Sleep Science

CASE REPORT

The influence of intensity of exercise over periodical leg movement and obstructive sleep apnea syndrome: a case report

A influência da intensidade do exercício físico nos movimentos periódicos das pernas e na síndrome da apneia obstrutiva do sono: um relato de caso

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ABSTRACT

There is evidence that regular exercises promote a number of changes and physiological benefits, and can be considered a non-pharmacological intervention, which improves the sleep quality of people who really do not have any sleep disorders. Yet, few studies have assessed how exercise can benefit a specific population with sleep disturbance. The aim of this study was to examine the influence of an aerobic training (AT) program of variable intensity in a sample of volunteers who had periodic leg movement (PLM) and obstructive sleep apnea syndrome (OSAS). The 51 year-old volunteer, body mass index (BMI) 28.67 kg/m^2, sedentary, presenting PLM (rate>53.5/hour) and OSAS (AHI index >12.8/hour) was submitted to aerobic training on a treadmill in the mornings 3 times a week, each session lasting 40 minutes, thus totaling 72 sessions. Prior to aerobic training , the volunteer took a maximum effort test (MET) so that prescription of safe aerobic training could be made. The first aerobic training was done at 60% of the VO2peak so that acute effects of exercise could be assessed. Sessions 2 up to 24 were performed at ventilation threshold 1 (VT1) and sessions 25 to 48 were performed between ventilation threshold 1 and ventilation threshold 2 while sessions 49 to 72 were done between VT1 and VT2, with predominance at VT2. Polysomnographic and maximum effort test tests were carried out every 6 months throughout the aerobic training period and one month after the 72 sessions (suspension of training). We demonstrate that aerobic training at the intensity of VT1 promoted a reduction in the rate of periodic leg movement in relation to baseline values (53.5/h to 38.6/h). After the increase in intensity of aerobic training and a shift to the VT1 and VT2 range with predominance in VT2, the rate of periodic leg movement rose (63.8/h), and after one month of training suspension this rate was still higher than that of its corresponding moment baseline value (72.8/h). The AHI also increased during heightened AT when AT was performed at the intensity of VT1 27.8/h during AT compared to baseline 12.8. Such rates approached baseline values at VT1 and VT2 with predominance at VT2 (13.0/h) only to increase once again after one month suspension of training. AT at loads compatible with VT1 promoted enough improvement in the rate of periodic leg movement to down-grade the condition from severe to moderate, but sleep apnea syndrome increased at that intensity of exercise climbing from mild to moderate. Such results suggest that the benefits credited to AT have distinct mechanisms of action in periodic leg movement and sleep apnea syndrome

Keywords: Sleep; Leg/physiopathology; Restless legs syndrome; Sleep disorders; Electromyography; Polysomnography; Sleep apnea, obstructive; Exercise therapy

RESUMO

Há evidências de que o exercício físico regular promove uma variedade de adaptações e benefícios fisiológicos. O exercício físico pode ser considerado uma intervenção não-farmacológica para a melhora da qualidade do sono em pessoas que apresentam algum distúrbio do sono. No entanto, poucos são os estudos que avaliam os benefícios decorrentes do exercício físico para essa população específica. O presente estudo teve por objetivo avaliar a influência de um programa de treinamento físico aeróbio (TFA) realizado em diferentes intensidades em um voluntário que apresentou, no exame polissonográfico, movimento periódico das pernas (MPP) e síndrome da apneia obstrutiva do sono (SAOS). O voluntário, com 51 anos, 91 kg, 178 cm, índice de massa corpórea (IMC) de 28,67, sedentário, apresentando MPP (>5/hora) e SAOS (índice >5 hora) foi submetido a um programa de TFA (esteira) pela manhã 3 vezes por semana, com duração de 40 minutos cada sessão, totalizando 72 sessões. Inicialmente, foi realizado um teste de esforço máximo (TEM) para a prescrição do treinamento físico aeróbio A primeira sessão de trei-namento físico aeróbio foi realizada a 60% do VO-2pico para análise do efeito agudo do exercício físico, as sessões 2 a 24 foram realizadas no limiar ventilatório 1 (LV1), as sessões 25 a 48 foram intervaladas entre o LV1 e LV2, e as sessões 49 a

