

Brazilian consensus on sleep physiotherapy

Consenso brasileiro de fisioterapia do sono

Susana Cristina Lerosa Telles^{1,2}, Luciana Badke Neves de Paiva³, Talita Arrais⁴, Ingrid Hashimoto Tadeu Marzola⁵, Claudia Santana Zerbinatti⁵, Eduardo Cassoli Ferraz⁵, Cintia Arruda⁵, Denis Mesquita⁵, Fabiano Moreno⁵, Alessandra Seixinho⁵, Jairo Fiorato Junior⁵, Lara Freitas Assumpção⁵, Cecília Tader⁵, Sergio Tufik⁶, Aline Marques Franco⁷, Daniela Morales⁷, Alan Luiz Eckeli⁷, Octávio Marques Pontes-Neto⁷, Amélia Pasqual Marques⁷, Ana Assumpção⁷, Alida Kellermann Borba^{8,9,10}, Lia Rita Azeredo Bittencourt¹¹

ABSTRACT

The Brazilian physiotherapists are working in Sleep field in Brazil, both in clinical practice and research. There are only a few professionals in light of the huge demand of sleep disorder's patients. This text aims to gather evidence and propose protocols to produce guidelines of clinical practice in the performance of physiotherapists in Brazil. It addresses Obstructive Sleep Apnea Syndrome, Neuromuscular Disorders, Restless Legs Syndrome, Periodic Leg Movements, Fibromyalgia and Sleep Bruxism.

Keywords: fibromyalgia, neuromuscular diseases, nocturnal myoclonus syndrome, obstructive, physical therapy specialty, sleep apnea.

RESUMO

Os fisioterapeutas brasileiros estão atuando na área de sono no Brasil, tanto na prática clínica quanto na pesquisa. Ainda são poucos em face da grande demanda de pacientes com distúrbios do sono. Este texto tem por objetivo reunir evidências e propor protocolos para servir como diretriz de prática clínica na atuação de fisioterapeutas no Brasil. Discutem-se Síndrome da Apneia Obstrutiva do Sono, Distúrbios Neuromusculares, Síndrome das Pernas Inquietas, Movimentos Periódicos dos Membros, Fibromialgia e Bruxismo do sono.

Descritores: doenças neuromusculares, fibromialgia, fisioterapia, mioclonia noturna, síndrome da apneia obstrutiva do sono.

INTRODUCTION

Physiotherapists already work on sleep disturbances field in Brazil, mainly focus on CPAP adherence. However, the professional does not find the proper clinical orientations to guide his practice. This text aims to propose guidelines for physiotherapy in sleep disorders based on scientific evidences. This work is the result of the first partnership between several reference centers, universities, physiotherapists and other sleep disorders in Brazil, organized by the Physiotherapy Commission of the Brazilian Sleep Association.

The text is divided in sections regarding physiotherapist's performance in sleep disorder's afflicted patients such as: respiratory, neurological, fibromyalgia and bruxism during sleep. For each case, there is a brief summary and discussion on how the physiotherapist can help.

Respiratory: Physiotherapy role on the obstructive sleep apnea syndrome (OSAS)

Authors: Luciana Badke Neves de Paiva; Talita Arrais; Sergio Tufik; Lia Rita Azeredo Bittencourt

Study carried out at Presidente da Comissão de Fisioterapia da Associação Brasileira de Sono.

¹ Presidente da Comissão de Fisioterapia da Associação Brasileira de Sono.

² Hospital do Servidor Estadual - São Paulo, Brazil.

³ Universidade Federal de São Paulo - São Paulo, Brazil.

⁴ Laboratório Fleury - São Paulo, Brazil.

⁵ Setor de Tratamento de Doenças Neuromusculares - TDN/AFIP - São Paulo, Brazil.

⁶ Departamento de Psicobiologia - Universidade Federal de São Paulo - São Paulo, Brazil.

⁷ Universidade de São Paulo - Ribeirão Preto, Brazil.

⁸ Hospital Santa Catarina - Blumenau, Brazil.

⁹ Clínica Dr. Martins de Cirurgia Maxilo Facial - Blumenau, Brazil.

¹⁰ Sonomax - Blumenau, Brazil.

¹¹ Universidade Federal de São Paulo - UNIFESP

Corresponding author: Susana Cristina Lerosa Telles. Hospital do Servidor Estadual de São Paulo. Rua Pedro de Toledo, nº 1800, Bloco F DAR 3º andar. Moema. São Paulo - SP, Brazil. CEP: 04039-000. E-mail: contato@reabilitarsono.com.br

Received: December 18, 2012; Accepted: June 28, 2013.

Definition:

The Obstructive Sleep Apnea Syndrome (OSAS) is characterized by recurrent events of obstruction of the upper airway (SAT) during sleep, involving respiratory-effort related arousals (RERA), limitation, reduction (hypopnea) or complete air flow cessation (apnea) while respiratory movements persist. In general, ventilation interruption results in oxyhemoglobin desaturation and hypercapnia in prolonged events. Often those events are ended by microarousals that fragment sleep^(1,2).

Epidemiology:

Epidemiological studies have reported OSAS prevalence in adults from 2 to 7.5%, considering the presence of excessive diurnal sleepiness and apnea and hypopnea indexes (AHI) above 5⁽³⁻⁷⁾.

A recent epidemiological study conducted in the city of São Paulo reported 32.9% OSAS prevalence according to the International Classification of Sleep Disorders as diagnostic criteria^(2,8).

Physiopathology:

Several theories have been proposed to explain OSAS physiopathology. OSAS patients present pharyngeal airway reduced in size and altered in shape, going from latero-lateral elliptical shape to a more prone to collapse shape⁽⁹⁾.

It is believed that the intra-thoracic negative pressure, generated by the respiratory effort, stimulates mechanoreceptors at the thoracic wall and in the SAT, leading to awakening and reopening the SAT, followed by sleep. This cycle repeats itself several times during the night and may be associated with the desaturation of oxyhemoglobin⁽⁹⁾.

OSAS predisposing factors are: obesity, mainly at the upper part of the body; lack of physical activity; male gender; age between 40 and 65 years; endocrinologic disorders such as hypothyroidism and acromegaly; craniofacial anomaly and family history^(2,10).

The main consequences of abnormal respiration during sleep include excessive daytime sleepiness, neurobehavioral dysfunctions, cardiovascular, metabolic and mood disorders and quality of life alterations⁽²⁾.

Diagnostic and classification of the Severity of OSAS:

OSAS is a syndrome characterized by signs, symptoms, and associated risk factors. Therefore, the diagnostic needs a proper follow up based on history and physical anamnesis, associated to complementary exams⁽¹¹⁾. Polysomnography (PSG) is considered the “gold standard” diagnostic method for sleep disorders^(12,13).

According to the American Academy of Sleep Medicine’s task force report⁽²⁾, the severity criteria of OSAS must be based in two distinctive components: severity of excessive sleepiness and the number of obstructive respiratory events, evaluated by a PSG. The severity level must be specified according with both components and must be based on the most severe of the components. The OSAS severity is then classified as follows:

- Mild OSAS: involuntary and unwanted sleep episodes occurring during activities that require little attention. The symptoms lead to a small social and occupational hazard. Presence of 5 to 15 obstructive episodes per hour of sleep.
- Moderate OSAS: involuntary and unwanted sleep episodes occurring during activities that require some attention. The symptoms lead to a moderate social and occupational hazard. Presence of 15 to 30 obstructive episodes per hour of sleep.
- Severe OSAS: involuntary and unwanted sleep episodes occurring during activities that require a lot of attention. The symptoms lead to a severe social and occupational hazard. Presence of more than 30 obstructive episodes per hour of sleep.

OSAS treatment:

OSAS therapy is performed according to symptoms and its comorbidities severity, as well as PSG alterations. Sleep hygiene and behavioral measures such as weight loss, physical activity, drug withdrawal (benzodiazepines, barbiturates and narcotics), alcohol avoidance at night and change of position during sleep (avoiding dorsal decubitus), must be always enforced⁽¹¹⁾.

Positive Air Pressure (PAP) treatment*Basic Concepts*

Several types of positive air pressure (PAP) equipments are commercially available: CPAP (“Continuous Positive Air Pressure”), BiPAP (“Bi-level Positive Airway Pressure”), automatic positive air pressure equipment and central apnea equipment (servoventilator).

The CPAP is considered the therapy of choice for the treatment of OSAS, especially in moderate and severe cases. However, in clinical practice, PAP may be used as well for mild apnea cases, resistance of the superior airway syndrome and snoring⁽¹⁴⁾.

CPAP is an equipment that generates and directs a continuous air flow by way of a flexible tube leading to a firmly attached mask over the patients face (nasal or facial), where the expiratory resistance is determined by the mask fenestration⁽¹¹⁾. The PAP is transmitted to the SAT, creating a pneumatic spint that projects the soft palate anteriorly (over the tongue’s base) leading to expansion and opening of the entire superior air tract.

The benefits of PAP’s use are immediate and result in the termination of apneas, hypopneas and snoring, nocturnal increase in oxihemoglobin saturation and decrease of awakenings related to respiratory events.

Physiotherapist Role in the Sleep Laboratory: practice in Brazil

The physiotherapist may work in night shift performing the polysomnography test or in diurnal shift performing the exam first interpretation that must be revised by a medical doctor specialized in sleep disorders.

The basal sleep study is performed to detect the most common sleep disturbances in the night shift. If the patient is diagnosed with OSAS and the use of PAP equipment is

indicated, he or she is directed to the sleep laboratory for a PSG to adjust the correct PAP pressure level (titration).

When the patient arrives at the laboratory and has never had any contact with CPAP, there are several approaches to explain and patient adaptation to the equipment:

Suggestion of a protocol:

1. Patient is informed about:
 - a. What is PAP;
 - b. Its mechanism;
 - c. The type of sensation he or she will feel when the equipment is turned on;
 - d. Keep a peaceful and nasal respiration.
2. At this moment, the best mask option is determined for:
 - a. Type: the first type of option is always the nasal, but when the patient has any difficulty breathing through its nose and having the need for an oral respiration, the facial mask is then selected;
 - b. Size.
3. Finally, the equipment is connected on the patient for about 20 minutes programmed in a minimum pressure of 4 cm of water for adaptation.

During titration, it must be noticed the variable sleep efficiency in order to evaluate the patient's comfort with the equipment, as well as the hypnogram visualization which illustrates the sleep stages distribution. REM (Rapid Eyes Movement) rebound sleep and/or sleep with slow waves is an important factor because obstructive events deprive the patient of these stages. The AHI is another important factor that must be observed during the test, informing the professional as to what type of CPAP and mask are more adequate for that particular patient.

Home Continuous Use of CPAP: physiotherapist role in the process of adaptation.

Scientific Evidences

The regular use of this equipment in severe patients decreases the complaint of excessive daytime sleepiness (EDS) and other symptoms associated with OSAS⁽¹⁵⁾. However, CPAP and BiPAP are only therapeutically effective when regularly used. The regularity of the pattern of CPAP use during the first days predicts its use in the long term⁽¹⁶⁾. Studies have shown that the patients that are educated in more detail by telephone, reading of related material, group meetings and home visits, demonstrated better therapy adherence when compared to patients that were not properly guided⁽¹⁴⁾. The physiotherapist should use this opportunity to explain PAP use in general and to stimulate adhesion showing the benefits of the long term treatment.

Practice in Brazil:

After the PSG to adjust the PAP and the medical prescription for the equipment, the continuous education program starts.

Suggestion of a Protocol to be performed by the physiotherapist:

According to the American Academy of Sleep Medicine's task force report⁽¹⁷⁾.

