Exhibit 528

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Expert Witness Report: Gary McElhiney

I. Table of Contents

Personal Background and Qualifications
Summary of Opinions
Methodology
- Medical record review
- Deposition review
- Medical literature review
- General causation reports review
- Telemedicine IME
- Additional Records

Discussion of Opinions
- Diagnostic differential etiology
- Causal relationship

Diagnostic and Causation Conclusion
Prognosis and Impact on quality of life
Reference Materials
Publications (last 10 years)
Testimony (last 4 years)
Rate sheet
CV (Attached)

II. Personal Background/Qualifications (change this entire section to 1st person)

I have been a practicing neurologist for over 30 years.

After completing a PhD in Neuroscience in the Department of Pathology at Northwestern University Medical Center, I entered medical school at Albert Einstein College of Medicine, completing my MD degree in 1989. I then pursued Neurology, with a residency at Columbia University in New York City where I was elected chief resident and completed my residency in 1993. Upon completion of my residency, I accepted a position as an Assistant Professor of Neurology at the University of Rochester. I have remained at the University of Rochester, working up through the academic ranks to become a full Professor in 2008. I became board certified by the American Board of Psychiatry and Neurology in 1994 and have remained board certified since.

I have remained clinically active over the last 31 years, practicing general neurology as well as subspecialty practices in peripheral nerve disorders and movement disorders. I

have practiced in an academic setting (University of Rochester), state setting (Rochester Psychiatric Center), and community settings (Rochester General Hospital) throughout my 31 years of practice, since joining the faculty at the University of Rochester. As such, I have broad experience in general neurology and movement disorders in particular.

During my tenure in Rochester, I have held several leadership positions including Director of the Botulinum Toxin Clinic, Chief of Neurology at Rochester General Hospital, and Chief of Physical Medicine and Rehabilitation, also at Rochester General Hospital. I have been an attending in the movement disorders division since 2001 and from 2012 through 2020 held the position of Chief of the Movement Disorders Division in the Department of Neurology at the University of Rochester.

I have published abstracts and articles in peer reviewed journals on topics including viral effects on the nervous system, peripheral neuropathies, botulinum toxin use, dystonia, and Parkinson's disease. Additionally, I have been active in clinical research of these same disorders, participating in over 35 funded projects over the years. I have also given multiple lectures on these same topics.

Other academic activities of note include participation in and member of the American Academy of Neurology Guideline Development Subcommittee (6 years) and Classifications of Evidence Committee (11 years). Since 2014, I have been the neurology section editor for an online resource tool for clinicians.

I have served as an expert witness in numerous medicolegal cases since 1996.

My experience positions me well to offer a clinical assessment, critically review the available literature, and render an expert opinion. This is supported by the International Parkinson's and Movement Disorder Society ("MDS") A 2023 MDS position paper on the diagnosis of Parkinson's disease notes that "criteria have been validated against the gold standard of expert clinical diagnosis (neurologists with > ten years' experience in PD diagnosis)." (https://www.movementdisorders.org/MDS/News/Newsroom/Position-Papers/MDS-Position-Diagnosis-of-PD.htm?FB_Values=&&&)

III. Summary of Opinions

After reviewing the medical records, relevant deposition testimony, general causation reports related to exposure and Parkinson's disease, a medical literature review, conducting a zoom interview, and in conjunction with my personal neurologic knowledge and experience, I offer the following core opinions pertaining to Mr. Gary McElhiney:

1. Mr. Gary McElhiney suffers from Parkinson's disease. This opinion is based on a review of his medical records, review of depositions, my review of the medical literature, my zoom interview of Mr. McElhiney, and my personal neurologic

knowledge and experience. This opinion is stated to a reasonable degree of medical certainty. The basis and support for this opinion is set out in detail below.

2. It is at least as likely as not that there is a causal relationship between Mr. McElhiney's Parkinson's disease and his exposure to the contaminated water at Camp Lejeune (including TCE and PCE). His exposure to known neurotoxic chemical such as TCE is at least as likely as not the cause of his Parkinson's disease, as set out generally in the 2017 ATSDR Assessment of Evidence that shows the exposure to TCE at Camp Lejeune is sufficient to reach equipoise or above. My opinion has considered and ruled out other potential causes of Parkinson's disease, other than Mr. McElhiney's exposure to the contaminated water at Camp Lejeune, as substantial contributors to the cause of his Parkinson's disease.

