Restless Legs Syndrome (RLS), Essential Tremor, Parkinson's Disease (PD), and Amyotrophic Lateral Sclerosis (ALS)

Following are descriptions of neurological movement disorders. Clinical Somatics exercises may be helpful for people with these conditions for two reasons:

1. Clinical Somatics exercises relax the nervous system, and in doing so they may reduce symptoms.

2. If people are pursuing exercise as part of their treatment/recovery, Clinical Somatics exercises will keep their muscles relaxed, improve their body use, and prevent injuries.

RESTLESS LEGS SYNDROME (RLS)

Restless legs syndrome (RLS; also called Willis-Ekbom Disease) is a condition that causes uncomfortable or unpleasant sensations in the legs, often described as creeping, crawling, itching, tingling, pulling, throbbing, or painful. People with RLS feel an irresistible urge to move their legs in order to relieve these sensations. For this reason, RLS can be classified as a movement disorder. It can also be classified as a sleep disorder because the symptoms are made worse by resting and trying to sleep. However, RLS is most appropriately classified as a neurological sensorimotor disorder, because the symptoms are produced within the brain.

RLS can have a significant negative effect on quality of life, mainly due to disrupted, shortened, and low-quality sleep. RLS symptoms typically occur in the evening and overnight; this is likely because natural melatonin production inhibits dopamine, causing symptoms to worsen at night when we produce melatonin. Lack of sleep can for many people lead to difficulty concentrating, impaired memory, poor performance at work and school, and mood disorders including depression and anxiety.

Restless legs syndrome is fairly common, occurring in between 3.9% and 15% of the world population. Symptoms of RLS may begin at any age, and tend to get worse with age. Women get RLS about twice as often as men, and researchers suggest that this could be due to childbearing and differences in hormones and social roles.

Across the board, scientists agree that more research needs to be done in order to fully understand RLS. So far, studies show that iron deficiency and dopaminergic (DA) system dysfunction are the two factors most often present in RLS sufferers, and the two are related.

How is RLS diagnosed?

There is currently no test for RLS, so the condition is diagnosed by a doctor's evaluation on the following five criteria:

- A strong or overwhelming urge to move the legs, often associated with uncomfortable sensations described as creeping, crawling, itching, tingling, pulling, throbbing, or painful
- Uncomfortable sensations and urge to move the legs begins or gets worse during rest or inactivity
- Moving relieves the uncomfortable sensations in the legs (at least partially or temporarily)
- Uncomfortable sensations and urge to move the legs begins or gets worse in the evening or at night
- The above features are not due to any other medical or behavioral condition

The following conditions can mimic and be misdiagnosed as RLS:

- leg cramps
- positional discomfort
- local leg injury
- arthritis
- leg edema
- venous stasis
- peripheral neuropathy
- radiculopathy (sciatica)
- habitual foot tapping/leg rocking
- anxiety
- myalgia
- drug-induced akathisia

Symptoms of RLS

It's common for symptoms to be experienced very mildly or intermittently at first, and to follow a gradual progression. Many years may pass before symptoms are experienced regularly. People most often feel symptoms in both legs, but they can occur on just one side or alternate sides. Some people develop symptoms in their arms as well, and in rare cases, the chest or head. More than 80% of people with RLS also experience periodic limb movement of sleep (PLMS), which involves involuntary leg and/or arm movements during sleep.

What causes RLS?

RLS can be related to or caused by the following factors and conditions:

- End-stage renal disease and hemodialysis
- Iron deficiency
- Vitamin D deficiency
- A condition involving inflammation or immune system changes, including Parkinson's disease, multiple sclerosis, ADHD, Alzheimer's disease, Celiac disease, Crohn's disease, rheumatoid arthritis, sleep apnea, diabetes, and depression
- Certain medications, such as antinausea drugs, antipsychotic drugs, antidepressants that increase serotonin, and some cold and allergy medications that contain antihistamines
- Use of alcohol, nicotine, and caffeine
- Pregnancy, especially in the last trimester (symptoms typically disappear within 4 weeks after delivery)
- Neuropathy (nerve damage)
- Sleep deprivation or sleep apnea

Iron deficiency and dopaminergic (DA) system dysfunction in restless legs syndrome

Research shows that people with RLS are often deficient in iron. Ultrasound, MRI, and autopsy studies show that a part of the brain called the substantia nigra in particular tends to have decreased levels of iron. Other parts of the brain including the putamen, thalamus, red nucleus, and pallidum have also been found to have lower than normal concentrations of iron in RLS patients.