- I. First consultation:
 - a. Investigate:
 - Sleep Habits (agitated, insomnia e daily habits);
 - If the patient breaths well through the nose; if does or did any ENT (Ear, Nose and Throat) treatment; or if has undergone any nose, tonsils or snoring surgery;
 - Cognition Alterations (Slow thinking, forgetfulness);
 - Mood Disorders (irritability, depression and anxiety);
 - History of smoking and alcohol addiction;
 - Ongoing physical activity and diet regimen;
 - Other health problems.
 - b. Explain the disease, risk factors, types of available treatments for each instance, what to look for and consequences of the lack of treatment;
 - c. Elucidate the importance of weight loss and sleep hygiene (sleep positioning, alcohol addiction, medications and benefits of a physical activity program, etc.);
 - d. Mask adaptation and the CPAP equipment.
- II. First, third and sixth month of CPAP use:
 - a. Evaluate the treatment adherence;
 - b. Investigate possible causes of mask and equipment intolerance.
- III. After this period, the patient may return every 6 months in order to verify treatment adherence and possible causes of treatment intolerance.

Recent studies evaluating OSAS patients using CPAP suggest that non-adherence is an important problem. There is no consensus on CPAP adherence definition. A use of 4 h/night in 70% of all nights was established as a clinical reference of treatment adherence to CPAP⁽¹⁸⁾.

The physiotherapist must investigate the possible causes of intolerance to CPAP, such as severe nasal obstructions that may impair the PAP utilization, dryness of the ocular mucosa during CPAP, headache when awakening, insomnia with OSAS, mask intolerance, skin lesions, poor hygiene when using the CPAP masks, equipment noises, humidifier application, among others⁽¹⁹⁾.

Several types of positive pressure equipments are available, which can also come with humidifiers, pressure graduation system (ramp), apnea and hypopnea detection system, mask leakage detection system and usage monitors⁽¹⁴⁾. The physiotherapist must know how to adjust every parameter according to the patient's needs.

Moreover, there are different types of masks: nasal, intranasal and facial. The nasal mask is more commonly used. The intranasal and facial masks are used in cases of nasal mask intolerance. However, intranasal mask is not indicated for pressures above 12 cm H₂O. The facial mask is indicated for patients that can't keep their mouth closed during sleep, even when using the nasal mask⁽¹⁴⁾.

The BiPAP is an optional therapy indicated in cases where higher pressures are necessary, when the patient presents difficulty in exhale the air against a fixed pressure, presence of central hypoventilation, treatment of restrictive pulmonary diseases or syndromes of hypoventilation associated with diurnal hypercapnia⁽¹⁴⁾.

The adaptive servoventilation is a method of ventilation that can be adjusted in a standard configuration or with a variable inspiratory and expiratory pressure (to secure the patency of the SAT). The ventilator adjusts itself with the Cheyne-Stokes respiration, characteristic of the central apnea, giving dynamic respiratory adjust (respiration per respiration), promoting hyperventilation and hypocapnia⁽²⁰⁾.

It is important that the physiotherapist working in this area also educates the patient about the continuous care of use and maintenance of the positive pressure equipment in the long term, as well as about the disease and the benefits of the treatment.

Rehabilitation program:

Scientific Evidences:

Although the results of some studies demonstrated the benefits of the regular physical exercise as an adjunctive treatment for OSAS, there are few studies in the area. According to Hong & Dimsdale⁽²¹⁾ (2003) the consequences of OSAS, such as diurnal somnolence and fatigue, may be the main reason for the limitation of physical exercise in this population.

Physical exercises in a regular fashion are indicated to aid in weight loss, increase SAT's muscular tonicity and also contribute to decrease the inflammatory response in OSAS individuals. However, the type, intensity and duration of the exercises are not established⁽²²⁾.

Shneerson & Wright⁽²²⁾ (2001), in a review, recommend regular physical exercise not only for weight loss, but also to modify the sleep structure. Some studies have shown improvement in subjective sleepiness, quality of life, mood, cognition and in decreasing of the respiratory disorder indexes (AHI + RERA)⁽²³⁻²⁵⁾. In another study, Sengul et al.⁽²⁶⁾ (2009) have shown an improvement in AHI, quality of life and sleep architecture after an aerobic physical and breathing exercise in patients with OSAS. Recently, Ackel-D'Elia et al.⁽²⁷⁾ (2011) have shown that an aerobic physical program during 2 months (3 times a week, with an average duration of 1 hour and intensity of 85% of maximum VO₂, as measured by cardiopulmonary tests), associated with CPAP in patients with OSAS, were more efficient in decreasing stress, fatigue and sleepiness's levels, presenting more positive effects on physical fitness and general health perception, when compared to patients using only CPAP.

Barnes et al.⁽²⁸⁾ (2009) have associated aerobic exercise (3 times a week), strength training (5 times a week from the fifth to the sixteenth week) and low caloric diet during 4 months in 12 individuals with mild to severe OSAS. The power of the strength exercises was measured in 80% of the 1 repetition max (RM) test, consisting of 3 series of 8 to 12 repetitions. The aerobic exercises were performed in a combination of biking, walking

and light jogging during 40 minutes in an intensity of 80% of the VO₂ peak on the maximum effort test in the treadmill. The results showed a significant reduction of the body mass index (BMI), abdominal and cervical circumference. However there were no significant changes on OSAS severity.

Therefore, there is no consensus concerning the benefits upon the sleep of apneic patients who underwent the rehabilitation program⁽²⁶⁾. On the other side, the efficacy of the early physical exercises is undeniable, once they prevent the progression of the disease and decreases morbidity and mortality within that population⁽²⁹⁾.

Practice in Brazil:

The physiotherapist's job is to plan the rehabilitation program for the patient. This program must be based on anamnesis formed by clinical history, muscular strength test, range of motion measures, kinesiotherapeutic evaluation of the respiratory system and cardiovascular evaluation by ergometric test or previous cardiopulmonary evaluation, with proper medical follow up. Based on the multi-systemic repercussion of OSAS, the assessment of sleep habits must be considered as fundamental within the context of rehabilitation and must be included as an important part of the clinical history of each patient when consulting for physiotherapy⁽¹⁾.

The rehabilitation program for apneic patients has excellent cost/benefit ratio, being one of the most accessible modalities for the majority of these patients^(27,30). The physiotherapist may use the proper low cost tools and provide sessions in public areas or at home, a very common practice in other physiotherapeutic clinical interventions in Brazil. There are benefits not only for the apneic patients, but and as well for patients with disabilities or already suffering from other and more severe pathologies. Sleep hygiene may be covered by a sleeping diary where the patient states: sleeping time, awakening time, number of times that they got up during the night. By analyzing this diary, the difficulties in CPAP adaptation are more easily revealed. The physiotherapist should be able to differentiate every patient's difficulties and integrate these interventions within the rehabilitation program of the apneic patient.

Neurological

Neuromuscular disorders and sleep

Authors: Ingrid Hashimoto Tadeu Marzola; Claudia Santana Zerbinatti; Eduardo Cassoli Ferraz; Cintia Arruda; Denis Mesquita; Fabiano Moreno; Alessandra Seixinho; Jairo Fiorato Junior; Lara Freitas Assumpção; Cecília Tadei; Sergio Tufik

Definition

Neuromuscular disorders involve a vast group of pathologies of genetic or acquired origin whose symptoms may appear from the neonatal period until middle age. Usually they are classified according to the specific compromised neuromuscular structure⁽³¹⁾.

- Muscular Fiber: myopathies
 - Congenital Muscular Dystrophy (Several subtypes)
 - Progressive Muscular Dystrophy: Duchenne, Becker, Waist, Umero-scapular Face, Steinert Myotonic Dystrophy
 - Myopathies: congenital and metabolic (several subtypes)
 - Myositis of different types, mainly polydermatomyositis
- Motor Neuron:
 - Spinal Amyotrophy (types I, II, III)
 - Poliomyelitis and sequel of Poliomyelitis (Post-Poliomyelitis Syndrome)
 - Amyotrophic Lateral Sclerosis(ALS)
 - Progressive Bulbar Paralysis
 - Primary Lateral Sclerosis
- Roots and peripheral nerves:
 - Sensitive-motor Hereditary Polyneuropathies: Charcot-Marie-Tooth type I and Déjerine- Sottas (type III)
 - Guillain-Barré Syndrome
- Myoneural Junction:
 - Congenital Myasthenic Syndrome
 - Myasthenia gravis
 - Botulism

Generally, the neuromuscular diseases compromise the skeletal muscles, marked by progressive and generalized loss of muscle strength, besides the bulbar and respiratory involvement in some of these pathologies. Pulmonary complications and respiratory failure are the main cause of death in this population⁽³²⁾.

Epidemiology

In children, the most prevalent forms of neuromuscular diseases are the Muscular Dystrophies, highlighted by Duchenne Muscular Dystrophy (DMD). It is an X-linked hereditary disease affecting 1 of 3,500 boys born alive. The Spinal Amyotrophy (SA) is subdivided in 3 groups, being the SA type I the most severe and frequent, appearing in 1 of every 20,000 newborns⁽³³⁾.

Among adults, the most aggressive and devastating disease is the Amyotrophic Lateral Sclerosis (ALS). Of unknown etiology, it affects both genders around the fourth of fifth decades of life, in a proportion of one or two cases for 100,000 individuals. The disease is characterized by progressive degeneration of the motor neurons and may show a bulbar beginning with dysarthria, dysphonia and dysphagia or in appendicular form, causing muscular weakness of the superior and inferior limbs. Regardless of its origin, the patients progress with muscular atrophy, general weakness and severe respiratory problems, keeping intact, however, their cognition and sensitivity⁽³⁴⁾.

Among the motor and sensitivity neuropathies, the disease type Charcot-Marie-Tooth, shows a prevalence of 1 to 100,000 and can affect both genders, especially between the first and second decades of life. The symptoms are linked to the demyelization process and the speed reduction of nervous impulse transmission, which results in both, sensitivity and motor dysfunction⁽³⁵⁾.

Another disorder is the Myasthenia Gravis, which appears in 200 to 400 individuals of every 1,000,000 people. It

is an autoimmune neuromuscular disease leading to fluctuating muscle weakness and fatigue. Muscle weakness is caused by circulating antibodies that block acetylcholine receptors at the postsynaptic neuromuscular junction⁽³⁶⁾.

Physiopathology

As a result of inspiratory, expiratory and bulbar muscles impairment, associated to physical deformities, there appear respiratory complications, most of them, not perceived during rest and only detectable at a very detailed clinical evaluation, but that can be rapidly aggravated. Among these, there are^(37,38):

- Reduction of volume and pulmonary capacity
- Micro-atelectasis
- Nocturnal Hypoventilation
- Diurnal Hypoventilation
- Defective Cough (peak of cough flow < 270 l/min) and difficulty in eliminating pulmonary secretion
- Dysphagia and frequent swallowing difficulties
- Recurrent respiratory infections
- Ventilatory Failure and Death

Sleep-related respiratory disturbances in neuromuscular disorders are also frequent, having prevalence 10 times higher than in the general population, which contributes to elevate the morbidity and mortality indexes⁽³⁹⁾. They are subdivided in 2 main groups:

- Nocturnal Hypoventilation: other than the sleep-related physiological effects, the respiratory muscle weakness increases during this period, developing frequent hypoxemia episodes (SpO₂ < 88% for more than 5 minutes of the total time of sleep) and hypercapnia (P_{ET}CO₂ > 50 mmHg for a period > 25% of the total time of sleep). This occurs initially during the REM sleep and may progress to other sleep phases, as well as compromising the alertness state in more advanced stages of the disease^(39,40). Symptomatology related to hypoventilation includes: fatigue, dyspnea, morning headache, frequent awakenings, suffocation episodes, excessive diurnal sleepiness, mood alterations. Other signs and symptoms are: notorious weight loss, recurrent respiratory infections, inability to control superior airways, reduced concentration and memory capacity, intellectual loss, libido reduction and growth deficiency^(41,42).
- Syndrome of Obstructive Apnea of the Sleep (OSAS): characterized by the presence of recurrent snoring episodes associated with nightmares of the suffocating type, agitated and fragmented nocturnal sleep, diurnal somnolence, morning headaches, fatigue, concentration difficulty and irritability. Besides respiratory and pharynx's muscles weaknesses, there are other risk factors such as: superior air tract deformities, obesity, craniofacial and anatomical alterations (adenotonsillar hypertrophy, retrognathism, macroglossia) and ventilator control abnormalities^(41,42).

