REVIEW ARTICLE

An Osteopathic, Non Pharmacologic Approach to Parkinson's Disease, Restless Leg Syndrome & Essential Tremor

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Movement disorders are a heterogeneous group of complex sensorimotor neurological conditions involving involuntary abnormal movements, deficiencies or changes in normal motion. Parkinson's disease, restless leg syndrome, and essential tremor are the most common movement disorders, and their prevalence is increasing with the growth of the aging population. These neurological conditions affect multiple body systems and are best managed with a multifaceted treatment approach. Behavioral, lifestyle, and/or psychosocial modifications and treatments for sleep, mood, exercise, and nutrition to meet the increased metabolic demands are critical. Evidence suggests that osteopathic manipulative medicine may improve gait and motor function in Parkinson's disease as well as quality of life in restless leg syndrome. In addition, osteopathic manipulative medicine may be used to treat many of the common symptoms in these patients such as constipation, rib dysfunction, back pain, and tendonopathy in the same way as in patients without movement disorders. The normalization of autonomic nervous system function through manipulation of the suboccipital area, cervical myofascia, and rib raising would be particularly beneficial in these disorders. Integration of the five models of osteopathic care by the family physician can improve symptom management and overall quality of life.

INTRODUCTION

Movement disorders are a heterogeneous group of complex sensorimotor neurological conditions that are classified as having either a general increase in movement, hyperkinetic, or a paucity of movement, hypokinetic. The frequency of most movement disorders tends to increase with age. With the increasing size of the elderly population, the prevalence of most movement disorders is expected to double by the year 2050.[1] Movement disorders that are routinely seen in a primary care setting include Parkinson's disease (PD), restless leg syndrome (RLS), and essential tremor (ET). Each of these conditions can negatively impact a patient's quality of life. Early diagnosis by osteopathic family physicians and education regarding treatment options may improve care, particularly in the awareness of the early symptoms that are often not attributed to a neurological disorder and their osteopathic treatment. Integration of osteopathic manipulative medicine (OMM) provides patients an adjunctive, non-invasive treatment option which may improve symptoms and quality of life.

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The central tenets of osteopathic medicine provide a framework for utilizing osteopathic diagnosis and manipulative techniques to improve our patients' natural physiologic functions. Conditions involving decreased joint range of motion or musculoskeletal pain are often manageable by techniques which improve mobility or reduce excessive muscle tone. Similarly, an infectious process of the lower respiratory tract may benefit from increased mobility of the thoracic cage and improved lymphatic flow.[2,3] The purpose of this article is to guide the family physician in an osteopathic approach to the care of PD, RLS and ET in addition to pharmacologic therapy.

THE FIVE MODELS OF OSTEOPATHIC TREATMENT

The five models of osteopathic care provide physicians with a framework to integrate osteopathic manual treatments (OMT) for PD, RLS, and ET. Application of direct active techniques can be helpful in engaging the patients' musculoskeletal system. Indirect passive techniques should be applied if the patient is too fatigued, unable to follow commands, or direct techniques are too painful to perform. The models may also aid the physician in providing patient education regarding goals of treatment and potential side effects prior to consent. The particular osteopathic techniques are outlined to the right. Osteopathic neuromusculoskeletal treatments may ease pain, provide symptomatic relief, and decrease the need for higher-risk medications or invasive procedures.

BIOMECHANICAL MODEL

The biomechanical model looks at the body as interconnected levers, pulleys, compression, and tension elements to address somatic dysfunctions of the musculoskeletal system. The hyperkinetic and hypokinetic control of movements frequently impacts the musculoskeletal system and connective tissues. Goals of OMT in this domain include restoring normal muscle tone, joint range of motion, natural alignment, and decreasing nociception from synovial joint distortion.

NEUROLOGICAL MODEL

The neurological model addresses sensorimotor and autonomic nervous system (ANS) control of the body by both the central and peripheral nervous systems, all of which are pertinent to PD, RLS, and ET. OMT for the nervous system relies on inherent reflexes, and it can be applied at the end muscle/organ or centrally at the spine or head. OMT to normalize ANS tone may decrease the need for pharmaceutical interventions.