Logically, iron supplementation has been tested and used as treatment for RLS. Some people's symptoms are improved or eliminated completely with iron supplementation, and others' are not. The blood-brain barrier poses a problem when it comes to iron; blood serum levels of iron (iron circulating throughout the body) and levels of iron in the brain are different. People can have normal or higher than normal blood iron levels, while their brain remains iron deficient. Research suggests that in RLS, brain iron uptake is dysregulated, leading to brain iron deficiency.

A 2011 in vitro study in rats found that iron deficiency can cause death of dopaminergic cells in the substantia nigra. When these dopamine-secreting neurons die, it leads to dysfunction of mesolimbic and nigrostriatal dopaminergic pathways. These pathways regulate movement as well as motivation and desire for reward. Researchers hypothesize that the dysfunction of these pathways in turn leads to dysregulation of limbic and sensorimotor networks, resulting in the symptoms of RLS.

Death of dopaminergic neurons in the substantia nigra and disruption of dopamine pathways also occurs in Parkinson's disease. And while rates of RLS are higher among Parkinson's

disease patients than controls and the two conditions have overlapping clinical symptoms, scientists believe that the underlying pathological processes that cause RLS versus RLS associated with Parkinson's disease are different. In RLS, iron deficiency causes death of dopamine-secreting neurons. But in Parkinson's disease, it has been found that *too much iron* in the substantia nigra causes oxidative stress, leading to the death of dopamine-secreting neurons.

Dopamine medications, which are largely used to treat Parkinson's disease, can prevent the breakdown of dopamine, mimic dopamine, or convert into dopamine when they reach the brain. These drugs can reduce the symptoms of RLS, and some have been approved to treat RLS. However, long-term use is typically not recommended because it can lead to worsening of symptoms. This occurs because over time, the brain adapts to the increased levels of dopamine or dopamine-like substances in the brain, and gradually makes less of its own dopamine. The initial dose can become less effective, and people may begin to experience symptoms earlier in the day or all day. Other side effects of dopamine medications include sleepiness and compulsive behavior. Taking into consideration the potential side effects, natural ways of stimulating dopamine production are far preferable for the long term.

Figuring out the cause and reducing or eliminating symptoms of RLS

If you have a student with RLS, you should recommend that they visit a doctor, preferably one that specializes in RLS, to get a blood test to find out the level of ferritin (a protein that stores iron) in their blood. Iron supplementation can reportedly relieve RLS symptoms in approximately 50% of people. They should also be tested for vitamin D deficiency, which is linked to RLS. Their doctor will also likely recommend that they eliminate caffeine, tobacco, and alcohol.

The following health conditions and lifestyle factors have also been linked to RLS; you can read more at: https://somaticmovementcenter.com/restless-legs-syndrome

- Immune system function and inflammation
- Small intestinal bacterial overgrowth (SIBO) and irritable bowel syndrome (IBS)
- Chronic stress
- Poor sleep habits and sleep disorders
- Lack of regular exercise

Yoga and meditation have been shown in studies to reduce symptoms of RLS. So while RLS is not caused by chronic muscle tension, stress may be a factor, and for this reason Clinical Somatics exercises may help to reduce symptoms. However, people with RLS should always visit a doctor, preferably one that specializes in RLS, to find out if they have an underlying health condition.