3. Parkinson's disease is a chronically progressive disease without any cure. Mr. McElhiney's Parkinson's disease will continue to progress and worsen with time and will continue to significantly impair his quality of life, most likely removing his ability to care for himself and ultimately confining him to a wheelchair.

Each of the opinions offered in this report are offered to a reasonable degree of medical certainty.

IV. Methodology:

In coming to my opinions, I conducted a differential diagnosis that is the standard methodology for diagnosing a disease/injury and assessing causation. A standard differential diagnosis requires a neurologist to review the relevant records and assess all of the available evidence to diagnose the disease and then consider all relevant potential causes. As part of the review, I evaluated all relevant potential causes for Mr. McElhiney's Parkinson's disease and then, based on the strength of the evidence, excluded other potential causes to determine the most likely cause or causes for his Parkinson's disease. In the present case, the exposure to the contaminated water at Camp Lejeune remained the only cause within my differential etiology. Mr. McElhiney's exposure to the contaminated water at Camp Lejeune was substantial:

Exposure dates: first month June, 1972; last month October, 1988
Estimated total months of exposure 99 months from living or working at
Hadnot Point, Mainside Barracks, and Tarawa Terrace

The substantial exposure to the contaminated water at Camp Lejeune combined with the absence of any other significant and/or substantial causal factors, allows me to conclude that Mr. McElhiney's Parkinson's disease was at least as likely as not caused by his exposure to the contaminated water at Camp Lejeune.

The evidence I reviewed consists of the following:

A. Medical Records

Gary Layne McElhiney, Sr
DOB: [redacted]/54

All available medical records of the plaintiff were reviewed including those prior to and after the diagnosis of Parkinson's disease was made. Particular attention was paid to all potential signs and symptoms of Parkinson's disease, when they were first noted, and their time course. Confounding or comorbid medical conditions or history which could impact such signs and symptoms were also considered.

Medical Records reviewed, including but not necessarily limited to:
- Veterans Administration
- VA, Tuskegee VA
- VA, Nashville VA
- VA, Tennessee Valley VA
- Humana Military
- Centennial Imaging Center
- Vanderbilt University Medical Center
- Dickson Medial Associates

B. Depositions

Depositions by the Plaintiff and his spouse were reviewed. These were reviewed for reported signs and symptoms of Parkinson's disease, as well as potential comorbid or confounding medical conditions. Other factors, including but not necessarily limited to family history, early life experiences including potential toxic exposures, as well as estimates of duration and degree of potentially contaminated water, were also considered.

Deposition, Gary McElhiney, 3/5/2024, 4/11/2024
Deposition, Simone McElhiney, 5/8/2024

Depositions of treating physicians, Vera Huffnagle, MD, Daniel Sherwood, MD, Heather Koons, MD, and Nicole Salloum, MD were also reviewed. These were evaluated for consistency with specific attention to the diagnoses, consistency with the plaintiff and spouse depositions, and opinions regarding diagnosis and causation:

Deposition, Vera Huffnagle, MD, 4/18/2024
Deposition, Daniel Sherwood, MD, 4/25/2024
Deposition, Heather Koons, MD, 5/14/2024
Deposition, Nicole Salloum, MD, 6/17/2024

C. Medical Literature

My literature search was performed using Pub Med. This was performed using search words, including but not necessarily limited to combinations of Parkinson's disease and parkinsonism with exposure, causative factors, etiology, toxins, head trauma, genetics and risk factors. Potentially pertinent citations from discovered articles were then secondarily investigated.

Articles were included for consideration if they were potentially important to formulating an opinion relative to this case as to what causative factors, in any, should be considered; whether they support or negate the theory of TCE as a causative factor in the development of Parkinson's disease; and, whether they support or negate other causative factors in the development of Parkinson's disease.

D. General Causation and Exposure Reports

General Causation Expert Reports, along with an Exposure Report, were reviewed. This information was considered as to whether it supports or does not support the opinion of causation of Parkinson's disease in the case of Mr. McElhiney.