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72 foram intervaladas entre o LV1 e LV2 com predominância no LV2. Foram realizados exames polissonográficos e TEM durante os 6 meses do treinamento físico aeróbio e após 1 mês do término das 72 sessões de TFA (destreino). Foi demonstrado que o treinamento físico aeróbio realizado na intensidade do LV1 promoveu uma redução no índice de movimento periódico das pernas em relação aos valores basais (53.5/h a 38.6/h). Após o aumento da intensidade do treinamento físico aeróbio para intervalado entre o LV1 e LV2 com predominância em LV2, o índice do movimento periódico das pernas se elevou (63.8/h), visto que após um mês de destreino esse índice se apresentou mais alto dos que o encontrado no momento basal (72.8/h). O índice da síndrome da apneia obstrutiva do sono apresentou aumento durante o treinamento físico aeróbio realizado na intensidade do LV1 em relação ao seu valor basal (12.8/h a 17.7/h), retornando próximo aos valores basais na intensidade de LV1 e LV2 com predominância em LV2 (13.0/h) e aumentando novamente após um mês de destreino (16.0/h). O exercício físico aeróbio com carga equivalente ao LV1 promoveu uma melhora no índice do movimento periódico das pernas, demonstrando mudanças na classificação de grave para moderado. No entanto, síndrome da apneia obstrutiva do sono demonstrou um aumento no seu índice com essa intensidade de treinamento, passando de leve para moderado. Esses resultados sugerem que o mecanismo de ação do exercício físico para esses dois distúrbios do sono age de maneira distinta em relação aos benefícios propostos pelo trei¬namento físico aeróbio.

Descritores: Sono; Perna/fisiopatologia; Síndrome das pernas inquietas; Transtornos do sono; Eletromiografia; Polissonografia; apneia do sono tipo obstrutiva; Terapia por exercício

INTRODUCTION

It is widely assumed that many factors bear influence upon the architecture of sleep. Periodic leg movements disturbance (PLMD), the restless leg syndrome (RLS) and obstructive sleep apnea syndrome (OSAS) are sleep disturbances that interfere in sleep architecture. The first two, PLMD and RLS, are characterized as movement disorders related to sleep, and their most impacting consequence upon the quality of life of the patient is the reduction of the quality and efficiency of sleep, a condition that is accompanied by daytime somnolence. OSAS, in turn, is mainly characterized by repetitive events of either partial (hypopnea) or total (apnea) obstruction of the upper airway causing hypoxemia, and in prolonged cases, hypercapnia that is often reverted by arousals causing sleep fragmentation[(1)]. Because of its ubiquity, the approach taken to deal with OSAS should be that of a public health issue[(2)]. The association of these disturbances is comum. In a study conducted by Al-Alawi et al.[(3)], it was demonstrated that the combination of OSAS and PLM resulted in increased daytime somnolence, and that PLM associated with OSAS proved to be more prevalent than in PLM in relation to RLS.

There is evidence that regular exercise produces a number of physiological benefits. Adaptation to exercise may be observed in the young, adult and aging populations as well as in those who suffer from some diseases. Factors such as the level of physical fitness, time and design of exercise, genetic factors, age and gender may bear influence in the extensive repertoire of adaptation strategies the body deploys in order to have a better quality of life[(4)].

Systemized exercise is considered a non-pharmacological intervention that improves the quality of sleep and its disturbances[(5)]. Epidemiological studies have demonstrated that exercise causes improvement of the quality of sleep and reduces daytime somnolence[(6,7)]. There are few studies that have examined the influence of exercise upon sleep disturbances alone. Recently, Aukermam[(8)] carried out a study that involved a program of combined exercises (aerobic and resisted exercise) with the purpose of analyzing its effectiveness in symptoms of RLS. Twenty-three volunteers (11 belonging to the experimental group and 12 to the control group) were submitted to aerobic and resisted exercise, 3 times a week for 12 weeks. In order to analyze the RLS variable, the questionnaire International RLS Study Group Scale (IRLSSG)[(9)] was applied, which assesses the symptoms of gravity, frequency, and impact of RLS upon the quality of life. Results demonstrated that from the sixth week on, symptoms of RLS presented a statistical reduction in relation to the Control Group. There was no significant difference between the 6th and the 12th week. The authors concluded that the program of combined exercises was effective in the improvement of symptoms of RLS, but further studies are required to demonstrate how different modes of exercise influence each symptom.