Role of the Physiotherapist

Scientific Evidences

According to the Consensus of the American Thoracic Society (2004), patients afflicted by neuromuscular disorders must undertake respiratory evaluation, at least, 2 times per year after losing walking capacity, presenting forced vital capacity of > 80% or being 12 years old or more⁽³³⁾.

The clinical evaluation should include:

- Pulmonary Functional Tests: forced vital capacity (FVC), forced expiratory volume at 1° Second (VEF1), expiratory flow peak. The test must be performed in a sitting and supine position to evaluate possible diaphragmatic impairment (loss of CVF value > 40%)⁽⁴⁰⁾.
- Measurement of maximum respiratory pressures: Maximum Inspiratory Pressure (Pimax), Respiratory Pressure (Pemax), Inspiratory Nasal Pressure (Sniff test)
- Measurement of cough flow peak
- Analysis of blood gases: non-invasive parameters of SpO₂ such as pulse oximetry and measurement of final exhaled CO₂ (P_{ET}C) or arterial gasometry
- Sleep pattern study through polysomnography and/or nocturnal oxymetry
- Presence of signs and symptoms indicating hypoventilation^(33,41,42).

Indications for the beginning of treatment with VMNI:

- Nocturnal VMNI: must be introduced during sleep respiratory disturbances such as OSAS (apnea index ≥ 5 and associated symptomatology), hypoventilation and clinical complaints already described. The process of adaptation to VMNI may occur electively during the sleep study, at ambulatory or home consultation or in emergency situations, during hospitalization for respiratory complications⁽⁴²⁻⁴⁴⁾. Equipment with two pressure levels may be used, or a conventional per volume ventilator, as well as several models of interfacing according to the respiratory pattern or patient adaptation (nasal, facial or intranasal mask).
- Diurnal VMNI: due to the progressive characteristic of the neuromuscular diseases, the hypo-ventilatory state may be aggravated even during incisive nocturnal ventilator support and progress to dyspnea, hypoxemia and diurnal hypercapnia (SpO₂ ≤ 92% and P_{ET}CO₂ > 50 mmHg), leading to a total dependency of the mechanical ventilator. Continuous ventilator support may be instituted in a non-invasive form, through the relay of masks and oral apparatus or invasively via tracheostomy to facilitate ventilator assistance and bronchial hygiene^(42,43).

Benefits of VMNI:

- Ventilatory improvement and balanced blood gases
- Improvement of the quality of sleep
- Reverse of symptomatology

- Increase of pulmonary volumes
- Minimizes respiratory muscle work
- Reduces the number of respiratory infections
- Reduces the number and time of hospitalizations
- Promotes longer and better quality of life^(43,44)

Contraindications of VMNI:

- Lowering of the conscious level, sleepiness, lethargy, agitation, confusion or patient refusal
- Hemodynamic instability with need of vasopressoric medication, shock, complex arrhythmias
- Facial trauma and fracture of the cranial base
- Deficient cough or swallowing difficulty
- Abdominal distension, nausea and vomiting
- Bleeding in high digestive tract^(43,45)

Other techniques and exercises are used to supplement the respiratory support in neuromuscular diseases, including:

- Maximum Insufflatory Capacity: technique of pulmonary expansion usually applied on the respiratory treatment of neuromuscular diseases, aiming optimization of the pulmonary flows and volumes, improvement of atelectasis areas, cough and pulmonary complacence. Consists in the use of ambu in sequential pulmonary insufflation until reaching total pulmonary capacity. In general, one must exercise daily, with a frequency of 3 times a day. It is indicated for patients with reduced forced vital capacity (FVC) to less than 50 to 60% of the normal value⁽³⁹⁾.
- Manually Assisted Cough: due to the impairment of the expiratory muscles, patients present inefficacious cough capacity, fatigue and recurrent pulmonary infections. To help eliminate pulmonary secretion, it is frequently used the maneuver of maximum insufflation, followed by thoracic and/or abdominal compression during the expiratory phase^(39,40).
- Equipment of cough mechanical assistance: portable machine known as In-Exsufflator (Cough-Assist - Respironics®), which mechanically assists the cough through deep pulmonary insufflations, followed by a rapid exsufflation stage, increasing the flow peak and removing the secretion. Regular pressure levels vary between + 30/40 cm H₂O and - 30/40 cm H₂O and attention must be drawn to the risk of pneumothorax^(39,40).

Contraindicated Therapies:

Considering the physiopathologic alterations involved in neuromuscular diseases, specific therapeutic measures are usually not indicated and its applications, when really necessary, demand cautious and clinical monitoring^(39,40):

- Oxygen-therapy: the isolated use of oxygen works on the carotid receptors reducing the respiratory muscles impulses, possibly leading to narcosis and death

- Respiratory enhancers of nonlinear or linear pressure (Respiron/Voldyne or Threshold): the not well indication of these equipments may lead to an excessive and unnecessary muscular overload, with an early respiratory fatigue installation.

Practice in Brazil

Brazilian Law (Portaria) nº 370, of July 4th, 2008, has amplified the group of neuromuscular diseases with free access to treatment with mechanical non-invasive ventilation (VMNI-Bipap). Nowadays in Brazil, the Sector for the Treatment of Neuromuscular Diseases (TDN) of the AFIP/Institute of Sleep, is the reference center of the São Paulo Estate, destined to the following up and support of ventilatory treatment.

The team is formed by neurological, pediatric, pneumologist doctors and physiotherapists, responsible for the initial clinical evaluation, VMNI indication and ambulatory and home following up in the long term.

Routine TDN consultation:

- Patients without ventilator support: clinical consultations with doctor and physiotherapist are done with a monthly, tri-monthly, semesterly or annually, according to the severity of the case and individual need. All consultations include: symptoms evaluation, measurements of vital signs and blood gases, pulmonary function tests, evaluation tests of respiratory and coughing muscular strength. The following up aims to an early detection of the need for VMNI and reduction of adverse reactions.
- Patients on VMNI: the ambulatory consultation usually takes place after the first month of VMNI initiation in order to evaluate the adaptation and adherence process, as well as the presence of possible adverse reactions. Later on, the consultations are undertaken at variable intervals varying from 1 to 3 months. Patients who reside in the city of São Paulo and nearby, also have monthly home visit of the physiotherapist. Patients from out of the state or out of reach of the center (country and maritime rim) are periodically followed up in our ambulatory location; and as frequently as necessary, there is a direct contact with the local team.

Indications of VMNI:

- Clinical criteria as the presence of signs and symptoms of hypoventilation and, at least, one of the following physiological criteria: C_{VF} < 50% of predicted value, P_{imax} < 60cm H₂O, nocturnal/diurnal hypoventilation or OSAS^(33,43).

Among the physiotherapist's functions, there are:

Initial and periodical overall evaluation:

- Detailed clinical history
- Physical examination (weight, height/breadth, pulmonary auscultation, presence of muscle/skeleton deformities)

- Signs and symptoms of hypoventilation
- Non-invasive measurements of blood gases
- Respiratory tests: spirometry (sitting and supine), measurement of maximum respiratory pressures (P_{imax}, P_{emax}, Sniff test), measurement of peak of expiratory flow and peak of cough flow

Explanation and orientation of the respiratory exercises with ambu: maximum insufflation capacity and assisted coughing

Analysis and interpretation of the polysomnography and other clinical/laboratorial exams

Adaptation and orientation of VMNI:

- Adjust and regulation of the ventilator parameters (average flow volume (8 to 10 ml/Kg)
- Selection and adaptation of interfaces (facial, nasal, intranasal, total face)
- Reading and Adhesion Data Interpretation
- Reduction of adverse reactions: rhinitis, nasal congestion and dryness, aerophagia, facial lesion, nausea
- Installation of warm humidification when necessary
- General guidelines to patients, family members and caretakers

Routine clinical ambulatory reevaluations

Clinical and ventilator evaluations and monthly home support

Respiratory disorders of sleep and cerebrovascular diseases

Authors: Aline Marques Franco; Daniela Morales; Alan Luiz Eckeli; Octávio Marques Pontes-Neto

Definition:

Cerebrovascular diseases are vascular disorders that affect the central nervous system, causing temporary or permanent damage by ischemia or bleeding to a part of the brain or spinal cord due to any pathological process.

Among cerebrovascular diseases, the encephalic vascular accident (EVA) is the most frequent and may be defined as a clinical syndrome of vascular origin, characterized by the rapid onset of signs and symptoms characterize of focal encephalic involvement. There are two fundamental types of EVA: the ischemic, caused by the focal blood flow rapid decrease to a determined brain area in about 80% of the cases; and the hemorrhagic type, responsible for the remaining 20% of the cases and caused by the spontaneous rupture (nontraumatic) of a blood vessel, with bleeding to the interior of the brain (intra-cerebral hemorrhage), to the ventricular system (intra-ventricular hemorrhage) and/or sub-arachnoidal space (sub-arachnoidal hemorrhage).

The OSAS is the most common respiratory sleep disorder in patients with EVA. Due to increasing scientific interest in the study of this association, the relationship between these two disease groups is progressively more notorious.

Epidemiology:

Cerebrovascular diseases are the second major cause of mortality in the entire world, being responsible for 9.7% of all deaths in 2004 and probably will reach 12.1% of the world mortality till 2030⁽⁴⁶⁾.

In Brazil, cerebrovascular diseases are the main cause of death, overtaking coronary diseases⁽⁴⁷⁾.

Several epidemiological studies indicate that the sleep obstructive apnea is an independent risk factor for cardiovascular and cerebrovascular diseases. Some studies have observed sleep apnea in up to 62% of the patients during the first night after an ischemic EVA and associated it to an early neurological deterioration⁽⁴⁸⁾. Besides, the excess of obstructive apneas during the acute phase was associated with the increased mortality in patients with ischemic EVA on the long term^(49,50).

Yaggi et al. have published in 2005 a prospective and longitudinal study demonstrating that severe apneic patients showed a risk 3.3 times bigger of EVA or death compared to the non-apneic patients, independently of age, sex, race, obesity, smoking history, alcoholism, hypertension, diabetes, hyperlipidemia or atrial fibrillation⁽⁵¹⁾.

Therefore, OSAS appears to be an independent risk factor for ischemic EVA. As far as the relationship between OSAS and EVA, there is a need for more studies to demonstrate that interaction.

Physiopathology:

The relationship between obstructive sleep apnea and EVA is probably a process involving several mechanisms.

The apnea episodes lead to an intermittent hypoxia, CO₂ retention, with alteration of the hemodynamic and autonomic responses during sleep. Besides, they generate an increase of sympathetic activity with consequent peripheral vasoconstriction and blood pressure elevation. This recurrent hypoxemic stress leads to the release of vasoactive peptides, trophic substances such as endothelin, which may be related to the genesis or to the worsening of chronic arterial hypertension. The hypoxemia leads to an increase of the systemic inflammatory responses raising levels of inflammatory adhesion molecules such as IL-6, TNF- α and C-reactive protein, and higher leukocyte activation. The hypoxemia and re-oxygenation episodes characteristic of OSAS are implicated in the generation of the oxidative stress mechanism and to a possible endothelial dysfunction. Additionally, it is believed that the catecholamine increase and sleep deprivation related to OSAS contribute to the development of insulin resistance and glucose intolerance, independently of obesity. OSAS has been associated with an increase of platelet activation, increase of fibrinogen levels and of other potential markers of a pre-

thrombotic state. Moreover the sudden oscillations of the intra-thoracic pressures during obstructive apnea episodes have been associated with a ventricular cardiac dysfunction, autonomic and hemodynamic instability and increase of intra-cardiac shunts with possible paradoxical embolism. These mechanisms may predispose to the occurrence of EVA in OSAS patients⁽⁵²⁾.