GC Expert Report, Amelia K Boehme, PhD, MSPH, FAHA
GC Expert Report, Briana R De Miranda, PhD
GC Expert Report, Gary W Miller, PhD
GC Expert Report, Jason Cannon, PhD
GC Expert Report, Lucio Costa, PhD
GC Expert Report, Michael D Freeman, MedDR, PhD, MScFMS, MPH
GC Expert Report, Steven B Bird, MD
Exposure Report, Kelly Reynolds, MSPH, PhD

E. Zoom Interview with Gary McElhiney

I conducted a Zoom interview of Mr. Gary McElhiney on 1/10/2025. He was at his home. The following information was obtained during the Zoom interview:

History: several points of clarification regarding his medical records were reviewed.

Mr. McElhiney grew up on 155-acre livestock farm. His farm did not use pesticides. He had no nearby neighbors, the nearest neighbor being approximately 2 miles away.

He clarified his prior head injuries. He describes that on December 25th, 1988, he was involved in an altercation in a bar as part of his policing duties and was punched in the face and had a broken nose. He reports that he had no loss of consciousness and no confusion afterwards, and he was able to continue on with his duties of his shift.

He reports that on 8/28/1991 he was playing softball and had a collision in the outfield. He broke his wrist. He had no loss of consciousness but did endorse staggering afterwards and feeling dazed briefly with the sensation that the world was "turning". He sought medical care for the broken wrist but was able to fly to the Philippines the next day.

Mr. McElhiney endorses an absent sense of smell with a subsequent decreased taste sensation, but he has been able to maintain his weight. He wakes up three times in the night to urinate. He has restless sleep and has dream enactment behavior. He reports that in 2016 his wife described him punching her while sleeping and sleepwalking. He has recently started CPAP. His constipation is under fairly good control. His writing has decreased in legibility is currently engaging in speech and swallow therapy.

He has diminished numbers of syncopal episodes. He reports that over the last year his symptoms have been increasingly left sided. He complains of right leg tremor when he's anxious. He reports decreased right hand grip and trouble with buttons and zippers. He has been tripping but has been given an AFO which has helped him walking. He also uses a walking stick. He reports decreased low back pain over the last six months.

He takes his Parkinson's disease medication four times a day and gets approximately 2 1/2 to three hours of benefit out of each dose. His medications continue to make him feel nauseated and tired. He has not noticed significant dyskinesias. He is in the process of planned deep brain stimulation surgery scheduled for 1/14/2025.

During the interview, he was about two hours after his last medication and considered himself "on". On observation, it was notable for the following: he had 2 forehead scabs from frame placement in preparation for his DBS surgery. His voice was strong. He had no hypomimia. He had bilateral resting tremor, right more so than left. He had right > left bradykinesia with decreased right sided rapid alternating movements including his finger tapping which included decrement, hand opening and closing, and pronation supination also both decreased. He had mild slowing of left finger taps and hand opening closing. Right toe taps were very slow. Left toe taps normal. He had slowed foot stomps bilaterally.

In summary, my zoom interview was consistent with my review of Mr. McElhiney's medical records and the deposition testimony.

F. Additional Records

The following additional records were reviewed:

Military records of Gary McElhiney
ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases, 1/13/2017
United States Supplemental Interrogatory Responses, 5/15/2024

VI. Discussion of Opinions

Mr. McElhenny has Parkinson's disease. While Mr. McElhiney also has other illnesses that impair his gait and balance and contribute to pain, including his chronic lumber degenerative spine issues and knee pain, Parkinson's disease is the most significant and substantial factor in his declining state of health. While these may add complexity to the evaluation of his signs and symptoms, they do not however negate the overwhelming evidence that he has Parkinson's disease.

The diagnosis of Parkinson's disease is supported by his medical records as well as the diagnoses of his treating physicians and the VA as set out in the medical records and their deposition testimony.

At about the age of 64, Mr. McElhiney was initially evaluated for the development of tremor, noted to have a resting component (*Huffnagle, 6/7/18; McElhiney_0000000216*) and right sided (*Huffnagle, 7/10/18; McElhiney_0000000209*). At that time, he was also noted to have possible change in his handwriting. The right-hand tremors worsened and were accompanied by loss of dexterity (*Huffnagle 10/11/18 McElhiney_0000000202*). On follow up exam, he was noted to have about a yearslong development of right-handed tremors which were mostly resting predominant and worse when stressed, along with decreased dexterity and trouble with buttons (*Koons, 2/5/19; McElhiney_VA_0000001886*).