Giebelhaus et al.[(10)] carried out a study on sedentary individuals with moderate and severe OSAS wherein an aerobic and resisted exercise program was deployed, under supervision, two hours a day, twice a week, for six months. No significant differences were encountered in cardiorespiratory parameters and body mass throughout training, but there was a significant reduction in the rate of apnea events per hour, from 32.8 to 23.6, which did not result in alteration of body mass or in sleep architecture.

However, the effect of exercise in patients who present a combination of two or more sleep disturbances, particularly of PLM and OSAS, was not found in the literature. Thus, which prompted us to examine the isolated case of a patient who had that combination (PLM and OSAS), the objective of the present study, was to evaluate the influence of an Aerobic Training (AT) program with variable intensity.

THE CASE

The study had the approval of the ethical board for research of the Hospital São Paulo of Universidade Federal de São Paulo (UNIFESP) (Protocol - 0948/05). Once the required clarifications were given and prior to the commencement of the protocol, the patient signed an informed consent form agreeing to partake in the study.

A white male, 51 years old, weighing 91 kg and measuring of 1.78 m in height who, for 8 years, has complained of sudden kicks during sleep that awaken him. The patient looked for a sleep specialist and was referred to polysomnography (PSG). The PSG revealed he had PLM and OSAS. To treat his sleep disturbances, the patient was offered the alternative of partaking in an aerobic exercise program for six months. Upon his engagement in the program, the patient was physically evaluated by means of electrocardiograms taken at rest and effort and by laboratory exams to determine whether or not he would be apt to participate in the study as well as to establish the dosages of iron (ferritin), so that its relation with PLM could be verified.

The experimental design

The patient took a 6-month aerobic training program wherein exercise was performed in the morning (7:00) 3 times a week, totaling 72 exercise sessions. After adapting to the exercise apparatus by exercising every other day for 3 days on the treadmill (Life Fitness® 9700 HR), the patient underwent a maximum effort test (MET) so that the adequate prescription of exercise intensity could be established. On the first session of exercise, at 7:00 a.m., a PSG was taken in order to assess the acute effect of exercise. The patient then took to his daily affairs as he normally would only to return to the lab again at 9:00 p.m. for PSG arrangements. To assess the effects of chronic exercise, PSG was taken on the day subsequent to the sessions two and six months into the training program. After six months, another MET was taken at 60% of maximum oxygen consumption, and on that same day a PSG recording was collected so that the acute effect of the training could be appreciated. After the last training day, the volunteer took a PSG and remained a month without doing any kind of exercise. A last PSG was taken at the end of the idle month to collect data on the period relative to loss of fitness.

The experimental protocol

Polysomnographic exam

The sleep parameters were recorded by means of PSG using the device EMBLA S7000® at epochs with 30-second intervals classified as awake, stages 1, 2, 3 and 4 of NREM and REM, according to the standardized protocol by Rechtschaffen and Kales[11].

The electroencephalographic recording was obtained by means of fixated electrodes according to the directives of the International System 10-20[12]. Four electroencephalographic derivations were used, two electrooculogram channels, two electromiogram channels (submentonian and legs) and an ECG derivation. Termistor and nasal canula were used to monitor airflow, and belts to monitor thoracic and abdominal effort, transcutaneous oximetrics to record oxygen saturation, and a sensor to verify the position of the trunk during sleep.

The recording of muscular tonus by electromiography was done by placing two electrodes in the mentonian/submentonian regions. These were placed on both legs over the anterior tibial muscle. PLM was gauged visually scored by a trained sleep in accordance with the criteria established by the ASDA Task Force[13].

The maximum effort test (MET)

Aerobic exercise and the maximum effort test (MET) were performed on a treadmill (Life Fitness® 9700 HR). Initial speed was set at 4 km/h, increasing 1 km/h every minute until the volunteer reached either exhaustion or maximum heart rate. The surface of the treadmill was set at an inclination of 1% to simulate open terrain[14]. VO_{2peak} was obtained by means of analysis of ventilation alterations during the MET after incremental loads. Recording of the data was done using the metabolic analyzer Quark PFT Ergo 4 (Cosmed®).

