Can Chiropractic Care have an Effective Role in Treating Restless Legs Syndrome?

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ABSTRACT

Objective: This article provides an overview of literature of Restless Leg syndrome (RLS). Following the overview of incidence, symptomatology, pharmacological treatment methods, and on the effectiveness of chiropractic care on patients diagnosed with Restless Leg Syndrome. Many types of therapies associated with chiropractic care will be discussed in this article including: nutrition, physiotherapy, acupuncture, and chiropractic manipulation.

Data Collection: The information and studies reviewed were obtained from Medline, the Index to Chiropractic Literature, EBSCO, Internet, Mercola, and PubMed.

Data Synthesis: The references utilized were selected on the basis of applicable information, especially studies from indexed journals that provided proposed mechanisms implicated in the pathogenesis of RLS and both pharmacological and non-pharmacological methods of treatment.

Conclusions: The literature from the articles reviewed shows that there are many different causes of RLS depending on whether it is primary or secondary. There are many affective ways in treating RLS both allopathic and non-allopathic. Unfortunately, no articles could be found on the effects of chiropractic manipulation on RLS. However, Doctors of Chiropractic are more than able to teach RLS patients about proper nutrition and able to perform many of the alternative methods of treatment. Chiropractors are able to effectively patients with RLS and D.C. should play a role in the patient's care.

Key Words: Restless Leg Syndrome, Chiropractic, Acupuncture, Physiotherapy, Nutrition.

INTRODUCTION

Restless Leg syndrome is a sensory/movement disorder that affects up to 29% of the general population. Women are affected more frequently than men. It is one of the most common causes of severe insomnia. Females are affected more frequently than males. The incidence of RLS increases with age. Children can also be affected by RLS. In over a third of patients symptoms start before the age of ten years. It is often mistaken for either Attention Deficit Hyperactive Disorder (ADHD) or "growing pains." [6]

The primary form of Restless Leg Syndrome is idiopathic, which is frequently in an autosomal dominant fashion. The secondary form of RLS means that it is caused by something else, such as peripheral neuropathy, uremia, iron deficiency, pregnancy, or in patients suffering from some neurodegenerative disorder such as Parkinson's disease. [6]

The pathophysiology of RLS is unclear, but central iron deregulation may alter central dopamine. Ferritin is one of the chief proteins' which allows irons to be stored in the body; it occurs in the gastrointestinal mucosa, liver, spleen, bone marrow, and reticuloendothial cells. Serum Ferriton levels are often low, even in the presence of normal values of hemoglobin, hematocrit, iron, and iron binding capacity. The only useful test to test for primary Restless Leg Syndrome is to check the serum ferritin level. [6]

Patients with restless leg syndrome cannot stay still for long because of the subjective need to move. The patient complains of a variety of sensations distributed in the legs, including pins and needles, crawling sensations, aching, itching, stabbing, heaviness, tension, burning, or coldness. The sensations appear during the transition from wake to sleep. Symptoms are relieved by movements or leg stimulation. [6]

The purpose of this paper is to review the literature regarding the etiology, symptomatology, differential diagnosis, and treatment of RLS. In addition, it is the intent of this review to serve as a resource for health care professionals so they are better able to recognize RLS as a clinical entity and to enhance their ability to care for RLS patients. The medications used for the treatment of RLS vary and have the possibility of side effects. This Literature review will also point out the possible pathological effects of the allopathic way of treating patients with RLS. [10]

DISCUSSION

Although experts have known about RLS for over thirty years, a broader lack of awareness appears to have severely limited diagnosis of the condition. A survey conducted by the Restless Legs Syndrome Foundation (RLSF) not only found that more than half of those who responded had never heard of RLS. “RLS is sometimes described as ‘the most common condition you’ve never heard of,’ “says Georgianna Bell, executive director of the RLSF. [10]

The National Institute of Neurological Disorders and Stroke **(**NINDS**)** say that some researchers estimate that RLS affects as many as 12 million people in the United States. However, others estimate a much higher occurrence because RLS is thought to be underdiagnosed or misdiagnosed. There are a number of people who do not seek medical attention, because they believe that their condition will not be taken seriously, their symptoms are too mild, or their condition is not treatable. [10]

Symptomatology of RLS

Restless Legs Syndrome (RLS) is a medical condition characterized by a circadian variation of unpleasant sensations in the legs, including burning, tugging, and tightening. These

symptoms occur will the patient is at rest, mainly in the evenings when the person is seated or at night in bed. Temporary relief happens when the patient moves his/her legs. [6] RLS is commonly associated with periodic leg movements of sleep and even involuntary leg movements when awake.