ESSENTIAL TREMOR

Essential tremor is a nervous system disorder that causes involuntary and rhythmic shaking. It most often affects the hands, but can affect almost any part of the body. Essential tremor is most common in people over 40, and it typically worsens over time. It can be confused with Parkinson's disease, so it is important to get an accurate diagnosis.

Essential tremor vs. Parkinson's disease

The two conditions differ in these important ways:

- Timing of tremors: Essential tremor of the hands usually occurs when the hands are being used, like when performing simple tasks. In contrast, tremors from Parkinson's disease are most often "resting tremors", which occur when the hands are not being used.

- Associated conditions: Essential tremor doesn't cause other health problems, but Parkinson's disease is associated with stooped posture, slow movement, and shuffling gait. However, people with essential tremor sometimes develop other neurological signs and symptoms, such as an unsteady gait (ataxia).

- Parts of body affected: Essential tremor mainly involves the hands, head, and voice. Parkinson's disease tremors may start in the hands, and can affect the legs, chin, and other parts of the body.

What causes essential tremor?

It is believed that about half of essential tremor cases are the result of a genetic mutation; this is referred to as familial tremor. For people without the genetic mutation, the cause is unknown.

How is essential tremor diagnosed?

There are no medical tests used to diagnose essential tremor. The doctor will review medical history, family history, symptoms, and conduct a physical exam. They will rule out other conditions that may be causing the symptoms by doing a neurological exam (to test sensation, reflexes, muscle function, posture, coordination, and gait) and lab tests to check for thyroid disease, metabolic problems, drug side effects, and abnormal levels of chemicals. The doctor may order a dopamine transporter scan, a test used to identify early signs of Parkinson's disease.

How is essential tremor treated?

If the tremor is mild, treatment may not be required. But if the tremor is making daily life difficult, medications including beta blockers, anti-seizure medications, tranquilizers, and Botox may be prescribed. If tremors are severe and medications don't help, deep brain

stimulation or focused ultrasound thalamotomy may be used.

Physical therapy may be recommended to improve muscle strength, control, and coordination. Occupational therapy may be recommended to help adapt daily activities.

Lifestyle changes that are recommended include:

- Avoid caffeine and other stimulants
- Avoid alcohol
- Reduce chronic stress
- Get regular, high-quality sleep

Clinical Somatics exercises may be helpful for people with essential tremor by reducing stress and improving muscular control and coordination. However, it is crucial that people with essential tremor be evaluated by a neurologist to determine whether or not they have Parkinson's disease.

PARKINSON'S DISEASE (PD)

Parkinson's disease (PD) is a degenerative neurological condition in which dopaminergic (dopamine-producing) neurons in a part of the brain called the substantia nigra die off. When there is not enough dopamine in the nigrostriatal pathway (one of the dopamine pathways), motor symptoms occur. Other parts of the brain suffer neurodegeneration as well, causing some of the non-motor symptoms of Parkinson's.

Parkinson's disease typically gets diagnosed when motor symptoms appear. The average age of diagnosis is age 60. When Parkinson's is diagnosed after age 50, it is called late-onset; diagnosis before age 50 is called early-onset or young-onset. Diagnosis before age 20 is referred to as juvenile-onset. The younger someone is at age of diagnosis, the more likely it is that genetics play a role in their disease.

A defining aspect of Parkinson's disease is Lewy bodies, which are misfolded clumps of the protein alpha-synuclein. Lewy bodies develop in the substantia nigra and other brain areas, as well as the enteric nervous system (ENS; neurons that control the gastrointestinal tract), and cause neuron death. Lewy bodies are present in almost all cases of Parkinson's disease. Typically when symptoms are present without Lewy bodies, it is referred to as parkinsonism. Parkinsonism can be the result of other neurological diseases, medications, and infections.

What are the symptoms of Parkinson's disease?