It is clear that he was developing early Parkinson's disease in the time frame of 2017-18, at about 63 or 64 years old. Unilateral onset resting tremor with loss of dexterity is classic for Parkinson's disease. Additionally, it was noted that he had associated micrographia and hypophonia – two cardinal features of Parkinson's disease. He was noted to have an unsteady gait for about two years. Additionally, he had problems with constipation, orthostasis, and incomplete bladder emptying. While early autonomic signs and symptoms can be a harbinger of other parkinsonian diseases, they are also seen in Parkinson's disease. Dr Koons assessment on 2/5/19 (*McElhiney_VA_0000001886)* notes that "...[he] has been previously diagnosed with Parkinson's, presenting for subspecialty evaluation." It is notable that while he had resting tremor, historical bradykinesia and typical associated features such as insomnia and RBD, he lacked rigidity or bradykinesia on exam and had atypical features including dystonic appearing head tremor and abdominal myoclonus. "While this may be atypical parkinsonism related to CTE/PCE exposure, chemical exposure as suggested, it is notable that his MRI brain is unremarkable without basal ganglia abnormality."

Mr. McElhiney also has noted prodromal syndrome of anosmia and REM behavior disorder, also supportive of the diagnosis of Parkinson's disease, with the patients displaying the latter carrying an estimated risk of about 75% over their lifetime *(Berg, D et al. Mov Disorders 2015; 30:1600)*. It is more likely than not that he had other pre-diagnostic signs and symptoms. Note that his signature on 6/25/15, while not particularly micrographic, gets

smaller at the end compared to earlier signatures. (writing assessment is a standard part of a parkinsonism evaluation and can appear before other symptoms are recognized) *(Counihan, TJ and Barbano, RL. Neurology 2000; 54:2107)*.

When Mr. McElhiney returns to Dr Koons (*5/14/19; McElhiney_VA_0000001849*) three months later after a trial of levodopa therapy, it was noted he had significant improvement in his tremors. While there was an initial differential etiology between Parkinson's disease versus atypical parkinsonism, Dr Koons concluded that the substantial improvement with Sinemet favored that Mr. McElhiney had Parkinson's disease. I fully agree with that opinion. By October of that year (*10/25/19; McElhiney_VA_0000001822*) Dr. Koons notes that Mr. McElhiney had progressive right sided bradykinesia and rigidity along with autonomic dysfunction. Again, the unilateral onset of his illness favors the diagnosis of Parkinson's disease.

His examination on 1/10/2025 continues to be consistent with the diagnosis of Parkinson's disease. Signs on examination of bilateral but asymmetric resting tremor and bilateral but asymmetric bradykinesia of movements is not only consistent with, but very likely to be Parkinson's disease considering the persistence of asymmetry of the cardinal features despite over 6 years of symptoms.

Further support for the diagnosis of Parkinson's disease is that Mr. McElhiney meets the International Movement Disorders Society criteria for clinically probable Parkinson's disease (*Postuma, R, et al. Movement Disorders 2015, 30: 1591- 1599*). It must be stressed that these criteria are the typically more stringent criteria for use in clinical research.

Per the Movement Disorder Society criteria, the diagnosis of probable Parkinson's disease requires:

1. The absence of absolute exclusion criteria
2. The presence of any "red flags" counterbalanced by supportive criteria.

Table 1, which sets out the criteria, is inset below:

TABLE 1. MDS Clinical Diagnostic Criteria for PD—Executive Summary

The first essential criterion is parkinsonism, which is defined as bradykinesia, in combination with at least 1 of rest tremor or rigidity. Examination of all cardinal manifestations should be carried out as described in the MDS–Unified Parkinson Disease Rating Scale. Once parkinsonism has been diagnosed:

Diagnosis of Clinically Probable PD requires:

1. *Absence of absolute exclusion criteria*
2. *Presence of red flags counterbalanced by supportive criteria*

If 1 red flag is present, there must also be at least 1 supportive criterion If 2 red flags, at least 2 supportive criteria are needed
No more than 2 red flags are allowed for this category

Supportive criteria
1. Clear and dramatic beneficial response to dopaminergic therapy. During initial treatment, patient returned to normal or near-normal level of function. In the absence of clear documentation of initial response, a dramatic response can be classified as:
a) Marked improvement with dose increases or marked worsening with dose decreases. Mild changes do not qualify. Document this either objectively (>30% in UPDRS III with change in treatment), or subjectively (clearly-documented history of marked changes from a reliable patient or caregiver). b) Unequivocal and marked on/off fluctuations, which must have at some point included predictable end-of-dose wearing off.