RESPIRATORY-CIRCULATORY MODEL

The respiratory-circulatory model focuses on the proper motion of intra- and extravascular fluid around the body as well as optimizing breathing. The focus of OMT is on cardiovascular and pulmonary health, flow of blood and lymph, and motion of the associated musculoskeletal structures. The major musculoskeletal structures facilitating pulmonary ventilation are the diaphragm, ribs and spine. These and the lower leg muscles also facilitate blood and lymph circulation. Improved lymphatic flow decreases local pooling of inflammatory mediators and metabolic waste products. These factors may play roles in the etiology and/or progression of PD and RLS. Decreasing edema tends to improve pain. Improvement in tissue perfusion may aide in supporting the excess cellular respiration demands. In conditions that disrupt sleep, PD and RLS, improving respiration may reduce nighttime awakenings, muscular pain in the morning, and daytime somnolence.

METABOLIC-ENERGY MODEL

The metabolic-energy model takes into account the biochemical, potential and kinetic energy dynamics aspects of physiology. The main goal of this model is to improve energy production and metabolic efficiency. Daytime fatigue is frequently problematic in RLS and PD. Changes in neuromuscular physiology may prevent efficient coordination of concentric and eccentric muscle contraction and passive stretch and relaxation. This decreased efficiency may cost the body more energy production than in healthy movement control. Along with the increased energy demands, the metabolic energy balance in movement disorders is often worsened by nutritional deficits such as Vitamin B12, magnesium and iron deficiencies. It is imperative to obtain sufficient nutrients to meet the increased metabolic demands of disease states while avoiding excessive consumption, which itself can also exacerbate disease.

BEHAVIORAL MODEL

The behavioral model addresses the complex cognitive, emotional, behavioral, and habitual aspects of disease and health. This model seeks treatment through building rapport, sharing information and resources, lifestyle modification, counseling, and/or cognitive behavioral therapy. All neuropsychiatric disorders are exacerbated by stress, and stress has been shown to worsen outcomes in many chronic diseases. It is important to counsel patients and their family on behavioral interventions to decrease the stressful impact of chronic diseases.

METHODS

PubMed and Google Scholar databases were searched for guidelines in the diagnosis and treatment of PD, RLS, and ET. The secondary texts *An Osteopathic Approach to Diagnosis and Treatment*, *Foundations of Osteopathic Medicine* and *Atlas of Osteopathic Techniques* and primary research articles on multidisciplinary and osteopathic care for PD, RLS, and ET as well as their most common symptoms (tremor, constipation, mood disorders, daytime fatigue and somnolence, mobility, and pain) were reviewed. The most recent literature on these topics was selected for review. The information was evaluated utilizing concepts from Grading of Recommendations Assessment, Development and Evaluation (GRADE) as well as the authors' experience from a clinical and research perspective on treating people with these neurological conditions.[4] Concepts from GRADE utilized in the selection of key references included outcomes of interest being important to patients, greater benefit than risk, and relation to the target audience of providers. OMT modalities were chosen based on clinical research and clinical experience from practicing physicians who treat these disorders.

PARKINSON'S DISEASE

Parkinson's disease is a neurodegenerative disorder that affects millions of people worldwide. There are approximately 1 million people in the United States living with PD, and over 60,000 new cases are diagnosed each year.[5] Research suggests that PD is associated with a variety of external risks factors, including head trauma, exposure to pesticides, drug use, and others **(Table 1)**.[5] Emerging theories implicate breakdown of the blood brain barrier in many of these etiologies.[6] Less than 30% of PD cases are associated with a genetic susceptibility.[5,7,8]

Parkinson's disease is a hypokinetic movement disorder currently diagnosed clinically by its characteristic motor features of bradykinesia including decreased facial expression, rigidity of the musculoskeletal system, postural instability, and sometimes tremor **(Table 2).** Non-motor symptoms may be the initial presentation of the disease. Some common non-motor symptoms include loss of smell (anosmia), REM sleep behavior disorder, constipation, mood disorders, or autonomic nervous system (ANS) dysregulation of other organ systems (neurogenic disorders).[9]

Currently, the primary treatment goal is to improve quality of life by decreasing symptoms using both pharmacological and non-pharmacological approaches. Not all patients with PD are responsive to levodopa therapy, which increases the importance of having multiple management options. Research is being conducted on non-pharmacological rehabilitative approaches such as OMM, physical activity, social activity programs and support groups.[10]