It is believed that OSAS is more often the cause and not the effect of EVA due to some factors:

- apnea is essentially obstructive and not central;
- the frequency of OSAS is not different from that of TIA (transient ischemic attack) and cerebral stroke;
- the frequency of OSAS is not different from that of localized brain stroke of the stem or cerebral hemisphere;

Role of the Physiotherapist:

Scientific Evidences:

The use of positive pressure in airways is the technique that has been more studied in patients with EVA and is physiotherapist's job the handling of the equipment, choosing and adjusting the masks, patient and caretaker's orientation, besides other techniques that may help improve the patient's adhesion to the treatment.

Although the treatment of obstructive apnea of sleep with positive pressure is well established, the technique is still not fully implemented for OSAS and EVA's patients, even though the treatment seems to be secure and present benefits for this population.

Recent studies bring evidences that the treatment of sleep apnea may reduce the risk of cerebral stroke and may prevent the early neurological deterioration in the acute phase of cerebral ischemic lesion, at which moment when the zone of critical collateral flow (penumbra) might be affected by episodes of desaturation and hypo-flow caused by apneas.

In the study of Sandberg e et al., patients with EVA and OSAS were treated with nasal CPAP during 4 weeks. The use of CPAP for a period longer than 4 nocturnal hours resulted in improvement of the depressive symptoms and a reduction of "delirium" during the rehabilitation phase⁽⁵³⁾.

Wessendorf et al. observed that the use of CPAP in patients with EVA and OSAS during the phase of rehabilitation, led to a subjective improvement of well-being and the normalization of nocturnal blood pressure within the first 10 days of treatment⁽⁵⁴⁾.

Martinez-Garcia et al., in a study with EVA and TIA, demonstrated that the patients selected to be treated with nasal CPAP showed significant reduction of the appearance of new vascular events (EVA and Acute Myocardial Infarction), when compared with the controls that did not follow the treatment, during a period of 18 months of treatment⁽⁵⁵⁾.

The main limitation for the treatment with CPAP equipment has been the adhesion to treatment, which can be stimulated by means of an intensive support and continuous education programs.

Palombini & Guilleminault⁽⁵⁶⁾ have shown that only 22% of treatment adherence during 8 weeks of treatment, with 34% of lack of signature on the agreement consent by the patients or caretakers, after been shown to the CPAP equipment.

Some studies evaluating the use of CPAP in those patients have attributed the poor treatment adherence to the severity of motor impairment, to spontaneous improvement of respiratory events, nocturnal disorientation, cognitive disorders, poor mask adjustment and absence of treatment belief.

Bassetti et al.⁽⁵⁰⁾ observed poor adherence to CPAP by EVA and OSAS's patients, after the patients release from the hospital, with only 30% of them using the equipment at home in a long term evaluation.

In 2011, Parra et al.⁽⁵⁷⁾ conducted the first controlled and randomized study with the objective of evaluate CPAP use during acute phase of EVA and OSAS; and the impact of the equipment use on these patients, followed up during two years. The study concluded that the early use of CPAP in patients victims of first ischemic episode and afflicted by mild or moderate OSAS, is associated to improvement of the neurological evaluating scales 1 month after the EVA on the CPAP group, when compared to the patients in the control group. The acceptance of nasal CPAP and treatment adherence were favorable and the mortality rate was smaller in the patients of the CPAP group.

A recent study by Svatikova et al.⁽⁵⁸⁾ suggests the use of alternative therapies due to poor adherence to CPAP. The study concludes that the positioning therapy to avoid the supine position, reduces the severity of the sleep apnea after an ischemic EVA.

Practical Considerations of Physiotherapeutic Role:

The use of PAP is of great importance in the management of patients with EVA or OSAS, however there exist some essential considerations:

- The patients generally have advanced age and possess some functional incapacitation due to the EVA. Considering that, they might have some difficulty in adaptation and use of the mask and equipment;
- Cognitive deficiencies and/or sensitive aphasia (or Wernicke's) may difficult the understanding and respect for the importance of the treatment with PAP and, consequently, difficult the patient's adherence to it;
- A great portion of patients require active caretaker's participation in order to achieve a good treatment adherence. Being so, it is essential that the physiotherapist develop a continuous education program not only for the patients, but as well as along with their caretakers;
- During the acute and sub-acute phase of EVA, the occurrence of central apneas may limit the use of PAP. A possible solution would be the use of equipments that were able to detect central apneas. However, up to now, the use of PAP during the

acute phase of EVA still needs more scientific evidences;

- The presence of facial paralysis may contribute to air leakages through the mouth, causing dryness of the nasal and pharyngeal mucosa, worsening the quality of sleep and the treatment adherence to PAP;
- One way to evaluate de improvement of PAP's adherence is a patient's referral of decreased diurnal sleepiness. In patients with EVA this evaluation may be limited. As a result, it is necessary to validate the improvement with specific scales of subjective evaluation of OSAS in patients with EVA.

The diagnostic and treatment of OSAS in patients with EVA may positively influence the functional performance, mortality and prevention of a new cerebrovascular event. Besides, according to literature, the treatment with PAP is safe for this type of patients. Thus, it is recommended that the clinical protocols of rehabilitation on EVA include the early investigation of OSAS and its treatment in these patients. The use of PAP during the early phase of EVA is under investigation and more robust scientific evidences for its indication must become available very soon.

Periodic limb movements and restless leg syndrome

Author: Susana Cristina Lerosa Telles

Definition and diagnostic:

The Periodic Limb Movements (PLM) disorder is characterized by repeated and stereotyped movements during sleep not derived from any other primary sleep disturbance. The movements comprise hallux extension, combined with ankle partial flexing and, sometimes of the hip as well. Similar movements may also occur on arms. The diagnostic of PLM is based on complete basal polysomnography, in addition to the clinical data⁽⁵⁹⁾.

The Restless Leg Syndrome (RLS) is a sensory-motor disorder frequently associated with PLM. It is characterized by an urge to move the legs in the evening or night. It worsens during rest and there is a partial or complete relief by moving the legs or walking. The relief is immediate though temporary. It may manifest in just one or both legs. It may be idiopathic or secondary to other conditions such as pregnancy, kidney disease, iron-deprived anemia and peripheral neuropathy⁽⁵⁹⁾.

The diagnostic of RLS is obtained only if the patient fulfill all the necessary criteria⁽⁶⁰⁾:

- I. Need for move the legs, usually associated with dysesthesia; patients may use the terms like itching, tingling, pain, discomfort, irritation, anxiety and others.
- II. Beginning or increase with rest: mainly at bed time, that characterizes the syndrome as a sleep disorder.
- III. Relief by movement: is immediate and dependent of continuous movement. It may need stretching or walking. In some cases, massage or rubbing of the member are resources that can alleviate the sensation.

- IV. Circadian pattern: it worsens at evening or beginning of the night, improves during late at night. It compromises the patient's sleep until the improvement of the symptoms.
- V. The occurrence of the abovementioned characteristics is not related to other medical or behavioral disorders (such as: myalgia, venous stasis, edema of the legs, arthritis, leg cramps, positional discomfort, tapping the foot on the floor by habit).

Other criteria that may support diagnosis:

- I. Family history
- II. Response to dopaminergic therapy
- III. Presence of PLM

Associated clinical characteristics:

- I. Natural evolution of the disease according to patterns: it is a chronic disease, which if started at young age tends to be insidious, and may present long periods of remission; or if starting late in life, may be sudden.
- II. Sleep disorder: reduces the sleeping time, but the sleep *per se* is not altered.
- III. Physical exam is normal: usually, the etiology is idiopathic. For this reason, one must be alert to conditions that may be related to a secondary RLS.

In children, the diagnostic follow other criteria:

- I. The child must fulfill all the essential adult criteria and
 - II. The child describes with its own words the leg discomfort
- OR
- I. The child must fulfill all the essential adult criteria and
 - II. Two of the three support criteria are present:
 - a. Sleep disorder for the age
 - b. Family history
 - c. Polysomnographic diagnosis of PLM

Epidemiology:

The prevalence of PLM in the population is 3.9%⁽⁶¹⁾. There are several genes related to the disorder transmission⁽⁶²⁾. Such genes mark the final proof of its real pathology, with clinical characteristics that correspond to a genetic transmission. The RLS has clinical diagnosis, therefore, the prevalence and incidence may vary according with the study criteria. If only the symptoms are considered, the prevalence varies from 9.4% to 15%. If the minimum diagnostic criteria from the International Restless Leg Syndrome Study Group⁽⁶⁰⁾, are considered, the prevalence varies from 3.9% to 14.3%. When factors like severity and frequency are taken in consideration, the prevalence varies from 2.2% to 7.9%. When performing the differential diagnostic procedure, the prevalence varies from 1.9% to 4.6%. The prevalence is higher in women and increases with age⁽⁶³⁾.

Physiopathology:

The physiopathology of these disorders has not yet been established. Both are related to other disorders, which are the sources of some physiopathology theories such as:

- a. Possible participation of generators of central pattern⁽⁶⁴⁾: related to the occurrence of these disorders in spinal cord injury patients.
- b. Iron deficiency⁽⁶²⁾: demonstrated in clinical studies and in patients that respond to the iron reposition treatment.
- c. Dopaminergic deregulation on the superior central nervous system⁽⁶²⁾: demonstrated in image studies and in patients that respond to anti-dopaminergic agents.

Role of the physiotherapist:

On PLM:

Scientific Evidences:

The PLM is a chronic disorder incurable until nowadays, but it is controllable by drug and nondrug therapies.

There are evidences that physical exercise decreases the symptoms of PLM, however, there is still no consensus about it⁽⁶¹⁾. No type of exercise has been defined as effective; however, the majority of protocols include treadmill bicycles and other exercises that vary according to the protocol. The physiotherapist who works in spinal cord injury rehabilitation must be alert because the patients can manifest PLM⁽⁶⁴⁾. There are evidences of improvement of the symptoms in these patients in protocols involving adapted treadmill bicycles⁽⁶¹⁾.

Practice in Brazil:

PLM has already been approached by programs including aerobic exercises (treadmill bicycles or treadmill) varying in intensity, duration, number of sessions and follow up with good results⁽⁶¹⁾. Lower limbs strengthening exercises have also been applied allied to aerobics, demonstrating good results⁽⁶¹⁾. The chronic character of these disorders reflects what happens in practice: the results are maintained only when the patient is adherent to the program. In these cases, the physiotherapist must:

- I. Perform a physiotherapeutic consultation, specific anamnesis for sleep disturbances with the use of Epworth's Sleepiness Scale, the Pittsburg Questionnaire on Quality of Sleep or evaluate the patient according to the International Functional Classification.
- II. Guide, favor and readapt the client/patient to the program of aerobic exercises.
- III. Determine the performance conditions for the aerobic program and, if necessary, recondition the client/patient through specific kinesiotherapy adequate for each case in order to prepare the patient for the aerobic program, establishing compensatory pauses and reassess the strategies for physiotherapeutic intervention.

In RLS

Scientific Evidences:

RLS is also a chronic and incurable disorder, although it shows some periods of spontaneous remission. Drugs and nondrug approaches are used in the control of RLS.