2. Presence of levodopa-induced dyskinesia

3. Rest tremor of a limb, documented on clinical examination (in past, or on current examination)

4. The presence of either olfactory loss or cardiac sympathetic denervation on MIBG scintigraphy

Absolute exclusion criteria: The presence of any of these features rules out PD:

1.Unequivocal cerebellar abnormalities, such as cerebellar gait, limb ataxia, or cerebellar oculomotor abnormalities (eg, sustained gaze evoked nystagmus, macro square wave jerks, hypermetric saccades)

2.Downward vertical supranuclear gaze palsy, or selective slowing of downward vertical saccades

3.Diagnosis of probable behavioral variant frontotemporal dementia or primary progressive aphasia, defined according to consensus criteria within the first 5 years of disease

4. Parkinsonian features restricted to the lower limbs for more than 3 years

5. Treatment with a dopamine receptor blocker or a dopamine-depleting agent in a dose and time-course consistent with drug-induced parkinsonism

6. Absence of observable response to high-dose levodopa despite at least moderate severity of disease

7.Unequivocal cortical sensory loss (ie, graphesthesia, stereognosis with intact primary sensory modalities), clear limb ideomotor apraxia, or progressive aphasia

8.Normal functional neuroimaging of the presynaptic dopaminergic system

9.Documentation of an alternative condition known to produce parkinsonism and plausibly connected to the patient's symptoms, or, the expert evaluating physician, based on the full diagnostic assessment feels that an alternative syndrome is more likely than PD

Red Flags

1. *Rapid progression of gait impairment requiring regular use of wheelchair within 5 years of onset*
2. *A complete absence of progression of motor symptoms or signs over 5 or more y unless stability is related to treatment*
3. *Early bulbar dysfunction: severe dysphonia or dysarthria (speech unintelligible most of the time) or severe dysphagia (requiring soft food, NG tube, or gastrostomy feeding) within first 5 y*
4. *Inspiratory respiratory dysfunction: either diurnal or nocturnal inspiratory stridor or frequent inspiratory sighs*
5. *Severe autonomic failure in the first 5 y of disease. This can include:*

    a) Orthostatic hypotension—orthostatic decrease of blood pressure within 3 min of standing by at least 30 mm Hg systolic or 15 mm Hg diastolic, in the absence of dehydration, medication, or other diseases that could plausibly explain autonomic dysfunction, or b) Severe urinary retention or urinary incontinence in the first 5 y of disease (excluding long-standing or small amount stress incontinence in women), that is not simply functional incontinence. In men, urinary retention must not be attributable to prostate disease, and must be associated with erectile dysfunction.

6. Recurrent (>1/y) falls because of impaired balance within 3 year of onset
7. Disproportionate anterocollis (dystonic) or contractures of hand or feet within the first 10
8. Absence of any of the common nonmotor features of disease despite 5 year disease duration. These include sleep dysfunction (sleep-maintenance insomnia, excessive daytime somnolence, symptoms of REM sleep behavior disorder), autonomic dysfunction (constipation, daytime urinary urgency, symptomatic orthostasis), hyposmia, or psychiatric dysfunction (depression, anxiety, or hallucinations)
9. Otherwise-unexplained pyramidal tract signs, defined as pyramidal weakness or clear pathologic hyperreflexia (excluding mild reflex asymmetry and isolated extensor plantar response)
10. Bilateral symmetric parkinsonism. The patient or caregiver reports bilateral

symptom onset with no side predominance, and no side predominance is observed on objective examination.

As per these criteria:

1. Mr. McElhiney has no absolute exclusion criteria.

2. Mr. McElhiney has one potential red flag.

   One red flag describes "severe autonomic failure in the first five years of the disease." This red flag has a note clarifying that autonomic dysfunction is a common feature of Parkinson's disease, but this red flag is added to ensure awareness of the possibility of Multi-System Atrophy (MSA), a related neurodegenerative disorder. The ensuing medical records portraying the course of Mr. McElhiney's illness strongly argue against MSA as a diagnosis *(this is consistent with the testimony of Dr Koons, P 109*). Furthermore, his records indicate that his orthostatic symptoms improved with medication adjustment. This indicates that the severity of his autonomic dysfunction was secondary to medications. Finally, he has a prolonged symptomatic response to levodopa, a feature unusual for MSA. Therefore, this "red flag" of "severe autonomic failure in the first five years of the disease" is weak at best.

