

# Integration of Osteopathic Manual Treatments in the Management of Foot Dystonia in Parkinson's Disease: A Case Series

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## Abstract

Parkinson's disease and focal limb dystonia are neurological movement disorders that may occur co-morbidly, often leading to more severe gait impairment and pain. Treatment options for foot dystonia in Parkinson's disease are limited and have variable outcomes. Osteopathic manual treatment techniques were previously found to improve gait in Parkinson's disease. Here, we present three cases in which osteopathic manual treatment was used to treat foot dystonia in patients with Parkinson's disease. The most prominent features of the foot dystonia were cramping and pain in the foot and ankle radiating up to the ipsilateral torso and involuntary foot inversion. The common musculoskeletal dysfunctions found were spasm of ipsilateral hip adductor muscles, psoas, gastrocnemius, and one or more intrinsic ankle and foot muscles as well as calcaneal inversion and restrictions to motion of the tarsal bones. All patients reported improvement in pain and mobility following treatment that lasted five to seven days. Future research is necessary to define and test the effectiveness of a specific treatment protocol in the long term management.

**Keywords:** Parkinson disease; Dystonia; Manual therapy; Muscle spasm

## Introduction

Parkinson's disease (PD) and focal limb dystonia are neurological movement disorders. Approximately 1 million people in the United States and over 7 million globally have PD [1]. Dystonia is a rare disorder, however, PD may present with limb dystonia of the lower leg [2]. PD occurs with a progressive degeneration of dopaminergic neurons in the basal ganglia that clinically manifests as motor signs of tremor, bradykinesia, and rigidity [3]. The underlying pathology of limb dystonia appears to involve a predisposition of genetic nature or a loss of inhibitory processes in the nervous system with subsequent environmental factors of repetitive activity and trauma [2]. The clinical signs include involuntary sustained or intermittent muscle contractions leading to abnormal postures and/or repetitive movements [2]. Both disorders impair gait.

Treatment for PD primarily includes oral medications and exercise that typically improve symptoms and may slow disease progression. Treatment for lower limb dystonia may involve botulinum toxin injections into the hypertonic muscle(s) [2]. Clinical improvement with botulinum injections is variable due to challenges in determining the most effective muscle(s) to inject without causing excessive weakness [2]. Few patients have found remarkable benefits with oral drugs. Limb dystonia without other neurological disorders typically responds well to neurosurgery [2]. There has been very little research on treatment of combined PD-limb dystonia. One potential treatment is osteopathic manual treatment (OMT).

OMT is a manual therapy utilized to treat illness and injury wherein clinical examination of posture, balance, mobility of body regions, and behavior lead to the diagnosis of somatic dysfunctions underlying clinical illnesses. The goal of OMT is to improve the physiology of structures such as muscle tone and connective tissue distortion that inhibit function or homeostasis. The use of OMT in PD is being increasingly studied, and it has been found to improve stride length, cadence, and upper and lower limb velocity in a pilot study [1]. OMT has also been used to significantly improve balance in the elderly [1]. The treatment can also be used for other ailments such as migraines [4]. OMT has been shown in multiple studies to be as effective as effective as a complement, or even an alternative, to drugs or surgery for symptom relief of various conditions. Here, we present cases of PD with foot

dystonia in which OMT was used with a goal of improving mobility and pain.

## Methods

The following cases are examples of the use of OMT to improve symptoms associated with foot dystonia in patients with PD.

### Case 1

M.S. is a 64 year old male with a history of PD for over 10 years and right foot dystonia that worsened after deep brain stimulation neurosurgery for PD symptoms. While his PD symptoms greatly improved with neurosurgery, his foot dystonia became much more severe with intermittent involuntary ankle inversions causing him to rely on his walker at all times to avoid falling. He had a botulinum toxin injection, which initially improved the intensity of the inverting. However, he developed a deep vein thrombosis in his right calve within weeks of the injection and was no longer a low risk candidate for the procedure. Along with poor balance, M.S. complains of 8/10 pain radiating into his back that is worse with weight-bearing and right foot movements interrupting his sleep.