In terms of RLS intervention, the role of the physiotherapist almost does not show scientific evidences. In a single revision of a nondrug treatment, the transcutaneous electrical neural stimulation (TENS) was used showing positive results⁽⁶⁵⁾. In the clinical practice of idiopathic RLS, presenting strong circadian mode of nocturnal appearance of the symptoms, the TENS would work if the patient applied it at home, after orientation from the physiotherapist. Another attempt of a nondrug treatment was undertaken to evaluate the efficacy of monochromatic infrared light on the decrease of the RLS's symptoms. In a randomized study, International Restless Legs Syndrome Study Group⁽⁶⁰⁾ evaluations were performed before, during and after applications. Thirty four patients were submitted to 12 applications on the legs for 30 minutes. The results showed a continuous decrease in the symptoms of the treatment group when compared with the control group ($p < 0.001$). The improvement was kept during 4 weeks after the treatment, when compared to the basal results, however pointing a small trend to increase the symptoms after five weeks. The system used in this therapy was the Anodyne[®] with a wavelength of 890 nm, whose effect, theoretically, occurs due to the release of nitric acid and, consequently, a chain of biochemical reactions on the blood vessels which leads to a better local circulation⁽⁶⁶⁾. The same group compared two infrared wavelengths and frequencies. In this randomized experiment, 25 individuals were submitted to the same scheme of sessions of the previous study and were evaluated in the same fashion. The differences between the Anodyne[®] system and the one denominated Heat Light[®], are the use of an infrared light of 650 nm in Heat Light[®], and the frequencies of 292 Hz for the Anodyne[®] and 4698 Hz for the Heat Light[®]. Both equipments improved significantly the symptoms of RLS, with no significant difference between the two equipments⁽⁶⁷⁾.

The physiotherapist, who especially in amputee's rehabilitation, must be alert as to the possibility of RLS of the amputated lower limb. In a 2009 article, evaluating 283 amputees due to a peripheral vascular disease or trauma, Giummarra & Bradsham detected 13% afflicted by RLS⁽⁶⁸⁾. There is no consensus of physiotherapeutic approach of amputees afflicted by RLS. It is a recent discovery, but the authors suggested that it must have a different approach from that of the phantom pain.

Practice in Brazil:

Knowing that the RLS symptoms relief is achieved by the movement of the legs, each patient comes to the clinic already performing its own procedure: walking, stretching, massaging, rubbing, among others, all of them with the same characteristics: decisive in efficacy and transient in duration. Therefore, the patient searches for an efficacious as well as a more lasting treatment available.

The physiotherapist dealing with a RLS patient may follow the orientations of *International RLS Study Group*⁽⁶⁰⁾. In summary, it is recommended the nondrug therapy: physical exercises, changing the periods of inactivity to daytime, follow a good sleep hygiene program, since the sleep deprivation increases the symptoms considerably, and avoid a series of substances such as nicotine, caffeine, alcohol and drugs as antidepressants, antihistaminics, anti-nausea and antipsychotics.

The physiotherapist dealing with patients affected by peripheral neuropathy, may detect associated RLS. This professional should then approach both disorders based on specific physiotherapeutic protocols. In this case, it is important to educate and inform the patient that he/she has two distinct conditions, therefore, with two different treatment approaches concerning their differences in terms of sensitivity and motor manifestations.

More researches are still necessary to validate or not the efficacy of TENS or infrared light on the relief of PLM. The physiotherapeutic approach of PLM, whichever adopted, must bear in mind that it is a daily and chronic discomfort that can be controlled; therefore, it must be conducted aiming the long term duration that could be accomplished independently by the patient.

Fibromyalgia and physiotherapy

Authors: Amélia Pasqual Marques; Ana Assumpção

Definition

Fibromyalgia (FM) is a painful and non-articular syndrome of undetermined nature that is more frequent among women, being characterized by general and chronic musculoskeletal pain, specific painful sites at palpation - tender points - frequently associated to sleep disorders, fatigue, migraine and psychological and intestinal disorders, without significant laboratory alterations suggestive of the disease⁽⁶⁹⁾. FM generates great incapacitation, physically, as well as emotionally, such as depression and anxiety^(70,72) leading to a great impact in the quality of life^(70,73).

Epidemiology and prevalence

FM has been described as one of the most frequent rheumatologic disorder in the world population and especially in Spain⁽⁷⁴⁾, Bangladesh⁽⁷⁵⁾, Brazil⁽⁷⁶⁻⁷⁸⁾ and México⁽⁷⁹⁾. In terms of medical health assistance, it may represent 7% of all health complaints⁽⁸⁰⁾. The prevalence reported in literature ranges from 0.66% and 10.5%⁽⁸¹⁾ and presence in 2% of the adult population, increasing to 3.4% in women⁽⁸²⁾ and, among the middle age population around 5%^(77,79). Among the Brazilian population, it varies from 2.5% in the adult population, 4.4% in the middle age population and 5.5 % among the older population^(74,76,77).

Physiopathology

Researches indicate FM as a disorder of the processing of the sensorial stimuli by the central nervous system^(83,84) resulting in hypersensitivity, especially in pain modality, but also, hearing, olfactory, etc. The more consistent findings refer to pain-related

neurotransmitters, inhibitory as well as excitatory ones. The biogenic amines, serotonin and norepinephrine (pain inhibitors) are reduced in patients with FM, meanwhile the P substance (an excitatory pain neurotransmitter) appears to be in greater concentration in the cerebrospinal fluid^(85,86). These alterations could justify also the other symptoms of the disease such as sleep disorders, which have not yet been clarified. There are researchers who consider that the poor quality of sleep causes the related pain in FM and there are others who believe on the opposite idea, relating it to the Restless Leg Syndrome and Temporomandibular Disorder^(87,88).

Another alteration related to FM symptomatology is the hypothalamus-hypophysis-adrenal axis, as highlighted by increased basal levels of the Adrenocorticotrophic and follicle stimulating hormones, associated with the decrease of the insulin-like growth factor I (IGF-1), growth hormone (GH), estrogen, urinary cortisol, among others. These findings justify the association of FM and symptoms of chronic stress as well as the interference upon the quality of sleep and anxiety⁽⁸⁹⁾. On other hand, stressful events are also being described as predisposing and inciting FM factors. Among the agents which increase the incidence of FM are traumas (as whiplash injury), infections (as hepatitis C and B, HIV, Lyme's disease), emotional distress, catastrophic events (war), surgery, auto-immune diseases and others (such as rheumatoid arthritis, systemic lupus erythematosus, Sjögren's Syndrome)^(89,90).

Physiotherapy

According to the American College of Sport Medicine⁽⁹¹⁾, aerobic exercises must be performed between three and five times a week, with an intensity of 55% to 65% at 90% of the maximum heart rate, or between 40% and 50% at 90% of the consumption of reserve oxygen (VO₂) lasting 20 to 60 minutes. These exercises appear to aid in the improvement of the pain⁽⁹²⁾, well-being^(93,94), fitness⁽⁹⁵⁾, sensitivity of the *tender points*^(95,96) and other symptoms^(95,96). The aerobic exercise programs for the treatment of FM patients have been conducted in several ways, including a walking^(97,98), bicycling⁽⁹⁸⁾, and group games⁽⁹⁹⁾, with a duration of eight⁽⁹⁴⁾ to 24 weeks⁽⁹⁷⁾. Combination of aerobic exercises, stretching and muscular strengthening^(100,101) are also included on the evidences of the positive effects of guided exercises. Sometimes these programs are conducted in a warm swimming pool^(102,103) or in association to other therapeutic measures such as educational programs⁽¹⁰⁴⁾, relaxing^(100,105) and biofeedback⁽¹⁰⁶⁾.

Stretching exercises aim to increase the range of motion, holding a position of medium discomfort for about 30 seconds and with a frequency of two to three times a week⁽¹⁰⁷⁾, allowing the muscle to recover its functional strength, improving the movement range of motion and reestablishing degrees of freedom, better improvement of postural alignment and the integrity of its physiological functions^(107,108).

Despite very little scientific evidence, recent studies suggest positive effects of the flexibility exercises. Valencia et al.⁽¹⁰⁹⁾ compared the effect of kinesiotherapy with general mobility and stretching exercises through the global stretching technique

of Mezières. The authors observed positive results over pain, flexibility and overall FM symptoms in both groups after the treatment, which, however, are lost during the following up.

Considerations

Up to now, fibromyalgia has an unknown cause and manifests itself through pain and compromising the quality of life. It has a huge prevalence since it permeates among a very productive age range and generates great social-economic impact. Physiotherapy has an important role in its treatment and control of the symptoms. Therapeutic exercises are being postulated as one of the main tools in the management of FM with solid scientific evidences, including the sleep disturbances patients also present.

Physiotherapist role on the sleep bruxism

Authors: Alida Kellerman Borba; Susana Cristina Lerosa Telles

Definition

Sleep Bruxism is defined as a motor activity of grinding or clenching of teeth during sleep⁽¹¹⁰⁾. But, for the physiotherapist, it is important to be studied as kinesiological and biomechanical phenomena, within the temporomandibular joint disorder (TMD), since the evaluation, patient's symptoms and physiotherapeutic approach will be similar, despite the etiological differences between these two pathologies.

TMD is defined as a group of pathologies that affect the masticatory muscles, the temporomandibular joint (TMJ) and related structures. The most usual symptoms are: pain, audible TMJ click, limited mandibular range of motion, muscle and articular tenderness, besides other symptoms related to the head and cervical spine⁽¹¹¹⁾.

Epidemiology

Sleep bruxism appears to be a persistent chronic disease with evolution from its start at early age or during teen ages to its adult phase. It has a prevalence of 14% to 20% of children under 11 years old, thus its incidence is strongly connected to age, appearing around childhood after teeth eruption or at the beginning of teen ages, and reducing incidence after 40 years of age. The prevalence of sleep bruxism is about 13% between 18 and 29 years of age, decreasing to 3% among individuals with more than 60 years of age or more. Longitudinal studies revealed that 35% to 90% of children with sleep bruxism carry on with those symptoms in adult age⁽¹¹²⁾.

Only 5% to 20% of patients with sleep bruxism are aware of the episodes of grinding their teeth⁽¹¹²⁾, probably due to the episodes of facial and TMJ pain.

Etiology and physiopathology

Sleep bruxism has several classifications: primary and idiopathic, secondary or iatrogenic. The etiology is still not well established in primary or idiopathic cases, however, there are several theories: problems related to dental occlusion, personality style, ability to support daily pressures and neurochemical maintenance of sleep⁽¹¹⁰⁾. It might be secondary to neurological, movement,

or sleep disorders; or related to the use of narcotics or drugs such as amphetamines, anti-dopaminergics, antipsychotics, antidepressants and medications for cardiopathies⁽¹¹⁰⁾.

As overall concept, it is important to bear in mind that the morphological integrity of masticatory system depends upon the balance between physiological tolerance (represented by the emotional stress absorption capacity) and the structural tolerance⁽¹¹³⁾. In this point of view the sleep bruxism, regardless of its cause, has to be studied within the context of TMD, a multi-factorial pathology. Among its main causes are the para-functional habits, which may be described as tightening of the teeth, biting nails and chewing gum among others, and, occlusion, structural, sleep and emotional alterations and traumas. Para-functional habits are generated by the central nervous system stimuli of isometric contraction, sustained by the correspondent muscle group involved, which leads to the overload of the articulations where these muscles are attached, decreasing the inter-articular space and leading to the intensification of the degenerative state. The para-functional habits most commonly observed in TMD patients are tightening of the teeth, bruxism and bad posture⁽¹¹⁴⁾.

Besides the muscular disorders related to TMD, there are other internal disarrays of the TMJ which are defined as mechanical failures related to the inadequate positioning of the articular disk⁽¹¹⁵⁾. The most common articular manifestation is the dislocation of the articular disk, with or without reduction, characterized by the loss of the normal condilo-distal ratio. This situation may also happen due to degenerative diseases, inflammatory disorders, modifications in the disk morphology and defects in the articular surfaces⁽¹¹⁶⁾.