BIOMECHANICAL

Techniques such as muscle energy, articulatory, counterstrain and myofascial release can improve range of motion, decrease pain and stiffness. In PD, OMM can target the motor features bradykinesia, rigidity, and "shuffling" gait. Goals of OMT following the biomechanical model are to decrease pain, increase the range of motion of rigid joints, and improve posture, gait, and balance. One study

demonstrated that OMT using muscle energy (ME) and passive articulatory techniques significantly improved stride length, limb velocity and upper extremity swing.[10] This study was limited due to the blinding process. The experimental design was non-uniform in that in the control group, participants received sham OMT without self-care pointers and in the active group participants received actual OMT with self-care pointers. Due to the continuation of treatment through self-care only in the active group, the researcher cannot know how much this contributed to the results. However, patients seeking help through OMT will most likely receive self-care techniques. A randomized, controlled trial demonstrated that a similar OMT protocol performed twice a week for six weeks significantly improved motor function in PD subjects compared to controls receiving counseling on PD-related topics, including mobility and nutrition, as measured by the Movement Disorder Society - Unified Parkinson's Disease Rating Scale (MDS-UPDRS) without changes in medications.[11] This study was limited in that there were a small number of participants. The active group was not uniform in results; some subjects had a dramatic improvement while one had less improvement and possibility of psychosomatic effect. However, considering the low risk of the OMT protocol in this population and patient and caregiver concern regarding motor function and the characteristic "shuffling" gait, the evidence highly supports that regular OMT using this protocol is recommended as a non-invasive approach. In PD there is also a five-fold increased risk of postural change which can result in marked pain and increased risk of falls.[12] OMT directed at the axial spine and connecting muscles to the extremities can potentially help to address camptocormia and other postural disorders. With RLS, ET, and PD resting tremor, there is excessive motor endplate stimulation which may yield an increase in resting muscle tone, muscle soreness or a chronic somatic dysfunction in the affected limbs similar to 'overuse' tendinopathy.

NEUROLOGICAL

In addition to the motor features of PD, there are non-motor features including dysregulation by the ANS, which can manifest as gastrointestinal dysmotility, shortness of breath, fatigue, and possibly decreased heart rate variability.[13, 14] OMT to normalize function is applied to the central nervous system, nerves, or ganglia of the ANS. Rib raising decreases molecular and physiological markers of sympathetic drive.[15] Research suggests that OMT to the suboccipital region affects the parasympathetic nervous system through the vagus nerve demonstrated by heart rate variability changes after suboccipital release and cervical myofascial techniques.[16] A proposed mechanism by which cranial OMM may improve central ANS and sensorimotor function is to improve lymph flow and venous drainage through the dural sinuses to decrease local toxin buildup.[17]

RESPIRATORY-CIRCULATORY

A clear link exists between PD and respiratory dysfunction. Patients often display a restrictive breathing pattern on pulmonary function testing. This may be multifactorial, including decreased respiratory drive associated with bradykinesia and hesitation, or trouble initiating motion, decreased thoracic excursion attributable to rigidity, and muscle spasms when forward-bending truncal dystonia (camptocormia) is present. In turn, the decreased thoracic excursion diminishes the intrathoracic pressure changes required for pulmonary ventilation and pumping of major lymphatic and afferent vessels. There is also an increased risk of cardiovascular disease in PD. Proposed mechanisms for this relationship includes dysautonomia and increased time spent sedentary [13,18] Among those immobilized by advanced disease, there is an increased risk of venous stasis with inability to clear inflammatory mediators and clotting that may be addressed with OMT. Techniques used in this model include muscle energy for rib somatic dysfunctions, doming the diaphragm, lymphatic pumps, thoracic inlet release, and paraspinal muscle inhibition.