His past medical history also included mitral valve prolapse in his 20s, nephrolithiasis, controlled hypertension, lumbar stenosis and radiculopathy, bilateral hand osteoarthritis, and obstructive and/or central sleep apnea. He was also in a severe motor vehicle accident prior to developing PD and dystonia. His family history included Alzheimer's disease in his mother. He had a history of quitting smoking tobacco in his 40 s and recovering from alcoholism. He consumed 2 cups of coffee a day and did not exercise except for physical therapy. Before becoming

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disabled, M.S. had owned a bar and was a professional race car driver. He lived with his female significant other. In addition, he had autonomic dysregulations associated with PD including urinary frequency and constipation. Medications included carbidopa-levodopa, carbidopa, entacapone, toseamide, clonazepam, oxycodone/acetaminophen, gabapentin and aspirin.

On physical exam, vital signs were within normal limits. In general, he was alert and oriented times three, in mild distress, but hopeful and desiring to do more of the activities he enjoys such as fishing. On neurological exam, muscle strength was normal except for in his right leg where hip abduction was 4/5. His right ankle was inverted and intermittently oscillating into further inversion. Straight leg raise was positive on the left. He exhibited mild resting tremor in his left arm. Muscle tone was increased globally, particularly in his right leg. His head was tilted to the right both supine and standing.

The somatic dysfunctions found on osteopathic structural exam were as follows. Severe somatic dysfunctions of the head were occiput extended, side-bent right and rotated left on C1, sphenobasilar synchondrosis compression and right torsional strain, right mastooccipital suture restriction, and left temporal bone internal rotation, which were treated with osteopathy in the cranial field and myofascial release. Severe somatic dysfunctions of the neck were C2 flexed, rotated and side-bent left, right scalene spasm, paravertebral muscle spasm on the right, hypertonicity of the right sternocleidomastoid and levator scapula muscles. These were treated with balanced ligamentous tension, myofascial release, and muscle energy. Severe somatic dysfunctions of the thoracic spine were T1 flexed, rotated and side-bent right, T3 extended, rotated and side-bent right, T8- T11 neutral, side-bent right and rotated left, which were treated with myofascial release, muscle energy, and balanced ligamentous tension. Both 12<sup>th</sup> ribs had inhalation somatic dysfunction treated with respiratory muscle energy. Severe somatic dysfunctions of the lumbar region were L5 extended, side-bent and rotated right, spasm of right psoas, quadratus lumborum, and paraspinal muscles, which were treated with myofascial release, muscle energy, and balanced ligamentous tension. Initially there was a unilateral sacral shear dysfunction, which was treated with balanced ligamentous tension. On reassessment, there was a left on right backward sacral torsion, which was treated with muscle energy. There was a right innominate superior shear that was treated with respiratory-assisted muscle energy. On reassessment, there was a right anterior rotation somatic dysfunction, which was treated with balanced ligamentous tension and muscle energy. The muscles of the pelvic floor (pelvic diaphragm) were in spasm on the right, and these were treated with inhibitory pressure applied medial to the ischial tuberosity, directed superiorly. Severe somatic dysfunctions of the right lower extremity were spasm of gluteus medius, gluteus maximus, hip adductors, semimembranosus and semitendinosus. There was hypertonicity of iliotibial band, gastrocnemius, posterior fibula head dysfunction, calcaneal inversion, forefoot supination with restricted talus, navicular, and cuboid mobility, which were treated with balanced ligamentous tension, myofascial release, muscle energy, and Still's technique.

All somatic dysfunctions improved with OMT. M.S. reported that he had improvement in his right ankle and foot symptoms including a reduction of pain to 4/10. With further treatments, M.S. stated that he experienced severely worsened pain, more difficulty walking, worse dystonia, and more frequent falls when he did not have weekly OMT.

## Case 2

E.M. is a 75 year old female with PD complaining of 9/10 constant,

right ankle and foot cramping and sharp pain intermittently radiating to her right buttock. The pain started a few days ago after she walked in flip flops because her right shoes would not fit comfortably. She had steroid injections after the foot pain started, but the cramping and pain persisted. This pain limited her ability to walk around her home. The pain improves temporarily with ice and elevation.