Role of the Physiotherapist on the treatment of sleep bruxism

Up to now, sleep bruxism has no available cure⁽¹¹⁰⁾. In its treatment, therapeutic exercises are placed as part of behavioral measures and may be associated to other therapeutic measures according to each case⁽¹¹⁰⁾.

Again, for the physiotherapist, sleep bruxism is integrated within TMD. The objective of TMD treatment is to control the pain, recover the masticatory system normal function, re-educate the patient and minimize the adverse loads that might perpetuate the problem⁽¹¹⁶⁾.

All studies that verify the influence of physiotherapy on TMDs, describe that it is inserted among those support therapies for the treatment of this disorder, aiming to abolish the signs and symptoms, keeping or recovering the functional activity within the least amount of time⁽¹¹⁶⁾.

In the study by Smékal et al., there was a significant reduction of the pain after the employment of physiotherapeutic techniques of relaxation and mobilization of the TMD's region, cervical column and its fascias. After the treatment protocol, 8.7% of the patients were totally free of the symptoms and 60.9% of them were very happy with the employed method of treatment⁽¹¹⁷⁾.

The physiotherapeutic exercises have positive effects over the pain and its sequelae on the chronic inactivity of the

musculo-skeletal system. The improvement of the symptoms and function is significantly better in the treated groups than in the control groups (with no treatment), observing no differences between the various forms of physiotherapeutic procedures⁽¹¹⁴⁾.

The results of a systematic review described by McNeely et al.⁽¹¹¹⁾, demonstrate that it is possible to use active and passive oral exercises to improve the posture and reduce the TMD's associated symptoms. There were no evidences to support the use of electro-physical modalities to reduce the pain; however, the results demonstrated improvement on mouth opening with the applied exercises.

Another systematic review by Medicott et al.⁽¹¹⁸⁾ analyzed the studies which evaluated the efficacy of some physiotherapeutic interventions in TMD, concluding that the active exercises, the manual joint mobilization, the postural training combined with other interventions, the laser therapy, the feedback programs (*biofeedback*), relaxing and proprioceptive reeducation may be more efficient than the placebo treatment or than the use of myorelaxant plates; and the combination of active exercises, manual therapy, postural correction and relaxing techniques, may be efficacious.

In general, the patient with sleep bruxism will search for the medical treatment due to the orofacial pain, pain in the TMJ or in the neck⁽¹¹⁰⁾. Therefore, the physiotherapy in the early stages of the dysfunction is the best treatment to prevent the installation of chronic pain. The treatment should not be limited to the ATM region, but should also take into consideration the relationship with other parts of the motor system and with the chain muscular interactions between the head and neck⁽¹¹⁷⁾. The option for the physiotherapeutic treatment may contribute to a better patient evolution, but its approach is still timid and requires more researches that can definitively prove its efficacy.

Acknowledgements

To the Brazilian Sleep Association, that make possible the union of physiotherapists, as well as to our professors, advisor and colleagues from other health professionals who guided us.

To Dr. Gustavo Antonio Moreira, Dr. Luis Fernando Grossklauss, Dr. Márcia Pradela Hallinan, Dr. Ricardo Tera Akamine, Dr. Sérgio Nacif. To Fabiana Cristina Ferreira and to the supporting staff from TDN. To the staff from DAR to the Medicina Física Department from Hospital do Servidor Estadual de São Paulo.

REFERENCES

1. Epstein LJ, Kristo D, Strollo PJ Jr, Friedman N, Malhotra A, Patil SP, et al.; Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med.* 2009;5(3):263-76.
2. American Academy of Sleep Medicine. International classification of sleep disorders, 2nd ed: Diagnostic and coding manual. Chicago: American Academy of Sleep Medicine; 2005.
3. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med.* 1993;328(17):1230-5. PMID: 8464434 DOI: <http://dx.doi.org/10.1056/NEJM199304293281704>

4. Bearpark H, Elliott L, Youn S, Kang K, Schneider H, Althaus W, et al. Snoring and sleep apnea. Snoring and sleep apnea. A population study in Australian men. *Am J Respir Crit Care Med.* 1995;151(5):1459-65. PMID: 7735600 DOI: <http://dx.doi.org/10.1164/ajrccm.151.5.7735600>
5. Bixler EO, Vgontzas AN, Lin HM, Ten Have T, Rein J, Vela-Bueno A, et al. Prevalence of sleep-disordered breathing in women: effects of gender. *Am J Respir Crit Care Med.* 2001;163(3 Pt 1):608-13. PMID: 11254512 DOI: <http://dx.doi.org/10.1164/ajrccm.163.3.9911064>
6. Ip MS, Lam B, Lauder IJ, Tsang KW, Chung KF, Mok YW, et al. A community study of sleep-disordered breathing in middle-aged Chinese men in Hong Kong. *Chest.* 2001;119(1):62-9. DOI: <http://dx.doi.org/10.1378/chest.119.1.62>
7. Kim J, In K, Kim J, You S, Kang K, Shim J, et al. Prevalence of sleep-disordered breathing in middle-aged Korean men and women. *Am J Respir Crit Care Med.* 2004;170(10):1108-13. PMID: 15347562 DOI: <http://dx.doi.org/10.1164/rccm.200404-519OC>
8. Tufik S, Santos-Silva R, Taddei JA, Bittencourt LR. Obstructive sleep apnea syndrome in the Sao Paulo Epidemiologic Sleep Study. *Sleep Med.* 2010;11(5):441-6. DOI: <http://dx.doi.org/10.1016/j.sleep.2009.10.005>
9. Bittencourt LRA, Palombini LO. Síndrome da apnéia e hipopnéia obstrutiva do sono: fisiopatologia. In: Tufik S. (Ed). *Medicina e Biologia do Sono*. 1a ed. São Paulo: Manole; 2008. p.240-7.
10. Norman JF, Von Essen SG, Fuchs RH, McElligott M. Exercise training effect on obstructive sleep apnea syndrome. *Sleep Res Online.* 2000;3(3):121-9.
11. Bittencourt LRA. Diagnóstico e tratamento da síndrome da apnéia obstrutiva do sono: guia prático. São Paulo: Livraria Médica Paulista; 2008.
12. Chesson AL Jr, Ferber RA, Fry JM, Grigg-Damberger M, Hartse KM, Hurwitz TD, et al. The indications for polysomnography and related procedures. *Sleep.* 1997;20(6):423-87.
13. Kushida CA, Littner MR, Morgenthaler T, Alessi CA, Bailey D, Coleman J Jr, et al. Practice parameters for the indications for polysomnography and related procedures: an update for 2005. *Sleep.* 2005;28(4):499-521.
14. Kushida CA, Littner MR, Hirshkowitz M, Morgenthaler TI, Alessi CA, Bailey D, et al. Practice parameters for the use of continuous and bilevel positive airway pressure devices to treat adult patients with sleep-related breathing disorders. *Sleep.* 2006;29(3):375-80. PMID: 16553024
15. McDaid C, Durée KH, Griffin SC, Weatherly HL, Stradling JR, Davies RJ, et al. A systematic review of continuous positive airway pressure for obstructive sleep apnoea-hypopnoea syndrome. *Sleep Med Rev.* 2009;13(6):427-36. DOI: <http://dx.doi.org/10.1016/j.smrv.2009.02.004>
16. Budhiraja R, Parthasarathy S, Drake CL, Roth T, Sharief I, Budhiraja P, et al. Early CPAP use identifies subsequent adherence to CPAP therapy. *Sleep.* 2007;30(3):320-4.
17. Epstein LJ, Kristo D, Strollo PJ Jr, Friedman N, Malhotra A, Patil SP; Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med.* 2009;5(3):263-76.
18. Weaver TE, Sawyer AM. Adherence to continuous positive airway pressure treatment for obstructive sleep apnoea: implications for future interventions. *Indian J Med Res.* 2010;131:245-58. PMID: 20308750
19. Rueda AD, Santos-Silva R, Togeiro SM, Tufik S, Bittencourt LRA. Improving CPAP compliance by a basic educational program with nurse support for obstructive sleep apnea syndrome patients. *Sleep Sci.* 2009;2(1):8-13.
20. Aurora RN, Chowdhuri S, Ramar K, Bista SR, Casey KR, Lamm CI, et al. The treatment of central sleep apnea syndromes in adults: practice parameters with an evidence-based literature review and meta-analyses. *Sleep.* 2012;35(1):17-40.
21. Hong S, Dimsdale JE. Physical activity and perception of energy and fatigue in obstructive sleep apnea. *Med Sci Sports Exerc.* 2003;35(7):1088-92. DOI: <http://dx.doi.org/10.1249/01.MSS.0000074566.94791.24>
22. Shneerson J, Wright J. Lifestyle modification for obstructive sleep apnoea. *Cochrane Database Syst Rev.* 2001;(1):CD002875.
23. Norman JF, Von Essen SG, Fuchs RH, McElligott M. Exercise training effect on obstructive sleep apnea syndrome. *Sleep Res Online.* 2000;3(3):121-9.
24. Netzer N, Lormes W, Giebelhaus V, Halle M, Keul J, Matthys H, et al. Physical training of patients with sleep apnea. *Pneumologie.* 1997;51 Suppl 3:779-82.
25. Giebelhaus V, Strohl KP, Lormes W, Lehmann M, Netzer N. Physical Exercise as an Adjunct Therapy in Sleep Apnea-An Open Trial. *Sleep Breath.* 2000;4(4):173-6.
26. Sengul YS, Ozalevli S, Oztura I, Itil O, Baklan B. The effect of exercise on obstructive sleep apnea: a randomized and controlled trial. *Sleep Breath.* 2011;15(1):49-56. DOI: <http://dx.doi.org/10.1007/s11325-009-0311-1>
27. Ackel-D'Elia C, da Silva AC, Silva RS, Truksinas E, Sousa BS, Tufik S, et al. Effects of exercise training associated with continuous positive airway pressure treatment in patients with obstructive sleep apnea syndrome. *Sleep Breath.* 2012;16(3):723-35. DOI: <http://dx.doi.org/10.1007/s11325-011-0567-0>
28. Barnes M, Goldsworthy UR, Cary BA, Hill CJ. A diet and exercise program to improve clinical outcomes in patients with obstructive sleep apnea—a feasibility study. *J Clin Sleep Med.* 2009;5(5):409-15.
29. Diabetes Prevention Program Research Group; Knowler WC, Fowler SE, Hamman RF, Christophi CA, Hoffman HJ, Brenneman AT, et al. 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. *Lancet.* 2009;374(9702):1677-86. DOI: [http://dx.doi.org/10.1016/S0140-6736\(09\)61457-4](http://dx.doi.org/10.1016/S0140-6736(09)61457-4)
30. Tuomilehto H, Gylling H, Peltonen M, Martikainen T, Sahlman J, Kokkarinen J, et al. Sustained improvement in mild obstructive sleep apnea after a diet- and physical activity-based lifestyle intervention: postinterventional follow-up. *Am J Clin Nutr.* 2010;92(4):688-96. PMID: 20702607 DOI: <http://dx.doi.org/10.3945/ajcn.2010.29485>
31. Reed UC. Doenças neuromusculares. *J Pediatr (Rio J).* 2002;78 Suppl 1:S89-S103. DOI: <http://dx.doi.org/10.1590/S0021-75572002000700012>
32. Gozal D. Pulmonary manifestations of neuromuscular disease with special reference to Duchenne muscular dystrophy and spinal muscular atrophy. *Pediatr Pulmonol.* 2000;29(2):141-50. DOI: [http://dx.doi.org/10.1002/\(SICI\)1099-0496\(200002\)29:2<141::AID-PPUL9>3.0.CO;2-Y](http://dx.doi.org/10.1002/(SICI)1099-0496(200002)29:2<141::AID-PPUL9>3.0.CO;2-Y)
33. Finder JD, Birnkrant D, Carl J, Farber HJ, Gozal D, Iannaccone ST, et al; American Thoracic Society. Respiratory care of the patient with Duchenne muscular dystrophy: ATS consensus statement. *Am J Respir Crit Care Med.* 2004;170(4):456-65. PMID: 15302625
34. Walling AD. Amyotrophic lateral sclerosis: Lou Gehrig's disease. *Am Fam Physician.* 1999;59(6):1489-96.
35. Aboussouan LS, Lewis RA, Shy ME. Disorders of pulmonary function, sleep, and the upper airway in Charcot-Marie-Tooth disease. *Lung.* 2007;185(1):1-7. PMID: 17294338 DOI: <http://dx.doi.org/10.1007/s00408-006-0053-9>
36. Conti-Fine BM, Milani M, Kaminski HJ. Myasthenia gravis: past, present, and future. *J Clin Invest.* 2006;116(11):2843-54. PMID: 17080188 DOI: <http://dx.doi.org/10.1172/JCI29894>
37. Perrin C, Unterborn JN, Ambrosio CD, Hill NS. Pulmonary complications of chronic neuromuscular diseases and their management. *Muscle Nerve.* 2004;29(1):5-27. PMID: 14694494 DOI: <http://dx.doi.org/10.1002/mus.10487>
38. Panitch HB. The pathophysiology of respiratory impairment in pediatric neuromuscular diseases. *Pediatrics.* 2009;123 Suppl 4:S215-8. DOI: <http://dx.doi.org/10.1542/peds.2008-2952C>
39. Otsuka MA, Boffa CFB, Vieira ABAM. Distúrbios musculares: Fisioterapia aplicada. 1 ed. São Paulo: Revinter; 2005.
40. Paschoal IA, Villalba WO, Pereira MC. Insuficiência respiratória nas doenças neuromusculares: diagnóstico e tratamento. *J Bras Pneumol.* 2007;33(1):81-92. DOI: <http://dx.doi.org/10.1590/S1806-37132007000100016>
41. Katz SL. Assessment of sleep-disordered breathing in pediatric neuromuscular diseases. *Pediatrics.* 2009;123 Suppl 4:S222-5. DOI: <http://dx.doi.org/10.1542/peds.2008-2952E>
42. Mellies U, Dohna-Schwake C, Stehling F, Voit T. Sleep disordered breathing in spinal muscular atrophy. *Neuromuscul Disord.* 2004;14(12):797-803. DOI: <http://dx.doi.org/10.1016/j.nmd.2004.09.004>
43. Benditt JO. Initiating noninvasive management of respiratory insufficiency in neuromuscular disease. *Pediatrics.* 2009;123 Suppl 4:S236-8. DOI: <http://dx.doi.org/10.1542/peds.2008-2952H>
44. Clinical indications for noninvasive positive pressure ventilation in chronic respiratory failure due to restrictive lung disease, COPD, and nocturnal hypoventilation—a consensus conference report. *Chest.* 1999;116(2):521-34. DOI: <http://dx.doi.org/10.1378/chest.116.2.521>
45. Robert D, Argaud L. Non-invasive positive ventilation in the treatment of sleep-related breathing disorders. *Sleep Med.* 2007;8(4):441-52. DOI: <http://dx.doi.org/10.1016/j.sleep.2007.03.009>
46. World Health Organization. *World Health Statistics 2008*. Geneva: WHO Press; 2008. p.29;36-31,55.
47. Lotufo PA, Goulart AC, Bensenor IM. Race, gender and stroke subtypes mortality in São Paulo, Brazil. *Arq Neuropsiquiatr.* 2007;65(3B):752-7. PMID: 17952275 DOI: <http://dx.doi.org/10.1590/S0004-282X2007000500004>