Motor symptoms of Parkinson's disease include:

- Resting tremor (shaking or trembling when at rest)
- Rigidity or stiffness

- Slow movement (bradykinesia) or the inability to move
- Shuffling gait
- Postural instability
- Impaired balance and coordination
- Dystonia (painful muscle cramps)
- Stooped posture
- Impaired fine motor dexterity and motor coordination
- Impaired gross motor coordination
- Decreased arm swing
- Akathisia (tendency to keep moving)
- Speech problems
- Difficulty swallowing
- Sexual dysfunction

Non-motor symptoms include disturbed sleep, anxiety, depression, pain, cognition difficulties, visual hallucinations, and dementia.

Unfortunately, motor symptoms typically appear years after the disease process has begun—often 20 or more years after. It's estimated that motor symptoms appear when approximately 30% to 60% of dopaminergic neurons in the substantia nigra are lost. When dopamine levels decrease to a certain critical threshold, tremor or other motor symptoms may be felt, sometimes suddenly. For many people, this is the first noticeable sign of the disease.

Once diagnosed, people are often able to recognize symptoms that occurred years earlier. Decades before motor symptoms begin, people may experience loss of sense of smell and constipation. These occur because the disease attacks the medulla (a part of the brain stem), the olfactory bulb (brain area responsible for sense of smell), and the enteric nervous system (ENS; neurons that control the gastrointestinal tract) early on.

Next, depression and rapid eye movement sleep behavior disorder may occur as the disease progresses to the pontine tegmentum (a part of the brain stem) and other areas. When the disease progresses to the substantia nigra and does sufficient damage, motor symptoms occur. Dementia and hallucinations may occur in the latest stages of the disease, as neural damage spreads to the neocortex.

What causes Parkinson's disease?

It is agreed in the scientific community that Parkinson's is most often the result of a complex interplay between genetic and environmental factors—this is what has made it so difficult to define, treat, and prevent. Having one risk factor is often not enough to trigger the condition, but having two or more increases the odds substantially. Scientists suggest that Parkinson's is not one disease but many, with different contributing factors.

The known risk factors for developing Parkinson's disease are:

- Genetics: It's estimated that between 10% and 15% of people with Parkinson's disease have a genetic predisposition. In most cases, this does not mean that they will definitely get the disease. Risk goes up significantly when other risk factors are present.
- Exposure to neurotoxic chemicals including pesticides, insecticides, herbicides, and solvents.
- Head trauma, especially repeated head trauma as experienced by members of the military, football players, and boxers.
- Lack of exercise
- A diet high in animal products
- Gut dysbiosis
- Chronic stress

To learn more about these risk factors, please read this post:
https://somaticmovementcenter.com/parkinsons-disease

How is Parkinson's disease treated?

There is no cure for Parkinson's disease. Medications are prescribed to manage symptoms. Levodopa, the most effective medication, passes into the brain and is converted to dopamine. Medications called dopamine agonists mimic the effects of dopamine in the brain. Other medications may be prescribed to prevent the breakdown of dopamine. Deep brain stimulation (DBS) may be used in some cases of advanced Parkinson's disease. While medications and DBS provide symptom relief, they do not prevent Parkinson's disease from progressing.

Scientists are currently exploring ways to enhance the natural process of producing neural stem cells (neurogenesis) to replace the dopaminergic neurons that are lost in Parkinson's disease. While these stem cell therapies are years away from being approved for use in humans, they offer promise. However, the newly formed dopaminergic neurons would likely be damaged by whatever caused Parkinson's in the first place; so these treatments will likely buy patients important time, but may need to be repeated.

Reversing Parkinson's symptoms with exercise and stress reduction

It is extremely important for Parkinson's patients to understand how to enhance neurogenesis and boost dopamine naturally. Exercise and stress reduction are two of the best ways to encourage neural stem cells to survive, migrate, and differentiate into the type of neurons that the brain needs. Parkinson's patients are already using exercise and stress reduction to boost their dopamine levels and restore dopaminergic neurons, and the result is a reduction or, in some cases, complete elimination of motor symptoms.