METABOLIC-ENERGY

Many PD patients report becoming easily fatigued with minimal exertion, and fatigue is higher among those with greater rigidity or who are akinetic because their body works harder to move.[14] Patients with advanced disease may struggle to get sufficient nutrition due to inability to procure, prepare, or serve food for themselves. Some of these dietary issues can be managed with patient education and/or direct nutritional supplementation. PD patients may have difficulty with chewing and swallowing and/or gastrointestinal dysmotility. Osteopathic treatment in these patients should focus on balancing the energy intake/expenditure ratio by both decreasing the work of moving and improving neuromuscular aspects of eating and digesting as well as promoting good quality rest. One approach is OMT to improve oral motor function and swallowing by addressing restrictions to the muscle physiology. Particular attention should be directed at the lower face, cranial, and cervical bones, muscle tone and balance, and impingement or congestion on the glossopharyngeal, vagus and hypoglossal nerves.

BEHAVIORAL

Patients with PD have higher than average incidence of REM-sleep behavior disorder with underlying impaired sleep rhythm patterns. Most PD patients have hypomimia, or masked facies, largely due to hypokinesia, but the hypomimia may also involve poor emotional functioning. The prevalence of depression is higher than average in PD, and studies have found independent deficiencies in basic emotional processes such as empathy or recognizing emotion on the faces of others.[19] Behavioral training can improve both the patient's outward expression of emotion and their ability to process emotion in themselves or others. PD patients may also benefit from tailored dance, boxing or other exercise programs which help improve motor planning, decrease falls, and improve motion amplitude.[20]

Majority of OMT research for PD falls under the biomechanical model. Dysautonomia in PD has only recently been characterized and further studies are needed. There is sufficient evidence to recommend nutritional and behavioral management in PD.

RESTLESS LEG SYNDROME

While diagnostic criteria for Restless Leg Syndrome have changed over time, modern estimates in the US show a prevalence of 10-15% of the population with higher rates among females.

Incidence increased with parity (nulliparous females have incidence rates nearer to that of males). The age of o set is typically before 45 years with increasing severity of symptoms and sleep disturbance with aging.[21,22]

RLS is a common neurological disorder with motor, sensory, and circadian disruption. Patients feel an extreme urge to move their legs. This drive to move is associated with other uncomfortable sensations such as warmth, fizzing, pulling, aching, itching, throbbing or skin crawling.[21] These symptoms worsen with prolonged rest, and they lessen or are alleviated by movement. This results in trouble initiating sleep as patients struggle to remain in bed for extended periods. Many people with RLS also experience periodic leg movements of sleep which are intermittent episodes of involuntary, forceful jerks of the foot into dorsiflexion during sleep. This can result in nighttime awakenings as well as soreness and pain of the legs in the morning. In many cases, the combination of trouble initiating sleep with frequent awakenings during the night results in daytime somnolence.[22] Diagnosis is clinical, and the essential criteria outlined by the International RLS Study Group is in Table 2.[23]

RLS can be either primary idiopathic or secondary. Primary idiopathic RLS is thought to stem from a genetic cause as 25-75% of cases are familial. Some of the causes of secondary RLS may be reversible (Table 3). Initial testing should include iron panel (including ferritin), CBC, and a metabolic panel. Although the pathophysiology of RLS is not clear, secondary causes have given insight to potential pathophysiologies and treatments. Early research found RLS associated with aberrant basal ganglia function and dopamine neurotransmission linked with D2 receptor density changes. Epidemiological studies show a higher prevalence of RLS in patients with multiple sclerosis having spinal cord lesions as well as lumbar radiculopathy. Recent research on RLS pathophysiology showed abnormalities in the A11 posterior hypothalamus nucleus, which provides the primary dopaminergic innervation to the D1 and D3 receptors of the spinal cord gray matter. This suggests that spinal cord tracts and/or reflexes may be involved. The treatment of secondary RLS includes first treating the etiology. In cases of iron deficiency, renal failure, and drug toxicity, there is often rapid resolution of RLS symptoms with iron supplementation, dialysis, and medication management, respectively.

BEHAVIORAL

The gold standard treatment in all patients is improved sleep hygiene and exercise, as they have been shown to be effective at decreasing symptoms and improving quality of life long-term.[24, 25] Patients should first be counseled on these low risk, high benefit lifestyle changes.