RLS is a form of sleep disorder that includes periodic limb movements in sleep (PLMS), a condition where involuntary motor movements of the limbs occur during the sleep cycle and lead to a shift in sleep stage and repeated awakenings. PMLS is more prevalent as age increases. Twenty percent of RLS patient do not report experience of involuntary motor movements during sleep. PMLS is the fourth-leading cause of insomnia. In one polysomnographic study of 26 RLS patients, sleep efficiency was decreased by 50 percent, leaving 30 percent of patients with less than 3.5 full hours of sleep nightly. [9] The presence of PLMS is supportive for the diagnosis of RLS. Approximately 80 percent of individuals with RLS experience PLMS, but less than one half of individuals with PLMS also have RLS. [2]

The problem with RLS is that it is either idiopathic or it may indicated a diverse range of underlying neurological and non-neurological disorders such as peripheral neuropathy, Parkinson's disease, uremia, iron deficiency, varicose veins, and rheumatoid arthritis, or may occur after gastrectomy. This will cause the treatment of RLS to be different from patient to patient. Restless legs may also develop in or be aggravated by pregnancy. [7]

The negative impact of RLS on quality of life has been shown to be equal to or greater than chronic obstructive pulmonary disease or myocardial infarction. [9]

Diagnostic Criteria

Many doctors are still unaware of the presentation of RLS and may mis-diagnose the patient. The International Restless Legs Syndrome Study Group has suggested four criteria for diagnosis: the desire to move the extremities, often associated with paraesthesiae or dyaethesiae, motor restlessness, aggravation of symptoms by rest and at least temporary relief by activity, and worsening of symptoms in the evening or night. [7]

RLS is classified as either familial (primary RLS), with a clear genetic component, or acquired (secondary RLS). Primary RLS occurs in approximately 50 percent of first-degree relatives of those with RLS and is believed to be related to an inherited defect in dopamine metabolism. Acquired RLS involves altered iron metabolism and occurs in a variety of patient populations. (Lyn Patrick) If RLS is secondary to another underlying condition, treatment of that condition may improve or resolve the symptoms of RLS. [7]

Differential Diagnosis

The differential diagnosis of RLS includes nocturnal leg cramps, claudication, peripheral neuropathy, and akathisia. [9] Several other disorders resemble RLS and are differentiated by symptom description, laboratory testing, and other appropriate diagnostic workup. Neuropathies and radiculopathies can be ruled in or out by electrophysiological assessment and testing blood levels of glucose, thyroid hormone, vitamin B6 and B12. [7]

Evaluation of RLS

Different tools are available that measure severity of the symptoms and their impact on a person's life and track a person's progress or decline: the IRLSSG rating scale, Johns Hopkins RLS severity scale, RLS quality of life instrument, Epworth sleepiness scale, and the fatigue visual analog scale. The IRLSSG rating scale is a 10-question scale, developed by the IRLSSG as a means of assessing the severity of RLS and of tracking changes in symptoms associated with this pathology. It assesses the impact of RLS in a patient's quality of life and function. Each question

can be answered with one of five possible answers with attached points (0–4) according to severity. Therefore, a maximal score of 40 can be obtained. Generally, an IRLSSG rating scale score between 1 and 10 corresponds to “mild”, 11–20 “moderate”, 21–30 “severe”, and 31–40 “very severe” RLS. The Johns Hopkins RLS severity scale was the first published clinical scale to assess RLS severity. It consists of only one item, which asks for the time of day the symptoms start. Four possible answers are linked to scores as follows: 0 = no symptoms, 1 = bedtime-only symptoms (after or within an hour of going to bed), 2 = evening and bedtime symptoms (starting at or after 6 pm), and 3 = day and night symptoms (starting before 6 pm). This scale was mostly created for screening and epidemiological research. The RLS quality of life instrument consists of 17 questions assessing domains such as daily function, social function, sleep quality, and emotional wellbeing. For each question, the possible answers range from “never” to “very often”, with an attached score ranging from 5 to 1 respectively. The scores for the different domains are calculated separately. The Epworth sleepiness scale is used to determine the level of daytime sleepiness by giving the patient eight situations and asking for the chance of dozing off or falling asleep during those scenarios. The four possible answers are each linked to 0–3 points respectively. A score of 10 or more is considered “sleepy”, a score of 18 or more is considered “very sleepy”. The fatigue visual analog scale is a unidimensional fatigue measurement. Here the patient has to mark his/her level of experienced fatigue (during a determined time frame) on a 10-cm line. Its anchors are “no fatigue” on one side and “worst possible fatigue” on the other. [8]