48. Pontes-Neto OM, Fernandes RM, Sander HH, da Silva LA, Mariano DC, Nobre F, et al. Obstructive sleep apnea is frequent in patients with hypertensive intracerebral hemorrhage and is related to perihematoma edema. *Cerebrovasc Dis*. 2010;29(1):36-42. DOI: <http://dx.doi.org/10.1159/000255972>
49. Kapen S, Goldberg J, Wynter J. The incidence and severity of obstructive sleep apnea in ischemic cerebrovascular disease. *Neurology*. 1991;41(suppl 1):125.
50. Bassetti CL, Milanova M, Gugger M. Sleep-disordered breathing and acute ischemic stroke: diagnosis, risk factors, treatment, evolution, and long-term clinical outcome. *Stroke*. 2006;37(4):967-72. PMID: 16543515 DOI: <http://dx.doi.org/10.1161/01.STR.0000208215.49243.c3>
51. Yaggi H, Mohsenin V. Obstructive sleep apnoea and stroke. *Lancet Neurol*. 2004;3(6):333-42. DOI: [http://dx.doi.org/10.1016/S1474-4422\(04\)00766-5](http://dx.doi.org/10.1016/S1474-4422(04)00766-5)
52. Somers VK, White DP, Amin R, Abraham WT, Costa F, Culebras A, et al. Sleep apnea and cardiovascular disease: an American Heart Association/American College of Cardiology Foundation Scientific Statement from the American Heart Association Council for High Blood Pressure Research Professional Education Committee, Council on Clinical Cardiology, Stroke Council, and Council on Cardiovascular Nursing. *J Am Coll Cardiol*. 2008;52(8):686-717. PMID: 18702977 DOI: <http://dx.doi.org/10.1016/j.jacc.2008.05.002>
53. Sandberg O, Franklin KA, Bucht G, Eriksson S, Gustafson Y. Nasal continuous positive airway pressure in stroke patients with sleep apnoea: a randomized treatment study. *Eur Respir J*. 2001;18(4):630-4. DOI: <http://dx.doi.org/10.1183/09031936.01.00070301>
54. Wessendorf TE, Wang YM, Thilmann AF, Sorgenfrei U, Konietzko N, Teschler H. Treatment of obstructive sleep apnoea with nasal continuous positive airway pressure in stroke. *Eur Respir J*. 2001;18(4):623-9. DOI: <http://dx.doi.org/10.1183/09031936.01.00057201>
55. Wessendorf TE, Wang YM, Thilmann AF, Sorgenfrei U, Konietzko N, Teschler H. Treatment of obstructive sleep apnoea with nasal continuous positive airway pressure in stroke. *Eur Respir J*. 2001;18(4):623-9. DOI: <http://dx.doi.org/10.1183/09031936.01.00057201>
56. Palombini L, Guilleminault C. Stroke and treatment with nasal CPAP. *Eur J Neurol*. 2006;13(2):198-200. DOI: <http://dx.doi.org/10.1111/j.1468-1331.2006.01169.x>
57. Parra O, Sánchez-Armengol A, Bonnin M, Arboix A, Campos-Rodríguez F, Pérez-Ronchel J, et al. Early treatment of obstructive apnoea and stroke outcome: a randomised controlled trial. *Eur Respir J*. 2011;37(5):1128-36. PMID: 20847081 DOI: <http://dx.doi.org/10.1183/09031936.00034410>
58. Svatikova A, Chervin RD, Wing JJ, Sanchez BN, Migda EM, Brown DL. Positional therapy in ischemic stroke patients with obstructive sleep apnea. *Sleep Med*. 2011;12(3):262-6. doi: 10.1016/j.sleep.2010.12.008. DOI: <http://dx.doi.org/10.1016/j.sleep.2010.12.008>
59. American Academy of Sleep Medicine. International classification of sleep disorders, revised: diagnostic and coding manual. 3rd ed. Chicago: American Academy of Sleep Medicine; 2001.
60. RLS Medical Bulletin: a Publication for Healthcare Providers. Rochester: RLS Foundation; 2005.
61. Pigeon WR, Yurcheshen M. Behavioral Sleep Medicine Interventions for Restless Legs Syndrome and Periodic Limb Movement Disorder. *Sleep Med Clin*. 2009;3(4):487-94. DOI: <http://dx.doi.org/10.1016/j.jsmc.2009.07.008>
62. Desseilles M, Dang-Vu T, Schabus M, Sterpenich V, Maquet P, Schwartz S. Neuroimaging insights into the pathophysiology of sleep disorders. *Sleep*. 2008;31(6):777-94.
63. Ohayon MM, O'Hara R, Vitiello MV. Epidemiology of restless legs syndrome: a synthesis of the literature. *Sleep Med Rev*. 2012;16(4):283-95. DOI: <http://dx.doi.org/10.1016/j.smrv.2011.05.002>
64. Telles SC, Alves RC, Chadi G. Periodic limb movements during sleep and restless legs syndrome in patients with ASIA A spinal cord injury. *J Neurol Sci*. 2011;303(1-2):119-23. DOI: <http://dx.doi.org/10.1016/j.jns.2010.12.019>
65. Krueger BR. Restless legs syndrome and periodic movements of sleep. *Mayo Clin Proc*. 1990;65(7):999-1006. PMID: 2198400 DOI: [http://dx.doi.org/10.1016/S0025-6196\(12\)65163-5](http://dx.doi.org/10.1016/S0025-6196(12)65163-5)
66. Mitchell UH, Myrer JW, Johnson AW, Hilton SC. Restless legs syndrome and near-infrared light: An alternative treatment option. *Physiother Theory Pract*. 2011;27(5):345-51. doi: 10.3109/09593985.2010.511440. DOI: <http://dx.doi.org/10.3109/09593985.2010.511440>
67. Mitchell UH, Johnson AW, Myrer B. Comparison of two infrared devices in their effectiveness in reducing symptoms associated with RLS. *Physiother Theory Pract*. 2011;27(5):352-9. doi: 10.3109/09593985.2010.502210. DOI: <http://dx.doi.org/10.3109/09593985.2010.502210>
68. Giummarra MJ, Bradshaw JL. The phantom of the night: restless legs syndrome in amputees. *Med Hypotheses*. 2010;74(6):968-72. doi: 10.1016/j.mehy.2009.12.009. DOI: <http://dx.doi.org/10.1016/j.mehy.2009.12.009>
69. Consensus document on fibromyalgia: the Copenhagen declaration. *J Musc Skel Pain*. 1993;1(3-4):295-312.
70. Hoffman DL, Dukes EM. The health status burden of people with fibromyalgia: a review of studies that assessed health status with the SF-36 or the SF-12. *Int J Clin Pract*. 2008;62(1):115-26. PMID: 18039330
71. Arnold LM, Crofford LJ, Mease PJ, Burgess SM, Palmer SC, Abetz L, et al. Patient perspectives on the impact of fibromyalgia. *Patient Educ Couns*. 2008;73(1):114-20. doi: 10.1016/j.pec.2008.06.005. DOI: <http://dx.doi.org/10.1016/j.pec.2008.06.005>
72. Govender C, Cassimjee N, Schoeman J, Meyer H. Psychological characteristics of FMS patients. *Scand J Caring Sci*. 2009;23(1):76-83. doi: 10.1111/j.1471-6712.2007.00592.x. DOI: <http://dx.doi.org/10.1111/j.1471-6712.2007.00592.x>
73. Neumann L, Buskila D. Quality of life and physical functioning of relatives of fibromyalgia patients. *Semin Arthritis Rheum*. 1997;26(6):834-9. DOI: [http://dx.doi.org/10.1016/S0049-0172\(97\)80027-3](http://dx.doi.org/10.1016/S0049-0172(97)80027-3)
74. Carmona L, Ballina J, Gabriel M, Laffon A; EPISER Study Group. The burden of musculoskeletal diseases in the general population of Spain: results from a national survey. *Ann Rheum Dis*. 2001;60(11):1040-5. DOI: <http://dx.doi.org/10.1136/ard.60.11.1040>
75. Haq AS, Darmawan J, Islam MN, UDGin Mz, Das BB, Rahman F, et al. Prevalence of rheumatic diseases and associated outcomes in rural and urban communities in Bangladesh: a COPCORD study. *J Rheumatol*. 2005;32(2):348-53.
76. Senna ER, De Barros AL, Silva EO, Costa IF, Pereira LV, Ciconelli RM, et al. Prevalence of rheumatic diseases in Brazil: a study using the COPCORD approach. *J Rheumatol*. 2004;31(3):594-7.
77. Assumpção A, Cavalcante AB, Capela CE, Sauer JF, Chalot SD, Pereira CA, et al. Prevalence of fibromyalgia in a low socioeconomic status population. *BMC Musculoskelet Disord*. 2009;10:64. doi: 10.1186/1471-2474-10-64.
78. Santos AM, Burti JS, Lopes JB, Scazufca M, Marques AP, Pereira RM. Prevalence of fibromyalgia and chronic widespread pain in community-dwelling elderly subjects living in São Paulo, Brazil. *Maturitas*. 2010;67(3):251-5. PMID: 20708357 DOI: <http://dx.doi.org/10.1016/j.maturitas.2010.07.006>
79. Cardiel MH, Rojas-Serrano J. Community based study to estimate prevalence, burden of illness and help seeking behavior in rheumatic diseases in Mexico City. A COPCORD study. *Clin Exp Rheumatol*. 2002;20(5):617-24.
80. Goldenberg DL, Simms RW, Geiger A, Komaroff AL. High frequency of fibromyalgia in patients with chronic fatigue seen in a primary care practice. *Arthritis Rheum*. 1990;33(3):381-7. DOI: <http://dx.doi.org/10.1002/art.1780330311>
81. Cavalcante AB, Sauer JF, Chalot SD, Assumpção A, Mastutani LA, Lage LV, et al. A prevalência de fibromialgia: uma revisão de literatura. *Rev Bras Reumatol*. 2006;46(1):40-8. DOI: <http://dx.doi.org/10.1590/S0482-50042006000100009>
82. Wolfe F, Ross K, Anderson J, Russell IJ, Hebert L. The prevalence and characteristics of fibromyalgia in the general population. *Arthritis Rheum*. 1995;38(1):19-28. PMID: 7818567 DOI: <http://dx.doi.org/10.1002/art.1780380104>
83. Yunus MB. Fibromyalgia and overlapping disorders: the unifying concept of central sensitivity syndromes. *Semin Arthritis Rheum*. 2007;36(6):339-56. PMID: 17350675 DOI: <http://dx.doi.org/10.1016/j.semarthrit.2006.12.009>
84. Brady DM, Schneider MJ. Fibromyalgia syndrome: a new paradigm for differential diagnosis and treatment. *J Manipulative Physiol Ther*. 2001;24(8):529-41. DOI: <http://dx.doi.org/10.1067/mmt.2001.118202>
85. Russell IJ, Orr MD, Littman B, Vipraio GA, Alboukrek D, Michalek JE, et al. Elevated cerebrospinal fluid levels of substance P in patients with the fibromyalgia syndrome. *Arthritis Rheum*. 1994;37(11):1593-601. PMID: 7526868 DOI: <http://dx.doi.org/10.1002/art.1780371106>
86. Larson AA, Giovengo SL, Russell IJ, Michalek JE. Changes in the concentrations of amino acids in the cerebrospinal fluid that correlate with pain in patients with fibromyalgia: implications for nitric oxide pathways. *Pain*. 2000;87(2):201-11. DOI: [http://dx.doi.org/10.1016/S0304-3959\(00\)00284-0](http://dx.doi.org/10.1016/S0304-3959(00)00284-0)
87. Moldofsky H. The significance of dysfunctions of the sleeping/waking brain to the pathogenesis and treatment of fibromyalgia syndrome. *Rheum Dis Clin North Am*. 2009;35(2):275-83. doi: 10.1016/j.rdc.2009.05.008. DOI: <http://dx.doi.org/10.1016/j.rdc.2009.05.008>