NEUROLOGICAL & BIOMECHANICAL

Various classes of drugs have been shown to be effective for short-term relief. The International RLS Study Group 2013 task force performed a meta-analysis using current ICSI stratification system which showed Level A or high quality evidence for use of the GABA analogue pregabalin and dopaminergic agents such as pramipexole, ropinirole and carbidopa-levodopa for short term, 6 months to one year depending on the drug. High quality evidence generally means that further research is very unlikely to change the confidence in the estimated effect of the medication. Considering patient concerns, tolerability of the medication side-effects may limit their usefulness. In addition, long-term use is unfortunately associated with either decreased efficacy, augmentation of symptom severity, or shifting symptoms to begin earlier in the day. No medications have shown reliable long term efficacy at this time.[24] There is moderate to high quality evidence suggesting that massage, a vibration device targeting the abductor and flexor hallucis (Relaxis),[26] or electrical stimulation (via TENS machine) would also significantly improve symptoms.[27, 28] There is potential for other non-pharmacological treatments such as OMM, as it may affect central and peripheral structure-function targets in RLS (Table 4).

The association of RLS with pathology of the spinal cord and nerve roots suggests that patients with lower body somatic dysfunctions would benefit from OMT and/or physical medicine to alleviate restrictions affecting spinal cord health. Counterstrain tender-points pertinent to RLS were identified by Dr. Douglas Longden in collaboration with Lawrence Jones, DO. The Counterstrain Assessed for Restless Legs (CARL) trial compared treatment of RLS patients with non-specific lower extremity counterstrain tender-points with treatment of the Longden RLS-specific points. The Longden RLS-specific points were those related to L5 with particular attention to the anterior points, progressing from the most severely tender to the less tender points. The Jones AL5 located at the anterior portion of the pubic bone, lateral to the pubic symphasis where the rectus abdominis inserts that is treated with marked hip flexion and ipsilateral rotation and contralateral sidebending for fine-tuning. In addition, there is AL5 1 cm lateral to the pubic symphasis pushing from superior to caudad that is treated in 20 to 30 degrees of ipsilateral hip extension and fine tuning. The Longden points showed statistically significant improvement in RLS symptom severity on the International Restless Legs Scale total score at six weeks over controls, similar to the effect size found with dopamine agonist. While the CARL study is limited by a small sample size, the benefits compared to risks of the counterstrain procedure and the lack of other tolerable treatments increase the level of evidence using GRADE criteria making it a justifiable recommendation in the management of RLS when the tender points are present.[29] In addition, evidence suggests that targeted biweekly massage using myofascial release, trigger point and other soft tissue techniques to lower extremity with emphasis on piriformis and hamstrings muscles were found to diminish symptoms.[27] Overall, use of the biomechanical model may decrease pain and restore muscle function.

METABOLIC-ENERGY

Correction of nutritional deficiencies such as vitamin B12, iron, magnesium, and folate with attention to genetic and autoimmune disorders affecting absorption and metabolism are specific concerns in RLS.

ESSENTIAL TREMOR

Essential tremor is the most common movement disorder. Reported prevalence rates vary but may be as high as 5.6% of the United States population. Mild familial ET is often well olerated, and therefore may be untreated. An inheritable component may account for up to 70% of cases.[30] ET is characterized by a persistent bilateral 4-12 hz action and/or postural tremor typically found symmetrically in the upper extremities with less common involvement of the head, voice/larynx or lower extremities. At times, only one upper extremity is involved at the onset of the tremor with subsequent spread to the opposite extremity. Asymmetry of severity is not uncommon between limbs. Similarly, initial onset may be intermittent with later progression. ET is exacerbated by high adrenergic states including extremes of emotional experiences such as moving, employment changes,

TABLE 1:

Risk factors for developing PD and known causes of secondary PD

Category	Examples
Pharmacological "*Drug- induced parkinsonism*"	Classic and atypical antipsychotics Metoclopramide Prochlorperazine Reserpine
Toxins	Carbon disulfide Carbon monoxide Cyanide MPTP Aerosolized Manganese Organic solvents
Head Trauma	Isolated or repetitive injury (boxing)
Structural Brain lesions	Hydrocephalus Chronic subdural hematoma Tumor
Other Disorders	Wilson's disease Hypoparathyroidism Pseudohypoparathyroidism Chronic liver failure Extra-pontine myelinolysis Neurodegeneration with brain iron accumulation Neuroacanthocytosis
Infection	Encephalitis lethargica (Economo's encephalitis) HIV/AIDS Neurosyphilis Prion disease Progressive multifocal leukoencephalopathy Toxoplasmosis
Cerebrovascular Disease	Vascular parkinsonism