Pathophysiology

Research has identified abnormalities in dopamine and iron function in the central nervous system in individuals with RLS, although these relationships are not fully understood. (AAFP)The symptoms of RLS reflect a circadian fluctuation of dopamine in the substantia nigra and the putamen. Patients with RLS have lower levels of dopamine in the putamen and the substantia nigra. Iron plays a central role in the etiology of RLS. It is a cofactor in tyrosine hydroxylase, the rate limiting step in the conversion of tyrosine to dopamine. [7]

If patient's ferritin levels are below 50 ug/L, he or she will be given 325 mg of ferrous sulfate two to three times per day for 3 to 4 months until ferritin levels exceed 50 ug/L and saturations surpass 20%. Patients are often placed on dopamine agonists. Patients with moderate to severe RLS will be placed on Pramipexole and Ropinrole for bedtime use. Benzodiazepines (e.g., clonazepam) and opioids (e.g., oxycodone) may be affective, but have higher risks in older people and are not first-line therapy. Carbidopa and Levodopa can be used on an "as needed basis" for in frequent symptoms. [6]

Although ferritin levels in RLS are often "low normal" a specific threshold of inadequate iron storage has been identified as the determining factor in these patients. Ferritin concentrations of <50 ng/mL have been correlated with decreased sleep efficiency, increased leg movements in sleep, and increased symptom severity. [7]

Iron concentrations in the blood have a circadian rhythm, exhibiting a 50- to 60-percent lower serum level at night compared to daytime levels. The lowest point in serum iron levels has been found to coincide with the maximal severity of RLS symptoms and responsible for the worsening of RLS symptoms in the evening. One study found that nighttime levels of ferritin in cerebrospinal fluid (CSF) were significantly lower in RLS. [7]

Even when levels of serum iron, serum ferritin, or serum transferring are not lower than normals, CSF levels of storage iron have been shown to be lower in RLS. CSF ferritin was 65% lower and CSF transferring was 300% higher in patients with RLS compared to age-matched controls. Elevation of CSF transferring is compensatory for low CNS iron levels. The substantia nigra appears to have both decreased iron uptake and storage activity in RLS. [7]

Iron is necessary for the production of tyrosine hydroxylase, which is a dopamine transporter and the rate limiting step in the production of levodopa, which is then decarboxylated to dopamine. This takes place in the substantia nigra, where low iron levels have been identified in RLS. [7]

At-Risk Populations for secondary RLS

Pregnancy, end-stage renal disease, obesity, certain medications, and iron deficiency are associated with a significantly higher incidence of RLS. [7]

Pregnancy

During pregnancy the incidence of iron deficiency is significant; less than optimal iron intake can occur in 90% of pregnant American women. Research estimates 11-27% of pregnant women experience RLS at some point during pregnancy, most commonly in the third trimester. RLS is the most common movement disorder in pregnancy and generally remits with delivery. [7]

The etiology of RLS in pregnancy is related to iron and folate insufficiency. During normal pregnancy, iron requirement increases 3- to 4-fold and folate requirement increases 8- to 10-fold. Deficiency of either nutrient has been found in pregnant women with RLS, and resolution of RLS appears to correlate with normalization of folate and iron levels. [7]

End-stage Renal Disease (ESRD)

A survey found that 62% ESRD patients complain of RLS. Evidence for a possible relationship of iron deficiency to RLS in this patient population has been explained by the universal occurrence of anemia, due to inadequate production of erythropoietin. Anemia in ESRD is associated with worsening iron deficiency due to loss from hemodialysis. Treatment of ERSD-associated anemia with erythropoietin has been shown to be highly effective in causing remission of RLS symptoms. [7]

Obesity and RLS

Obesity and RLS are both associated with hypofunction of dopamine in the CNS. In a case-study, obese individuals (BMI greater than 40) had a significantly lower number of striatal

dopamine D2 receptors. A study was performed by Xiang Gao MD and Michael Schwarzchild MD using 4,196 women and 948 men with true RLS. All of the patients Body Mass Index (BMI) were greater than 30. Results have shown that patients with a larger BMI in early adulthood (18-21 years) and mid-life or later (age 40 years and higher) were shown to have a higher prevalence of RLS. [13]