Aerobic exercise was done at ventilation thresholds VT1 and VT2. The term VT refers to the point in which pulmonary ventilation increases out of proportion in relation to oxygen consumption during exercise with incremental loads[15]. It is assumed that H+ ions from lactic acid are tamponated by blood bicarbonate, thereby producing excess of CO_2 which, in part, increases ventilation (VE). The theory holds that the initial concentration of lactic acid coincides with the hyperventilation induced by exercise when it is done with incremental loads[16]. At VT1 (moderate exercise), exercise with load increment is related to the moment in which there is an equivalent increase of oxygen ventilation (VE/VO_2) as well as oxygen pressure ($PETO_2$), but without an equivalent change in CO_2 ventilation (VE/VCO_2) or in CO_2 pressure ($PETCO_2$). As intensity of exercise exceeds this threshold, metabolic acidosis causes a plunge in pH followed by an increase of VE/VCO_2 and reduction of $PETCO_2$, reaching the "point of respiratory compensation" of metabolic acidosis or VT2 (intense exercise)[17].

The use of VT in the determination of the capacity to execute prolonged exercise and limitation of performance has grown in the past years as it is an objective parameter collected in a noninvasive manner[18], and one that can be used as a variable or parameter for the prescription of exercise.

Training prescription

The 72 training sessions were distributed into 4 periods, 40 minutes each:

- period I (3 sessions): volunteers adapted to the exercise and equipment;
- period II (11st – 24th session): training at VT1 intensity, the 1st session being at 60% of VO2peak for 30 minutes for the determination of acute effects of exercise;
- period III (25th - 48th session): training at intensities between VT1 and VT2;
- period IV (49th - 72nd session): training with emphasis on VT2 intensity and recovery at VT1.

All tests were carried out within the laboratory at controlled temperature (22°C and 24°C) and relative air humidity between 40 and 60%.

Table 1: Results obtained in the assessments of cardiorespiratory capacity along the six months of exercise

Assessment of the cardiorespiratory capacity	Basal	6 Months	After idle period
VO_2 max (L/min)	3.3	3.72	3.23
VO_2 max (mL/kg/min)	38.07	42.83	36.76
VE max (L/min)	121.6	131.7	111.1
HR max (bpm)	162	168	176
HR prev (bpm)	170	169	169
Oxygen consumption (VT1) l/min	1.72	2.34	1.94
Oxygen consumption (VT2) mL/kg/min	19.36	26.3	22.1
HR (VT1)	120	128	141
Speed (VT1) Km/h	7	9	8
Oxygen consumption (VT2) L/min	2.39	3.03	2.47
Oxygen consumption (VT2) mL/kg/min	26.36	34.05	28.12
HR (VT2)	148	151	157
Speed (VT2) km/h	9	11	9

Descriptive data of the sample.
VO_2: oxygen consumption; VE: ventilation maximum; HR: heart rate; VT: ventilation thresholds.

RESULTS

Analyses of blood iron (170 ug/dl) and ferritin (411,1ng/ml) were within normality for the population.

Table 1 depicts the values of the ergospirometical variables that were found after the MET. Results that refer to the sleep pattern after exercise are depicted in Table 2. It was observed that PLM decreased in the two-month training period and returned to values classified as severe after six months' training. The classification of OSAS, in turn, shifted to moderate after the first two months of training only to resume baseline values six months after training.

DISCUSSION

After a six-month, three-time-a-week aerobic training program, it was demonstrated that a patient who had PLM and OSAS presented different responses in the rate of these disturbances in relation to the intensity of exercise.

When the exercise was moderate, the rate of PLM reduced after two month's exercise, but resumed baseline values after six months of intense exercise. A partially inverted scenario occurred with OSAS, which is to say that AHI increased in the two months of moderate exercise (VT1) and resumed baseline values after six months of intense (VT2) exercise.

PSG recordings taken on the day of acute exercise and at the end of the day of aerobic exercise (for chronic effects) did not reveal significant differences in the variables of the sleep pattern. Physical exercise itself does influence

Table 2: Results obtained from polysomnographic exams along the six-month exercise period