However, even if one "allowed" that isolated red flag, Mr. McElhiney would still meet the criteria for probable Parkinson's disease based on the counterbalancing with supportive criteria. In the case of Mr. McElhiney, these supportive criteria include a clear and dramatic beneficial response to dopaminergic therapy as noted in Dr Koon's notes, as well as rest tremor of a limb also noted from the beginning of his clinical course. In my opinion, one cannot consider his early impaired balance as part of his parkinsonism as this symptom is more than readily attributable to his long-standing lumbar spine degenerative disease, radiculopathy, and neuropathy (*objectively documented by electrodiagnostic testing on 2/25/16; McElhiney_VA_0000001672*), as well as Vitamin B12 deficiency (*documented 3/7/16; McElhiney_VA_0000001671*). All of these are amply and repetitively documented in the medical records.

Thus, based on the medical records documenting clinical signs and symptoms of Parkinson's disease and its associated prodromal symptoms, Mr. McElhiney's treating providers, and the International Movement Disorder Society's more stringent research criteria, it is more likely than not that Mr. McElhiney has Parkinson's disease.

Causality:

Mr. McElhiney's Parkinson's disease is at least as likely as not to be caused by his exposure to the contaminated water at Camp Lejeune. Stated differently, Mr. McElhiney has Parkinson's disease secondary to his exposure to an environmental contaminant – the water at Camp Lejeune.

Overarching considerations:

In many instances, practicing neurologists commonly diagnose patients with Idiopathic Parkinson's disease – often because a causality assessment is not conducted. The term 'idiopathic' is frequently used when an underlying etiology is unknown. This however does not indicate that the underlying etiology is not knowable. Many disorders which have been labeled as idiopathic in the past have come to be recognized as caused by certain agents. For example, in 1950 a long-time smoker with lung cancer would be labeled as "idiopathic"; in 1980 that same lung cancer would be classified as lung cancer secondary to smoking. The term 'idiopathic' itself is thus a diagnosis in time. A more useful concept might be 'genetic' vs 'acquired' with 'acquired' having unknown ('idiopathic'), possible, and established causes. It is also important to recognize that what is considered 'idiopathic' Parkinson's disease is one in which the disease itself is the primary disorder, rather than secondary to another disorder. Different etiologies can cause the same disease.

Thus, while the diagnosis of "idiopathic Parkinson's disease" could be made in the case of Mr. McElhiney, given our expanding knowledge of causative etiologies including, in his case, exposure to the contaminated water at Camp Lejeune (including hazardous levels of trichloroethylene (TCE)), in my opinion, one can conclude that his diagnosis of Parkinson's disease is at least as likely to be caused by his exposure to TCE. My opinion is clearly supported by Mr. McElhiney's doctors – Dr. Huffnagle and Dr. Sherwood. In her deposition, Dr Huffnagle categorizes Mr. McElhiney's Parkinson's disease as 'not idiopathic' but rather 'secondary'. This is consistent with the opinion that his Parkinson's disease is at least as likely as not to have been caused by his exposure to TCE. In his deposition, Dr Sherwood would go further and opine that 'it is more likely than not' that his exposure played a role in his acquired Parkinson's disease.

While Dr. Huffnagle did testify that causes of Parkinson's disease are generally multifactorial, this response, from the perspective of a clinical neurologist such as myself, requires context.

Parkinson's disease is known to be associated with a number of risk factors. This is an important concept to address as it is dealing with a population of patients with that disease rather than an individual. However, for the individual patient, not all such factors are present, and one must consider the possibility that the factors interact with each other to produce the final illness. For example, if a patient has cardiac atherosclerosis and is chased by an assailant and has a heart attack, one can say that if not for the chase, the patient would

not have had a heart attack. Likewise, for example, people with genetic deficiency in alpha-1 antitrypsin deficiency are prone to develop COPD if they smoke cigarettes. A patient with both these risk factors has 'multifactorial' disease with both factors contributing to the development of the disease.

In my opinion, it would be more accurate to say that there are likely multiple factors effecting the development of Parkinson's disease, including certain genetic factors and environmental exposures. These factors are not mutually exclusive.