Medications that can Produce RLS Side-effects

Medications that can induce or exacerbate RLS include selective serotonin reuptake inhibitors (SSLIs), lithium, dopamine agonists, tricyclic antidepressancts, antiemetrics, antipsychotics, and any medication with antidopaminergic activity. [7]

Treatment of RLS

Pharmacological Treatment

Dopaminergic agents are considered the pharmacological treatment of choice for primary RLS. Some of the dopaminergic drugs include: pergolide, ropinrole, pramipexole, and cabergoline.

Dopamine Precursor: Levodopa

Levodopa plus dopa-decarboxylase inhibitor (DDI) is useful to treat intermittent RLS symptoms. Because the effect of levodopa can wear off early in the night (duration of action 2–4 hours), longer acting forms may also be useful. An important side effect of levodopa, which limits its use to intermittent therapy, is augmentation. Augmentation is an increase in severity of symptoms with regular long term therapy. Typically, symptoms start to occur earlier in the day compared to the start of therapy. Often the symptoms are more intense or spread to the trunk and arms. Up to 70% of patients taking levodopa develop augmentation, and the risk increases with higher doses. [14]

Importantly, increasing the levodopa dose may make augmentation worse and if it occurs, levodopa should be stopped and another agent trialled. Rebound, the recurrence of RLS in the early morning, also occurs in 20–35% of patients taking levodopa. Rebound probably reflects the end of the drug action of the dopamine precursor.

Common adverse effects from taking Levodopa include: nausea, vomiting, orthostatic hypotentsion, hallucination, and insomnia. [14]

Dopamine Agonists: Pergolide, Pramipexole, and Ropinrole

These agents have a longer onset of action than levodopa (90–120 minutes) and therefore are not ideal for intermittent use once symptoms have started. They are the drugs of choice for daily RLS.42,43 Augmentation is less common (30%) compared to levodopa (70%). Unlike dopamine precursors, augmentation with dopamine agonists (DA) may be treated by giving divided doses and/or increasing the dosage. Dopamine agonists are either ergot derived or nonergot derived:

• Ergot derived DA such as pergolide, cabergoline and bromocriptine, are indicated to treat Parkinson disease or endocrine disorders. Low dose cabergoline (1 mg) was shown to be effective in moderate to severe RLS. However, ergot derived DA are associated with rare but significant side effects, namely valvular heart disease and retroperitoneal or pleuropulmonary fibrosis. Hence, these drugs are not commonly used or recommended for RLS

• Nonergot DA, including ropinirole and pramipexole, are becoming more popular as first line treatment options for moderate to severe RLS because of their more attractive side effect profile compared to the ergot agents as described above. Ropinirole has been studied in the largest double blind, placebo controlled trial in RLS supporting its effectiveness in reducing RLS symptoms. Pramipexole has only recently been introduced in Australia, despite widespread use elsewhere. Interestingly, there have been several reports of unusual compulsive behaviors occurring in those taking dopamine agonists, including pathological gambling and hypersexuality. Encouragingly very recent studies have confirmed the efficacy of a 24 hour transdermal patch using the dopamine agonist rotigotine to relieve both night and day time symptoms.This drug is not currently available in Australia, but may have a great

potential to alleviate many of the difficulties associated with dosing patients with RLS. [14]

Pergolide

Pergolide requires special monitoring due to increased incidence of valvular fibrosis and other fibrotic side effects. The FDA has withdrawn pergolide from the market following recent safety information published by the New England Journal of Medicine (January 2007) confirming the association of valvular heart disease in patients exposed to pergolide. Pergolide is contraindicated in patients with a history of cardiac, pulmonary or retroperitineal fibrosis, or signs of cardiac valve abnormalities. Common side effects are nausea (59%), headache (32%), asthenia (18%), rhinitis (21%), vomiting (18%), and dizziness (22%). [2]

Cabergoline

Cabergoline is contraindicated in patients with a history of cardiac, pulmonary and retroperitineal fibrosis, or signs of cardiac abnormalities. The most common side effects were nausea (35%), constipation (20%), and headache (20%). [2]