Polysomnographic data	Basal	Acute	2 Months	6 Months	Acute	After idle period
Total sleep time (minutes)	427.5	368	374.5	351.5	398.5	419
Sleep efficiency (%)	91.3	89.9	80.2	89.5	92.4	87.2
Sleep latency (minutes)	6.4	0.6	3.2	4.7	7.3	4.2
REM sleep latency (minutes)	87	56	73.5	71	49	70
Time awake after sleep onset (minutes)	34.5	40.7	89.1	36.8	25.5	57.1
Stage 1 (%)	3.9	4.2	4.1	2.7	3.1	4.5
Stage 2 (%)	56.3	54.1	56.7	60.3	55	62.3
Slow wave sleep (%)	16.7	17.6	16.7	12.3	14	9.8
REM (%)	23.2	24	22.4	24.8	28	23.4
Microarousals rate (/h)	19.7	20.3	31.6	36.6	27.3	29.3
PLM Rate (/h)	53.5	68.9	38.6	63.8	81.5	72.8
Apnea-Hypopnea Index (h)	12.8	11.1	27.8	16	13	16

Descriptive data of the sample.
REM: rapid eye movement; PLM: periodical leg movement.

the sleep architecture, as Hague et al.[19] verified that systemized training promotes an increase in slow wave sleep, in total sleep time, in stage 2 of NREM sleep, and also in latency for the onset of REM sleep, and may still reduce the total sleep time of REM and latency for the onset of sleep in athletes. This corroborates the view held by most researchers examining the influence of exercise upon sleep who promptly verify that exercise causes an increase in slow wave sleep and increase of latency for REM sleep, as well as a reduction of REM sleep[20,21]. Notwithstanding, the current investigation did not verify any marked alteration in the sleep stages of the volunteer, only a reduction of latency for REM sleep, but no reduction of slow wave sleep in the first half of the night and/or increase of REM sleep when PSG was recorded on the exercise day to assess acute and chronic effects of the training. It should be said at this point that there is evidence that the volume of exercise should be higher before significant sleep alterations can be expected[22,23]. In the current study, the intensity and duration of exercise show that moderate exercise reduced the intensity of PLM but increased the number of microarousals and AHI enough to re-classify it from mild to moderate. Increased intensity exercise led rates of PLM to exceed baseline values while those of AHI reduced approaching baseline values.

According to Rodrigues et al.[24], the association of OSAS and RLS are responsible for complaints of daytime somnolence. This investigation attempted to verify whether fatigue symptoms and excessive daytime somnolence prior to and after Continuous Positive Airway Pressure (CPAP) treatment in patients with OSAS and RLS. At the beginning of the study, no significant differences were found between groups, but after the CPAP treatment those patients who presented an association with disturbances presented higher scores in the Epworth Sleepiness Scale and in the Pichot fatigue/depression questionnaire. This leads to the conclusion that CPAP was more effective in the treatment of OSA, the symptoms remained more due to PLM.

Norman et al.[25] carried out a study about the effects of exercise over OSAS in patients who had moderate to mild cases and who were requested to participate in an aerobic training program that would last six months. The following pre and post-training exams were: PSG, treadmill effort test, and anthropometric measurements. In addition, a questionnaire was given on quality of life. At the 4th month, a new effort test was done on the treadmill in order to adjust the training load if necessary. The results showed that there was a significant reduction in the AHI, with improved total sleep time, sleep efficiency, number of arousals per hour, and body mass. Thus, it can be inferred that physical training had a positive impact upon not only OSAS but also the conditioning of aerobic capacity as well as quality of life. This comes to show that exercise can indeed be adopted as a non-pharmacological means of dealing with OSAS.

Herein, the volunteer presented improvement in the cardiorespiratory capacity, reduced fat percentage with no alteration in body mass, and, even suffering of OSAS, there was good quality and efficiency of sleep, and no alteration in sleep architecture was verified.

De Mello et al.[26] observed, by means of PSG, that exercise in athletes who suffered medullar lesion significantly reduced PLM during sleep. One hypothesis for the relation between exercise and reduction of PLM is the release of β-endorphin that occurs with exercise. It is known that blood concentration of β-endorphin increases with exercise, depending on the intensity, duration and volume of training, and that the substance acts in very much the same way that the exogenous opioids that are used in the treatment of PLM[27].

Although there is no consensus in the literature as to the best type, time, intensity or duration of exercise, physical training is definitely recommended in the promotion of quality of life. And we may suggest that the results collected herein reflect mechanisms distinctly dependent upon either PLM or OSAS, which means that exercise presents diverse effects on each of these two disturbances that are verifiable in the relevant association between the modulation of PLM and OSAS symptoms and intensity and volume of exercise.

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