88. Juhl JH. Fibromyalgia and the serotonin pathway. *Altern Med Rev*. 1998;3(5):367-75.
89. Mease P. Fibromyalgia syndrome: review of clinical presentation, pathogenesis, outcome measures, and treatment. *J Rheumatol Suppl*. 2005;75:6-21. PMID: 16078356
90. Lautenbacher S, Rollman GB. Possible deficiencies of pain modulation in fibromyalgia. *Clin J Pain*. 1997;13(3):189-96. PMID: 9303250 DOI: <http://dx.doi.org/10.1097/00002508-199709000-00003>
91. American College of Sports Medicine Position Stand. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults. *Med Sci Sports Exerc*. 1998;30(6):975-91.
92. Goldenberg DL, Burckhardt C, Crofford L. Management of fibromyalgia syndrome. *JAMA*. 2004;292(19):2388-95. PMID: 15547167 DOI: <http://dx.doi.org/10.1001/jama.292.19.2388>
93. Busch AJ, Barber KA, Overend TJ, Peloso PM, Schachter CL. Exercise for treating fibromyalgia syndrome. *Cochrane Database Syst Rev*. 2007;17(4):CD003786.
94. Brosseau L, Wells GA, Tugwell P, Egan M, Wilson KG, Dubouloz CJ, et al. Ottawa Panel evidence-based clinical practice guidelines for aerobic fitness exercises in the management of fibromyalgia: part 1. *Phys Ther*. 2008;88(7):857-71. doi: 10.2522/ptj.20070200. DOI: <http://dx.doi.org/10.2522/ptj.20070200>
95. Nichols DS, Glenn TM. Effects of aerobic exercise on pain perception, affect, and level of disability in individuals with fibromyalgia. *Phys Ther*. 1994;74(4):327-32.
96. Meiworm L, Jakob E, Walker UA, Peter HH, Keul J. Patients with fibromyalgia benefit from aerobic endurance exercise. *Clin Rheumatol*. 2000;19(4):253-7. DOI: <http://dx.doi.org/10.1007/s100670070040>
97. Meyer BB, Lemley KJ. Utilizing exercise to affect the symptomology of fibromyalgia: a pilot study. *Med Sci Sports Exerc*. 2000;32(10):1691-7. PMID: 11039639 DOI: <http://dx.doi.org/10.1097/00005768-200010000-00005>
98. Holtgreffe K, McCloy C, Rome L. Changes associated with a quota-based approach on a walking program for individuals with fibromyalgia. *J Orthop Sports Phys Ther*. 2007;37(12):717-24. doi: 10.2519/jospt.2007.2607. DOI: <http://dx.doi.org/10.2519/jospt.2007.2607>
99. Ramsay C, Moreland J, Ho M, Joyce S, Walker S, Pullar T. An observer-blinded comparison of supervised and unsupervised aerobic exercise regimens in fibromyalgia. *Rheumatology (Oxford)*. 2000;39(5):501-5. DOI: <http://dx.doi.org/10.1093/rheumatology/39.5.501>
100. Martin L, Nutting A, MacIntosh BR, Edworthy SM, Butterwick D, Cook J. An exercise program in the treatment of fibromyalgia. *J Rheumatol*. 1996;23(6):1050-3. PMID: 8782139
101. Rooks DS, Gautam S, Romeling M, Cross ML, Stratigakis D, Evans B, et al. Group exercise, education, and combination self-management in women with fibromyalgia: a randomized trial. *Arch Intern Med*. 2007;167(20):2192-200. PMID: 17998491 DOI: <http://dx.doi.org/10.1001/archinte.167.20.2192>
102. Jentoft ES, Kvalvik AG, Mengschoel AM. Effects of pool-based and land-based aerobic exercise on women with fibromyalgia/chronic widespread muscle pain. *Arthritis Rheum*. 2001;45(1):42-7. DOI: [http://dx.doi.org/10.1002/1529-0131\(200102\)45:1<42::AID-ANR82>3.0.CO;2-A](http://dx.doi.org/10.1002/1529-0131(200102)45:1<42::AID-ANR82>3.0.CO;2-A)
103. Altan I, Bingöl U, Aykaç M, Koç Z, Yurtkuran M. Investigation of the effects of pool-based exercise on fibromyalgia syndrome. *Rheumatol Int*. 2004;24(5):272-7. DOI: <http://dx.doi.org/10.1007/s00296-003-0371-7>
104. Mannerkorpi K, Nyberg B, Ahlmén M, Ekdahl C. Pool exercise combined with an education program for patients with fibromyalgia syndrome. A prospective, randomized study. *J Rheumatol*. 2000;27(10):2473-81.
105. Keel PJ, Bodoky C, Gerhard U, Müller W. Comparison of integrated group therapy and group relaxation training for fibromyalgia. *Clin J Pain*. 1998;14(3):232-8. PMID: 9758073 DOI: <http://dx.doi.org/10.1097/00002508-199809000-00010>
106. van Santen M, Bolwijn P, Verstappen F, Bakker C, Hidding A, Houben H, et al. A randomized clinical trial comparing fitness and biofeedback training versus basic treatment in patients with fibromyalgia. *J Rheumatol*. 2002;29(3):575-81.
107. Marques AP, Ferreira EAG, Matsutani LA, Assumpção A, Capela CE, Pereira CAB. Efeito dos exercícios de alongamento na melhora da dor, flexibilidade e qualidade de vida em pacientes com fibromialgia. *Fisioter Mov*. 2004;17(4):35-41.
108. Marques AP, Assumpção A, Sousa A, Matsutani LA, Lage LV. Exercícios de alongamento ativo em pacientes com fibromialgia: efeito nos sintomas. *Fisioter Pesq*. 2007;14(3):18-24.
109. Valencia M, Alonso B, Alvarez MJ, Barrientos MJ, Ayán C, Martín Sánchez V. Effects of 2 physiotherapy programs on pain perception, muscular flexibility, and illness impact in women with fibromyalgia: a pilot study. *J Manipulative Physiol Ther*. 2009;32(1):84-92. doi: 10.1016/j.jmpt.2008.07.003. DOI: <http://dx.doi.org/10.1016/j.jmpt.2008.07.003>
110. Lavigne G, Manzini C, Huynh NT. Sleep Bruxism. In: Kryger MH, Roth T, Dement WC, eds. *Principles and Practice of Sleep Medicine*. 5th ed. Saint Louis: Elsevier; 2011. p.1128-39.
111. McNeely ML, Armijo Olivo S, Magee DJ. A systematic review of the effectiveness of physical therapy interventions for temporomandibular disorders. *Phys Ther*. 2006;86(5):710-25. PMID: 16649894
112. Alóe F, Gonçalves LR, Azevedo A, Barbosa RC. Bruxismo durante o sono. *Rev Neurociências*. 2003;11(1):4-17.
113. Okeson JP. *Tratamento das desordens temporomandibulares e oclusão*. 4a ed. São Paulo: Artes Médicas; 2000.
114. Maluf SA. *Efeitos da reeducação postural global e do alongamento estático segmentar em portadoras de disfunção temporomandibular: um estudo comparativo*. [Tese de doutorado]. São Paulo: Universidade de São Paulo; 2006.
115. Maydana AV, Tesch RS, Denardin OVP, Ursi WJS, Dworkin SF. Possíveis efeitos etiológicos para desordens temporomandibulares de origem articular. *Dental Press J Orthod*. 2010;15(3):78-86. DOI: <http://dx.doi.org/10.1590/S2176-94512010000300010>
116. Tenreiro M, Santos R. *Terapia Manual nas disfunções da ATM*. 1a ed. Rubio; 2011.
117. Smékal D, Velebová K, Hanáková D, Lepsiková M. The effectiveness of specific physiotherapy in the treatment of temporomandibular disorders. *Acta Univ Palacki Olomuc*. 2008;38(2):45-53.
118. Medicott MS, Harris SR. A systematic review of the effectiveness of exercise, manual therapy, electrotherapy, relaxation training, and biofeedback in the management of temporomandibular disorder. *Phys Ther*. 2006;86(7):955-73. PMID: 16813476