TABLE 3:

Known causes of secondary RLS

Category	Examples
Drug adverse event or withdrawal	**Psychiatric medications:** Anti-dopaminergic medications (antipsychotics/neuroleptics) Tricyclic antidepressants (TCAs) Selective serotonin reuptake inhibitors (SSRIs) Serotonin-norepinephrine reuptake inhibitors (SNRIs) Lithium **Other classes of drugs:** H1 blockers (e.g. Diphenhydramine) Alcohol (described with both abuse and withdrawal) Caffeine Beta blockers Opiates (Withdrawal)
Metabolic Conditions	Uremia Hyperphosphatemia
Vitamin and Mineral Deficiencies	Iron deficiency Folate or magnesium deficiency Vitamin B-12 deficiency
Neuropathy	Diabetic polyneuropathy Lumbosacral radiculopathy Lyme disease
Immune / Autoimmune Disturbances	MGUS (Monoclonal gammopathy of undetermined significance) Rheumatoid arthritis Sjögren syndrome Spinal multiple sclerosis

divorce, and death of a loved one, exercise, hunger and sleep disorders or deprivation. ET is relieved by ethanol consumption, which is part of the diagnostic criteria (Table 2). ET is most severe during the end of execution of fine-motor tasks, but can often be voluntarily suppressed with attention.

BEHAVIORAL

The first-line treatment recommendations for mild ET are conservative, starting with decreasing exacerbating factors. ET, while not necessarily triggered by stress states (unlike psychogenic tremor), can be exacerbated by even a little bit of stress. In fact, rates of social phobia among people with ET are higher than the general population.[31] Stress management techniques such as 90 minutes of exercise daily, psychotherapy, meditation, and relaxation therapies could be crucial. People with ET have been shown to have lower average SPO_2 during sleep.[32] Utilizing techniques to improve sleep (ex. opening up the airway in those with sleep apnea, increasing hours devoted to sleep or improving sleep hygiene in general) can decrease symptoms and improve overall wellbeing. Interventions that decrease the impact of common life stressors, improve sleep, and modify nutrition and exercise lifestyle should be offered upon diagnosis.

TABLE 2:

The key clinical diagnostic features of PD, RLS, and ET and their treatments. The diagnosis of PD, RLS, and ET are based on the history and physical exam. There are secondary causes of PD and RLS to diagnose and treat (Tables 1 & 3) in addition to the treatments outlined.

Disorder	Key Clinical Features [35]	Treatment [17,25,28]
Parkinson's Disease	1. Bradykinesia	Dopaminergic Medications Physical Therapy Occupational Therapy Speech Therapy Deep brain stimulation (DBS) Exercise
	2. Rigidity	
	3. Postural Instability- not caused by primary visual, vestibular, cerebellar, or proprioceptive dysfunction	
	4. Resting Tremor- may or may not be present	
Restless Leg Syndrome	1. An urge to move the legs, usually accompanied or caused by uncomfortable and unpleasant sensations in the legs	Sleep hygiene Regular exercise Counterstrain to L5 tender-points Dopaminergic drugs and anti-convulsants may provide short term symptomatic relief, but they move the onset of symptoms to earlier in the day over time. Sleep aids and sedatives may also improve sleep and thus patient quality of life.
	2. The urge to move or unpleasant sensations begin or worsen during periods of rest or inactivity such as lying or sitting	
	3. The urge to move or unpleasant sensations are partially or totally relieved by movement, at least as long as the activity continues	
	4. The urge to move or unpleasant sensations are worse in the evening or at night than during the day or only occur in the evening or at night	
Essential Tremor	1. Action tremor (i.e. worse during use of the muscle) 4-12 Hz (Faster than Intention tremor)	*1st line options:* Stress management Beta Blockers- *Propranolol Anticonvulsant- *Primidone *Severe/refractory:* Topiramate, clozapine, mirtazapine, benzodiazepines and botulinum toxin injection. *Surgical options:* Deep Brain Stimulation Thalamotomy
	2. Usually has a positive family history	
	3. Alleviated at rest	
	4. Alleviated with alcohol consumption	

NEUROLOGICAL

Patients may choose to take medications for temporary reprieve during times of greater stress. Small amounts of ethanol, primidone, or propranolol (a beta blocker and sympatholytic) may improve symptoms. Primidone and propranolol are effective in up to 70% of patients, but they lose efficacy when used regularly. Most studies suggest that one year is to be expected before disease progression or drug tolerance renders them ineffective. In severe cases where pharmacological management was ineffective or contraindicated, surgical interventions such as DBS or lesion therapy of the thalamus may be appropriate.