E.M. has a past medical history of osteoporosis, glaucoma, depression, osteoarthritis, spinal stenosis, degenerative disk disease, chronic right knee pain, and urinary incontinence. Her surgical history was hysterectomy secondary fibroids, one abortion, a lipoma removal, and meniscal repair in her right knee. Her medications included Estradiol, Vitamin D, Calcium, Magnesium oxide, and ibuprofen. She did not have a good response to levodopa, nor too many other medications. The patient has never smoked and her exercise includes physical therapy. The patient resides in a private home with her husband. Her father was deceased due to an abdominal aortic aneurysm rupture, and her mother was deceased at 99 years old for unclear reasons. The patient's siblings are alive and she has 3 healthy daughters. Her review of systems was remarkable for PD symptoms of stiffness, resting tremor, freezing, and, more recently, poor balance.

On physical exam, vital signs were normal. In general, she was alert and oriented times three and in mild distress due to pain and frustration. On neurological exam, E.M. had decreased blink, masked faces, right shoulder lag and bradykinesia on the right more than the left. Cranial nerves, motor strength, and sensory exam were otherwise normal. Reflexes were +2/4 in the lower extremity bilaterally and symmetrical. There were no cerebellar signs. Her gait was impaired by PD and right foot dystonia. She walked with a cane in a stooped posture and turned en bloc.

The somatic dysfunctions found on osteopathic structural exam were as follows. Severe somatic dysfunctions of the head were occiput extended side-bent right and rotated left on C1 as well as right condylar compression, which were treated with osteopathy in the cranial field. Severe somatic dysfunctions of the neck were right scalene spasm, C2 flexed, side-bent and rotated right, C5 flexed, side-bent and rotated right and C7 extended, side-bent and rotated right, which was treated with facilitated positional release and articulatory technique.

There was an elevated right 1st rib treated with articulatory technique and sternal restrictions treated with balanced ligamentous tension. Severe somatic dysfunctions of the thoracic spine included T1 flexed, side-bent and rotated right, T3-4 extended, side-bent and rotated left, T6 flexed, side-bent and rotated right, T12 flexed, side-bent and rotated right, which were treated with facilitated positional release, muscle energy, and myofascial release. There was inhalation dysfunction of the bilateral 12<sup>th</sup> ribs treated with respiratory assisted muscle energy. In the lumbar region, there was paravertebral muscle spasm on right, hypertonicity of the psoas muscles bilaterally, L1 flexed, side-bent and rotated right and L4 extended, side-bent and rotated right, which were treated with myofascial release and balanced ligamentous tension. There was also an imbalance in the tension of the iliolumbar ligaments treated with balanced ligamentous tension. Initially there was a unilateral sacral flexion shear dysfunction, which was treated with balanced ligamentous tension. On reassessment, there was a left on right backward sacral torsion, which was treated with muscle energy. There was a right innominate superior shear that was treated with respiratory-assisted muscle energy. On reassessment, there was a right anterior rotation somatic dysfunction, which was treated with balanced ligamentous tension and muscle energy. The muscles of the pelvic floor were in spasm on the right, and these were treated with

inhibitory pressure. There was left on right backward sacral torsion that was treated with balanced ligamentous tension and myofascial release. There was a right anterior innominate rotation with inferior pubic shear dysfunction treated with balanced ligamentous tension and spasm of the right pelvic floor muscles treated with inhibitory pressure. Somatic dysfunctions of the right lower extremity included hypertonicity of the right piriformis, quadriceps, gastrocnemius, popliteus, tibialis posterior, and hamstring muscles as well as right foot plantar flexion, calcaneal inversion, navicular external rotation, and posterior fibula head dysfunctions. In addition, there was a dropped 2<sup>nd</sup> metatarsal phalangeal joint and lateral patellar tracking. Dysfunctions of the right lower extremity were treated with fascial unwinding, balanced ligamentous tension, myofascial release, and muscle energy.

E.M. tolerated the techniques used and reported a decrease in pain to 6/10 and improved the comfort of her foot in her regular shoes.

### Case 3

R.T. is a 67 year old male with a 9 year history of PD and recent diagnosis of left foot dystonia presented with constant severe 10/10 left foot and ankle pain with intermittent cramping radiating to his stomach causing him nausea for 4 months. He occasionally experiences tingling and numbness in his left foot. He started physical therapy for his left foot, but his home health aide reports that he refuses to try his prescribed exercises at home due to pain.