Therefore, in my opinion, whether Mr. McElhiney has other risk factors for the development of Parkinson's disease is irrelevant unless they were so overwhelming that his exposure to TCE was non-contributory. In my opinion, that clearly was not the case.

Mr. McElhiney does not have other risk factors that rise to the standard of 'at least as likely as not' other than his exposure to contaminated water at Camp Lejeune. The possibility of alternative causes or contributing factors for Mr. McElhiney's 'illnesses and conditions' has been raised (*US Supp Interrog Resp., p 16*). The term 'may' is imprecise and it is not clear if it is meant to convey the concept of 'as likely as not'. Likewise, the phrase 'illnesses and conditions' is overly inclusive. Mr. McElhiney, like the majority of people his age, has more than one medical condition. The issue I address in this report is his Parkinson's disease.

It is noted that Mr. McElhiney "has a long, well-documented history of muscular-skeletal issues- including back pain, joint pain, leg pain, neuropathy, and knee pain- that pre-dated Mr. McElhiney's diagnosis with Parkinson's disease." This is true, and while the signs and symptoms of these disorders might confound the early diagnosis of Parkinson's disease, they did not impact his eventual diagnosis, which is beyond question. Similarly, the fact that Mr. McElhiney had these orthopedic issues has no bearing on the development of Parkinson's disease. In fact, the existence of these issues may compound some of the symptoms of his Parkinson's disease, like making him more susceptible to falls, for example. Furthermore, while musculoskeletal conditions can certainly cause issues with ambulation, none of these are risk factors for developing Parkinson's disease. If anything, they would only serve to make his subsequent development of Parkinson's disease more debilitating.

It is also noted that to date Mr. McElhiney has not received treatment for his smoldering multiple myeloma, as this is only a pre-cancerous condition. His lack of treatment for this pre-cancerous condition would neither cause nor contribute to his development of Parkinson's disease.

Contaminated Water at Camp Lejeune - TCE and PCE Exposure[1]

In my opinion, Mr. McElhiney had substantial exposure to TCE and PCE from his time at Camp Lejeune through the date of the official exposure period (December 31, 1987). This exposure was substantially more than nominal. He lived on and off base for 13 years, but even when living off base had continued to be exposed from showers and drinking during on-base activities.

TCE and PCE have been long known to cause health risks to human such that the US Environmental Protection Agent has recently banned their use. These two chemicals are recognized contaminants in the water at Camp Lejeune (*ATSDR 2017*). Exposures at Camp Lejeune have been shown to be a risk factor for the development of Parkinson's disease (*Goldman et al JAMA Neurol 2023; 80:673-681*)

There is ample evidence that neurotoxins are sufficient to cause neurodegenerative diseases such as Parkinson's disease *(Shaw and Hoglinger, Neuromolecular Med 2008; 10:8016-8)*. TCE is clearly one of those neurotoxins *(Goldman S. Ann Rev Pharmacol Toxicol 2014; 54:141-64)*. Epidemiological evidence for long-term TCE exposure being a risk factor for the development of Parkinson's disease has been increasing since it became apparent in 2008 *(Gash DM, et al. 2008; Ann Neurol 63:184-92)*. Strong evidence for this includes increased risk associated with TCE exposure in biological twins discordant for Parkinson's disease *(Goldman S, et al. Ann Neurol 71: 776-84)*. Twin studies are used to minimize the contribution of genetic factors as well as early life exposure factors.

TCE and PCE are structurally similar. While there are fewer existing data regarding PCE, there is no significanct difference between the two chemicals to suspect that PCE would not be equally toxic (*Cannon, General Causation report)*. It is notable that absorption of TCE occurs through skin absorption, gut absorption, and inhalation. This is an important consideration in the cases of Camp LeJeune as residents there both drank and showered in the water, resulting in exposure to all modes of entry into the body.

Given the weight of the evidence, exposure to the contaminated water at Camp Lejeune, is clearly an appropriate factor to consider when conducting a causality assessment using a differential diagnosis for Parkinson's disease.