Alternative Treatments for RLS

Lifestyle Factors

Little information is available about the effects of lifestyle on the symptoms of RLS. Limiting caffeine, tobacco, and alcohol use may improve symptoms. Activities that provide mental stimulation may also provide relief. One survey showed a higher prevalence of RLS in persons who were sedentary and overweight. A small randomized controlled trial of 23 patients demonstrated improvement in symptoms of RLS with a program consisting of lower body resistance training and aerobic exercise. [2]

Iron Replacement

Iron replacement in RLS has not been shown to be effective when ferritin levels are above 50 ng/mL. Although there is no standard treatment for iron deficiency in RLS, a suggested protocol includes 50-65 mg elemental iron with 200 mg vitamin C on an empty stomach 1-3 times daily, depending on the degree of deficiency. The goal is to reach a serum ferritin level of 60 ng/mL, with iron studies repeated every 6 months. To avoid worsening of an undiagnosed hemochromatosis, transferring saturation levels should not rise over 45 percent. [7]

Folic Acid

Folic acid supplementation has been shown to improve the symptoms in some RLS patients. Folic acid is involved in the production of dopamine in the CNS. Folic acid, as 5-ethyltetrahydrofolare, increases production of CNS tetrahydrobiopterin, a cofactor in tyrosine hydroxylase production. [7]

Physical Activity

Epidemiologic evidence indicates that lack of exercise is a strong predictor of and a significant risk factor for RLS. Physical activity and exercise have long been the only nonpharmacological treatment options available to RLS sufferers. In fact, by definition, RLS is the urge to move that is at least partially relieved by movement. It was recently shown in a RCT that a 3-day per week exercise program of aerobic and lower-body resistance training significantly decreased RLS symptom severity. While the authors did not attempt to explain the reasons for why or how exercise was successful in decreasing RLS severity, one can surmise that the increase in blood flow brought on by activity could play a role. Shear forces between the inner wall of the endothelium and the moving blood stimulate the enzyme nitric oxide (NO) synthase. Once generated, NO diffuses into the smooth muscle of the endothelium and then

quickly diffuses through the muscle tissue of the blood vessel. There it activates guanylate cyclase, which then activates the second messenger cGMP (cyclic guanosine monophosphate).

Several steps follow and culminate in the relaxation of smooth muscles in the blood vessel. This leads to vasodilation and consequently to increased local blood flow. NO is also scavenged by hemoglobin in the blood. Under low pO_2 (partial pressure of oxygen) conditions, during physiologic "hypoxia", the red blood cells release NO to increase blood flow. Another possible reason for the success of exercises in the treatment of RLS symptoms could be the exercise-induced release of endorphins. Endorphins are endogenous opioid polypeptide compounds, produced by the pituitary gland and hypothalamus that produce analgesia and a sense of well-being. Another central change occurring with exercise, and therefore a further potential mechanism with which physical activity can aid in decreasing RLS symptoms, is the increased release of dopamine. It has been shown that especially during high-intensity exercise dopaminergic neurotransmission changes. A study assessing the incidence of RLS during sleep following acute physical activity in spinal cord injury subjects found a significant reduction in RLS as measured by polysomnographic sleep parameters and decrease of leg movements. The authors reject the hypothesis that dopamine deficiency could be involved in the symptoms felt by spinal cord-injured EKD sufferers because of their spinal cord trauma. Instead, they suggest that the release of endorphins after physical activity may be the cause for the symptom reduction. [8]

Pneumatic Compression Devices

One of the first modern-day (1940s and 1950s) hypothesis attempting to explain the etiology of RLS symptoms associated it with decreased blood flow. Ekbom, too, believed that vasodilators given to RLS sufferers would decrease the symptoms. The vascular hypothesis was later neglected but revived in 2005, when increased vascular blood flow with pulsed compression devices was shown to significantly decrease RLS symptoms in six patients. Other

studies have followed confirming these findings. The pneumatic compression devices were applied to the thigh and leg regions; the parameters used were 40 cm H_2O of air pressure intermittently for 1 hour. It is hypothesized that vascular compression stimulates the release of endothelial mediators (ie, NO) that then can modulate RLS symptoms. It is also possible that intermittent compression, which enhances venous and lymphatic drainage, could relieve subclinical ischemia. These findings are not surprising, taking into account that a high prevalence (36%) of RLS in patients present with chronic venous disorder. [8]