His past medical history includes gastroesophageal reflux disease, controlled hypertension, coronary artery disease, prostate cancer, constipation, hypercholesterolemia, and a severe concussion at age 43 years. Surgical history was a radical prostatectomy at age 55 years and two coronary artery stents. His medications were carbidopa/levodopa, rasagiline, aspirin, clopidogrel, clonazepam, metoprolol, valsartan, carbidopa, furosemide, rosuvastatin, solifenacin, vilazodone, amlodipine, ranitidine, amphetamine/dextroamphetamine and dextlansoprazole. R.T. was working as a law professor. He was living with his girlfriend and his 19 year old son in a two story home. He reported not being able to walk independently at home since the dystonia started. He never smoked tobacco and he did not drink alcohol. He drank one cup of coffee per day. Family history was non-contributory. Review of systems was otherwise negative.

On physical exam, vital signs were normal. In general, R.T. was alert and oriented times three and in moderate distress due to pain and disability. He had some mild, non-pitting edema without any joint effusion present around the left ankle and dorsum of the foot. Hypertonicity of the intrinsic foot muscles and plantar contracture with a pes cavus shape was noted. His back had decreased cervical lordosis, markedly increased thoracic kyphosis and decreased lumbar lordosis. Seborrheic dermatitis was present on the patient's face and there was a shoulder lag on the left side as well as a decreased nasolabial fold on the left. On neurological exam, motor strength testing presented significant rigidity. There was reduced sensation in the feet and decreased hair follicles and texture to skin of distal bilateral lower extremities up to approximately 4-5 inches above the ankles. He ambulated slowly and stiffly with arms held by his side. He did not exhibit tremor during the exam.

The somatic dysfunctions found on osteopathic structural exam were as follows. Severe somatic dysfunctions of the head include

Sphenobasilar synchondrosis compression, compression of the occiput on C1, mastooccipital suture restriction, occipital intraosseous strain with deformity, a left tentorium cerebelli strain involving temporal bone internal rotation dysfunction and C2, which were treated with myofascial release and osteopathy in the cranial field. Severe somatic dysfunctions of the neck included C2 flexed, rotated and side-bent right, spasm of left scalene and sternocleidomastoid muscles, and bilateral paravertebral muscle hypertonicity, which were treated with myofascial release and balanced ligamentous tension. Severe somatic dysfunctions of the thoracic spine were T10 flexed, rotated and side-bent left, T12 flexed, rotated and side-bent right, which were treated with Still's technique. There was a left unilateral sacral shear that was treated with balanced ligamentous tension and myofascial release. Initially there was a left superior innominate shear that was treated with respiratory assisted muscle energy. On reassessment, there was a left anterior innominate rotation dysfunction that was treated with muscle energy. Spasm of the left pelvic floor muscles was treated with inhibitory pressure. Severe somatic dysfunctions of the left lower extremity included hypertonicity of the psoas, gastrocnemius, soleus, hamstring muscles, hip adductor muscles, popliteus, posterior tibialis and flexor hallucis brevis. There were posterior fibula head, calcaneal inversion, forefoot pronation and talo-tibial dorsiflexion somatic dysfunctions as well as restricted motion of the navicular and cuboid. These dysfunctions were treated with myofascial release, muscle energy and balanced ligamentous tension.

R.T. reported a decrease in left foot and ankle pain as well as the associated nausea with OMT. Without weekly OMT, R.T. had "excruciating" foot cramps that he felt also exacerbated his depression.

### Conclusion

OMT may be used safely as a complimentary treatment for foot dystonia patients with PD. The most prominent features of the foot dystonia among these cases were cramping and severe pain in the foot and ankle radiating up to the ipsilateral torso and involuntary foot inversion. The pain and decreased mobility was distressing in each case. The common musculoskeletal dysfunctions found were spasm of ipsilateral hip adductor muscles, psoas, gastrocnemius, and one or more intrinsic ankle and foot muscles as well as calcaneal inversion and restrictions to motion of the tarsal bones. In addition the patients had severe sacral and pelvic dysfunctions. All patients reported improvement in pain and mobility following treatment that lasted five to seven days, leading them to pursue weekly treatments. Future research is necessary to define a specific OMT protocol and test its effectiveness in the long term management of foot dystonia in PD utilizing a randomized, control trial.

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