While all types of exercise can benefit people with Parkinson's, there is extremely promising research being carried out by researchers at the Cleveland Clinic in Ohio. They have discovered

that forced exercise, in which the patient is forced to exercise at a faster rate than they would choose to on their own, results in significant and lasting improvement of motor symptoms. Brain scans show that forced exercise and medication activate the same brain areas. The researchers explain: “Both levodopa therapy and forced exercise are thought to increase the amount of available dopamine within the dorsolateral striatum.” In addition, exercise stimulates neurogenesis, leading to gradual repair of the damaged brain. Study participants have even experienced their lost sense of smell returning. The research is conclusive enough that it will likely lead to neurologists prescribing forced exercise to Parkinson's patients in the future.

Researchers note that there is currently no pharmacological treatment that can modify or slow Parkinson's disease or protect dopaminergic neurons. For Parkinson's patients, exercise does what medication can't.

There has been less research on the positive effects of stress reduction on Parkinson's symptoms, but it is an emerging area of research. It is becoming more widely accepted that chronic stress causes neuroinflammation, oxidative stress, and elevated cortisol levels, which lead to a loss of dopaminergic neurons in the substantia nigra. Chronic stress also decreases neurogenesis. Stressful life events, job-related stress, and stress-related personality traits all increase the risk of developing Parkinson's. Stress level also worsens motor symptoms and predicts worse disease progression.

Research shows that eight weeks of a mindfulness practice leads to increased gray matter density (number of neurons) in neural networks that play a role in Parkinson's disease. In a separate study, the same researchers found that an eight-week mindfulness program led to a significant improvement in motor symptoms in Parkinson's patients.

Other research has found that relaxation guided imagery dramatically decreases tremor in Parkinson's patients. Patients reported improvements lasting 2-14 hours afterward. Studies of meditation have shown significant increases in the release of dopamine during meditation.

The most compelling argument for the effects of stress reduction on Parkinson's symptoms are the stories of people who have eliminated their Parkinson's symptoms using meditation, mindful movement, Tai Chi, Qigong, and positive thinking.

To learn more about how forced exercise and stress reduction can reverse Parkinson's symptoms, please read this post:

https://somaticmovementcenter.com/recovery-from-parkinsons

Clinical Somatics may be helpful for Parkinson's patients because:

- Clinical Somatics exercises relax the nervous system, and in doing so they may reduce symptoms.
- If someone with Parkinson's is pursuing exercise as part of their treatment/recovery, Clinical Somatics exercises will keep their muscles relaxed, improve their body use, and prevent injuries.

AMYOTROPHIC LATERAL SCLEROSIS (ALS)

Amyotrophic lateral sclerosis (ALS) is a progressive neurodegenerative disease that affects motor neurons in the brain and the spinal cord. Motor neurons extend from the brain to the spinal cord and from the spinal cord to the muscles throughout the body. In ALS, motor neurons progressively degenerate and then die. When motor neurons die, the brain cannot initiate and control voluntary movement. As a result, people with ALS can lose the ability to speak, eat, move, and breathe.

Most people with ALS die within two to five years after diagnosis. Twenty percent live five years or more after diagnosis, and up to 10% live more than 10 years after diagnosis. There are rare cases of reversals in which motor function improves significantly.

What are the symptoms of ALS?

Symptoms of ALS can vary greatly from person to person, depending on which neurons are affected. Signs and symptoms include:

•Difficulty walking or doing normal daily activities
•Tripping and falling
•Weakness in the legs, feet, or ankles
•Hand weakness or clumsiness
•Slurred speech or trouble swallowing
•Muscle cramps and twitching in the arms, shoulders, and tongue
•Inappropriate crying, laughing, or yawning
•Cognitive and behavioral changes

ALS often starts in the hands, feet, or limbs, and then spreads to other parts of the body. As the disease advances and nerve cells are destroyed, muscles get weaker. This eventually affects chewing, swallowing, speaking and breathing. Pain is not a common symptom.

What causes ALS?

