4.1**	−1.7±3.6	**0.042**
PSQI score	7.0±3.3	0.8±2.4*	7.4±3.7	−0.6±2.8	−1.4±2.6	**0.015**
Exercise and anthropometric parameters						
Peak V_{O_2} mL·min^{-1}·kg^{-1}	22.8±5.7	0.3±2.4	23.6±6.0	2.3±3.1***	2.0±2.7	**0.001**
V_{O_2} at VT mL·min^{-1}·kg^{-1}	17.4±4.1	−0.4±3.6	18.2±5.0	1.8±4.2**	2.4±3.9	**0.001**
O_2 pulse mL·bpm^{-1}	12.7±3.1	0.5±1.5	13.4±3.3	1.4±1.7***	0.9±1.6	**0.031**
Sedentary min·day^{-1}	489±168	−2±158	497±220	−11±131	−9±145	0.758
LPA min·day^{-1}	261±118	−4±123	238±109	−3±64	1±94	0.667
MVPA MET-min·week^{-1}	2898±2087	79±1109	3295±2326	795±1841***	716±1467	**0.002**
DEE kJ·24 h^{-1}	11 556±1699	−25±846	11 676±2008	273±963**	298±903	**0.047**
Body mass index kg·m^{-2}	28.3±4.3	0.1±0.9	28.5±4.1	−0.1±0.9	0.2±0.9	0.422
Waist circumference cm	97.9±12.0	1.4±3.1**	100.7±12.6	0.1±2.9	−1.3±3.0	0.060
Waist-to-hip ratio	0.94±0.08	0±0.05	0.97±0.08	−0.02±0.03**	−0.02±0.04	0.067
Neck circumference cm	37.7±3.6	0.8±1.1***	38.8±3.3	0.3±1.2	−0.5±1.1	**0.028**

Data are presented as mean±SD, unless otherwise stated. AHI: apnoea–hypopnoea index; TST: total sleep time; ODI: oxygen desaturation index; S_{pO_2}: pulse oxygen saturation; ESS: Epworth Sleepiness Scale; PSQI: Pittsburgh Sleep Quality Index; V_{O_2}: oxygen consumption; VT: ventilatory threshold; LPA: light physical activity; MVPA: moderate-to-vigorous physical activity; MET: metabolic equivalent of task; DEE: daily energy expenditure. Significant changes from baseline values to 9 months within groups: *: p<0.05; **: p<0.01; ***: p<0.001 using paired t-test or Wilcoxon signed-rank test as appropriate. Differences in changes from baseline between groups were calculated using t-test or Mann–Whitney U-test as appropriate. Significant between-group differences are highlighted in bold.

Although weight and BMI were stable in both groups, neck circumference increased in the control group while waist-to-hip ratio decreased in the exercise group (table 1).

Overall, we showed that 58% of the moderate OSA patients reached an AHI <15 events·h^{-1} in response to a 9-month supervised community physical activity programme. However, the reduction in AHI of 4.1 events·h^{-1} found in the ITT group with exercise was lower than previously reported. This may be explained by more severe OSA in other trials [7, 8], major obesity, or younger age [5]; some trials included dietary interventions in the exercise group [8], which may catalyse the favourable effect of exercise training. Interestingly, our per-protocol analysis demonstrated an AHI improvement reaching −6.2±7.5 events·h^{-1}, which was consistent with results of a previous meta-analysis [11].

By contrast, OSA spontaneously increased in controls after 9 months, as evidenced by a higher time at S_{pO_2} <90% and lower mean S_{pO_2}, two independent predictors of sudden cardiac death [12].

The strength of our trial lies in the 9-month management outside the healthcare sector as compared to previous studies designed on shorter periods in hospital settings [5, 6, 8]. It is worth noting that the improvement in maximal oxygen uptake suggests benefits in cardiovascular risk reduction and improvement of life expectancy [13], whereas recent studies have revealed that CPAP therapy alone does not prevent cardiovascular mortality [14].

The main limitation of our study was the OSA assessment on a single night upon inclusion and follow-up, which may have introduced additional variability [15], even though it remains a standard. Moreover, participants were aware of their intervention assignments and some patients randomised to the control group may have trained independently; however, this would have reduced the magnitude of the effects of the programme we observed.

Our exploratory analyses revealed that the exercise effect was greater in obese patients, suggesting a potential role of change in pharyngeal fat mass distribution through exercise training which may contribute to reduce OSA severity in obese patients, although this was not assessed in the present study.

In conclusion, long-term physical activity programmes in real-life community settings may help improve moderate OSA, although AHI improvement remains modest. Obese patients are probably the best candidates for such programmes.

Mathieu Berger[1,2,3], Jean-Claude Barthélémy[1,2], David Hupin[1,2], Jérémy Raffin[1,2], Caroline Dupré[1], Pierre Labeix[1,2], Frédéric Costes[4,5], Jean-Michel Gaspoz[6] and Frédéric Roche[1,2]

[1]SNA-EPIS Laboratory, EA 4607, University of Lyon, Saint-Etienne, France. [2]Dept of Clinical and Exercise Physiology, University Hospital of Saint-Etienne, Saint-Etienne, France. [3]IRAPS, Fédération Française d'Education Physique et de Gymnastique Volontaire (FFEPGV), Montreuil, France. [4]Unit of Human Nutrition, INRA UMR 1019, University of Auvergne, Clermont-Ferrand, France. [5]Dept of Physiology, University Hospital Gabriel Montpied, Clermont-Ferrand, France. [6]Division of Primary Care Medicine, Dept of Community Medicine, Primary Care and Emergency Medicine, Geneva University Hospitals, Geneva, Switzerland.

Correspondence: Mathieu Berger, CHU Saint-Etienne, Hôpital Nord, Centre VISAS, 42055 Saint-Etienne Cedex 2, France. E-mail: mathieuberger@outlook.com

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