Massage

Tactile and temperature stimulation, including massage or hot baths, can also be successful in decreasing symptoms associated with RLS. While many authors mention these modalities as potential treatment options and numerous websites recommend them, there is a paucity of scientific trials confirming their efficacy. However, one case report describes a 3-

week massage regimen that decreased RLS symptoms significantly. This massage was given for 45 minutes twice a week using techniques such as Swedish massage (effleurage, petrissage), myofascial release, friction to tendinous attachments, stretches, and direct pressure to hip and lower extremity muscles. The symptoms returned after 2 weeks post treatment. The author suggests that the natural release of dopamine following massage could have been responsible for the amelioration of the symptoms. Massage has shown to increase dopamine levels in urine by an average of 28% in different conditions. Another speculation on the working mechanism of massage in the treatment of RLS is the counter stimulation it provides to the cerebral cortex. The tactile stimulation could supersede afferent input associated with RLS symptoms or at least partially modulate the perception of discomfort in the legs. Another explanation involving the central nervous system is the possibility that tactile and temperature stimulations traveling in the spinothalamic tract may modulate neural activity in the thalamus. Bucher et al have shown that activation of the thalamus is associated with sensory leg discomfort in idiopathic RLS

patients. A fourth explanation could be the mechanically induced increase of circulation. Massage moves venous blood to the heart, transporting nutrients to the tissue and metabolic products away from the tissue. Consequently, the potentially de-oxygenated tissue receives oxygen which then can restore vascular blood gas balance. [8]

Near-infrared spectroscopy (NIR) Light

NIR light has been used in the treatment of neuropathy to increase sensation and decrease pain, wound healing, and more recently in the treatment of RLS. The proposed mechanism of NIR light therapy is its ability to generate NO in the endothelium by activating the enzyme nitric oxide synthase (NOS)-3, similar to exercise-induced NOS-3 activation. “Intensive illumination”, such as during NIR light treatment, can also free NO from hemoglobin and thus make it bio-available. The discomfort that accompanies the RLS-related urge to move could be caused by the lack of tissue oxygen, which would be offset by an increase in blood flow. The urge to move may be a subconsciously driven mechanism to augment blood flow and tissue perfusion. Moving, such as walking or rubbing of legs, diminishes RLS symptoms as it enhances circulation. Therefore, treatment with a vasodilator, such as NIR-induced NO, could conceivably temporarily decrease the symptoms associated with RLS. Even a prolonged reduction of symptoms (up to a couple of weeks post treatment) has been observed. This was explained with a potential systemic effect of light therapy. This systemic effect could be responsible for either continued NO production or other changes in the tissue, leading to diminished symptoms. Additionally, NO has influence on neurotransmission. It is one of the substances that influences nerve impulse transfer as it assists in converting nerve signals as they cross synapses. This quality of NO might also be involved in reducing symptoms associated with RLS. All things considered, NIR could positively impact RLS symptoms by numerous methods. [8]

Chiropractic Manipulation

Chiropractic manipulation has been shown to have a powerful influence on all three

branches of the nervous system (Peripheral Nervous System, Central Nervous System, and Autonomic Nervous System). It plays an important role in balancing one's nervous system by stimulating perprioceptive nerves that are in a hypofuction state. This intern has an inhibiting effect on the production nociceptive nerve's neurotransmitter, Substance P. With fewer Substance P neurotransmitter in the receptors area, there is a decreased chance of a spillover effect onto the surrounding nerves. This has a calming effect on the surrounding nerves whether it is an autonomic nerve, a peripherial nerve, or a central nerve. No articles were able to be found on the effectiveness of treating RLS with chiropractic manipulation.

Prognosis

RLS has a variable course, but symptoms tend to progress with advancing age. Some people may experience a spontaneous improvement in their symptoms for a period of time, but symptoms tend to recur. Individuals with RLS secondary to an underlying condition may have improvement or resolution of symptoms if the underlying condition is treated. [2]

Conclusion

The literature from the articles reviewed shows that there are many different causes of RLS depending on whether it is primary or secondary. There are many affective ways in treating RLS both allopathic and non-allopathic. Unfortunately, no articles could be found on the effects of chiropractic manipulation on RLS. However, Doctors of Chiropractic are able to teach RLS patients about proper nutrition and able to perform many of the alternative methods of treatment. Therefore, chiropractors are able to effectively patients with RLS and Doctors of Chiropractic should play a role in the patient's care.

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