Between 5% and 10% of ALS cases are familial, meaning it is inherited. The other 90% to 95% of cases are sporadic, meaning that the cause is unknown. Known risk factors for sporadic ALS include:

- Smoking
- Exposure to neurotoxic chemicals
- Military service (possibly as a result of chemical exposure, traumatic injuries, viral infections, or intense exertion)

Other risk factors that are being researched include:

- Low intake of antioxidants
- Intense physical exertion
- Traumatic head injury
- Exposure to chemicals, pesticides, the neurotoxin BMAA, metals, and electromagnetic fields
- Viral infection
- Type 1 diabetes

How is ALS treated?

There is no cure for ALS, but treatments can slow the progression of symptoms, prevent complications, and maintain quality of life. FDA approved medications can increase life expectancy, reduce the decline in daily functioning, and alleviate other symptoms. Doctors may also prescribe medications that relieve symptoms like muscle cramps and spasms, constipation, fatigue, depression, sleep problems, and more.

Other treatments and therapies that may be recommended are:

- A ventilator to assist with breathing, especially at night
- Physical therapy to improve walking, mobility, cardiovascular fitness, and muscle strength
- Regular exercise to maintain fitness and improve sense of well-being
- Stretching to prevent pain and keep muscles loose
- Occupational therapy and adaptive equipment to aide in maintaining independence
- Speech therapy to make speech more understandable, and to explore other methods of communication
- Dietary changes in order to ensure proper nutrition while eating foods that are easy to swallow
- Psychological and social support

Since physical therapy, exercise, and stretching are all recommended for ALS, it is safe to say that Clinical Somatics exercises may be helpful in keeping muscles relaxed and improving muscular control.

Using lifestyle changes to manage and reverse ALS

When symptoms of ALS improve, it is referred to as "ALS reversal." Within the ALS community, similar to the Parkinson's community, there are people focused on using lifestyle changes and alternative medicine to reverse ALS symptoms. However, much less research has been done on the effects of lifestyle interventions for ALS, and the information available is somewhat inconsistent.

There are people who reverse their symptoms and others who stabilize their condition and live long, active lives. If you have a student with ALS, these links may give them some hope and direction:

https://healingals.org/
https://alsadotorg.wordpress.com/2016/07/06/als-reversals-what-are-they-and-how-can-we-make-them-happen-more-often
https://www.alswinners.com/
http://www.awarenessmag.com/mayjun5/mj5_als.html

A promising study (https://www.ncbi.nlm.nih.gov/pubmed/16584562) showed the beneficial effects of a ketogenic diet for ALS. In ALS, motor neurons die because there is failure of energy production for the neurons. A ketogenic diet burns fat instead of carbohydrates, and is potentially effective in protecting nerve cells and preserving their ability to make energy. A ketogenic diet showed a substantial benefit in the treatment of ALS in an animal model.

Many people with ALS and health professionals who treat ALS promote other dietary changes including eating lots of fruits and vegetables (for the fiber and antioxidants) and taking supplements. Since ALS results from lack of energy for motor neurons, a proper diet makes sense. There also may be an autoimmune component to ALS, in which case dietary changes also make sense.

While stressful life events have not been shown in clinical studies to increase the risk of ALS, there are doctors who believe that internalizing chronic stress is a factor in the condition. ALS sufferers are often described as being workaholics, over-achievers, and having a compulsive sense of duty to others. They also tend to not complain or express anger—both of which are signs of suppressing stress and emotions. Dr. Gabor Maté, author of *When the Body Says No*, describes a phenomenon that has long been observed by doctors who treat patients with ALS: the patients are almost always extremely nice people. They are pleasant and easy to deal with, don't ask for help and never complain. Neurologists from the Cleveland Clinic presented a paper called "Why Are Patients with ALS So Nice?" at an international symposium. When technicians have completed their tests on people who may have ALS and don't know the results yet, they often include a comment like "This patient cannot have ALS, he or she is not nice enough" and almost invariably, they are correct.

Much remains to be learned about ALS, but if you have a student with the condition, encourage them to do their own research and know that they should not accept the death sentence that their doctor will likely give them—it is very possible for them to live a long, active life.