Exacerbations of moderate to severe ET management strategies could include OMM, which has previously been found to decrease biomarkers of stress.[15,33,34] OMM aimed at normalizing sympathetic tone could also potentially be helpful. Finally, tremor may cause soreness and change in resting tone in the muscles of the affected limbs. OMM may be helpful for managing such cases.

TABLE 4:

OMT techniques in specific regions for treatment of PD, RLS, and ET.[17,25,35-37] Each physician will select the treatment modalities best suited for each patient's specific complaints. The table is designed to help guide the practitioner in creating a treatment plan that is individualized to optimize treatment effect. ROM: range of motion of the joint

Body Region	OMT Technique	Goals of treatment
Head	Occipito-Atlanto (OA) release	Increase ROM Normalize vagal tone
	Compression of the 4th ventricle (CV-4)	Normalize autonomic tone
	Occipitomastoid spread (V-Spread)	Normalize vagal tone Reduce compression of glossopharyngeal & accessory nerves Improve cranial bone motion Reduce jugular vein compression
	Balance membranous tension (BMT)	Reduce strains Improve cranial motion
Anterior Neck	Hyoid & tracheal articulation	Improve swallowing
	Anterior counterstrain points	Improve neck ROM & posture
	Muscle energy to the sternocleidomastoid & scalene muscles	Reduce compression of neurovasculature
Spine	Muscle energy to the paraspinal muscles & quadratus lumborum	Increase spinal ROM Improve posture & ambulation
	Articulation: Seated & Prone	
	Active or passive myofascial stretch	
	Paraspinal inhibition	Normalize sympathetic tone
Thoracic Cage	Rib Raising: Seated or Supine	Normalize sympathetic tone Increase rib motion
	Thoracic inlet release	Improve lymphatic & blood circulation Improve thoracic cage excursion
	Sternal myofascial release	
	Thoracic diaphragm release/ Doming of the diaphragm	
	Pectoral Lift	
Extremities	Spencer's technique and other articulatory techniques to upper extremity joints	Increase ROM Improve ability to perform activities of daily living
	Muscle energy to upper extremities dysfunctions	
	Scapular balanced ligamentous tension	
	Muscle energy for lower extremities (Especially hamstrings, adductors, and psoas muscles)	Improve ambulation
	Circumduction / articulatory techniques to lower extremity joints	
Pelvis & Sacrum	Sacroiliac joint gapping	Improve ambulation, decrease pain
	Pelvic diaphragm release	Improve lymphatic & blood circulation Promote digestion
Other	Mesenteric lifts	Promote digestion
	Celiac & mesenteric ganglia inhibition	

CONCLUSION

The movement disorders, PD, RLS, and ET have a wide variety of debilitating motor and non-motor symptoms to assess during patient care. Integration of the five models of osteopathic care can improve symptom management and overall quality of life. The behavioral component appears to be a critical part of care for PD, RLS, and ET. Poor quality and quantity of sleep is an exacerbating factor in all three movement disorders. The neurological perspective involves individualization of both pharmaceutical and OMT interventions (Table 4) based on patient physical examination and specific complaints. In movement disorders, the biomechanical approach is largely intertwined with the neurological model. The normalization of ANS tone or afferent input through musculoskeletal manipulation would benefit these disorders. In PD and RLS, the metabolic-energy model is integral to long-term optimization of function and quality of life. The respiratory-circulatory model is also a major concern in PD. OMM may minimize the stress-related sequelae of the conditions or, in some cases, decrease symptom severity or frequency. OMM can potentially help with treating many of the common complaints and symptoms in patients with movement disorders.

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