Mr. McElhiney was exposed to the contaminated water at Camp Lejeune. Mr. McElhiney was stationed at Camp Lejeune for significant periods of time between June, 1972 and October, 1988. ATSDR Assessment (*Jan 13, 2017*), "A marine in training at Camp Lejeune

[1] The causal relationship between the water at Camp Lejeune (most notably TCE and PCE) and Parkinson's disease is set out in detail in the general causation reports of Drs. Boehme, De Miranda, Miller, Cannon, Costa, Freeman, and Bird). As noted above, I have reviewed, considered, and understand these reports as to the general issues concerning the causal relationship between TCE, PCE and Parkinson's disease. Although I have deferred to their specific expertise, I have reviewed these reports and I agree that these toxins are at least as likely as not to cause Parkinson's disease.

consumes an estimated 6 liters of water per day for three days per week and 3 liters per day the rest of the week (*ATSDR 2017)*. Under warm weather conditions, a marine may consume between 1 and 2 quarts of water per hour and shower twice a day (*Bove et al. Environ Health 2014; 13:10*). It is likely that during training, the water supplied in the field came from the Hadnot Point water system with both measured and estimated levels of TCE and PCE substantially higher than their MCLs."

It is estimated that Mr. McElhiney sustained a total of 99 months of exposure from living or working at Hadnot Point, Mainside Barracks, and Tarawa Terrace. Importantly, this exposure involved both ingestion, as well as inhalation as documented in his deposition (*McElhiney, P 72 ff*). In my opinion, this is well above a minimum threshold dose. ATSDR Assessment (*ATSDR Jan 13, 2017),* notes substantially elevated levels of TCE at the Hadnot Point system and the Tawara Terrace system, to both of which Mr. McElhiney had substantial exposure, that even one of his treating doctors, Dr Sherwood opined on March 11, 2024 that it is "more likely than not that his exposure played a role in his acquired PD..." (*McElhiney_Sherwood_0000000049)*.

Studies estimate that between 1975 and 1985, TCE monthly median levels at Camp Lejeune were 366 mcg/L, far exceeding the EPA maximum contaminant levels of 5 mcg/L. Calculated median cumulative exposure during that 10-year period was 4970 mcg/L-months, >50-fold the permissible level *(Goldman et al Mov Disord 2024)*. Incorporating such studies and using conservative estimates, it is calculated that a cumulative dose of 150 mg (150,000 mcg) of TCE is sufficient to increase the incidence of Parkinson's disease with a latency of 30-50 yrs (*Gary Miller, PhD, GC Expert report*). Dr Lucio Costa notes that a dose exposure of 366 mcg/L for 3 months or longer may also cause Parkinson's disease (*Lucio Costa,PhD, GC Expert report*), but that such estimates do not exclude that shorter periods or lower levels of exposure may also cause Parkinson's disease.

Mr. McElhiney's estimated cumulative exposure exceeds that conservative limit. I have reviewed exposure charts provided to me from Plaintiff's expert Kelly Reynolds. Dr. Reynolds' charts support my opinion that Mr. McElhiney had substantial exposure to the toxins at Camp Lejeune. The charts detail a reasonable estimated dose of ingestion exposure for Mr. McElhiney.

Dr Reynolds charts are found below:

	Cumulative consumption (total ug= days*concentration per L)	Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions)
Totals HP & TT		Scenario 1	Scenario 2	Scenario 3
TCE	58,623	250,846	131,339	200,472
PCE	40,677	84,716	103,164	130,336
VC	5,836	18,395	13,960	19,311
BZ	2,842	12,342	6,343	9,737

It can be seen that Mr. McElhiney's cumulative oral dose of TCE is well over the conservative estimate to cause disease in 2 of the 3 exposure assumption scenarios, and in the third is minimally below. It should be noted that the likely true exposure is somewhere between the lowest and highest estimates, the midpoint of which would be over 191,000, well over the 150,000 conservative threshold. It must be stressed that Mr. McElhiney was also exposed to inhalation and dermal sources of exposure. Animal models indicate that inhalation exposure to TCE is equal if not even more toxic. Therefore, his total exposure is even higher than the above numbers.

Furthermore, he was also exposed to an estimated cumulative exposure of PCE of 84 to over 130mg (likely to be additive in their effects). PCE breaks down to TCE, and PCE and TCE both breakdown to Vinyl chloride, another known contaminant in the water at Camp Lejeune (*Michael Freeman, MedDR, PhD, GC Expert report*). These would only add to the already established toxicity of TCE alone. In sum, exposure to the above levels of toxins is clearly in the range that has been shown to be related to Parkinson's disease.

Given his substantial exposure, it is my opinion that Mr. McElhiney's Parkinson's disease was at least as likely as not caused by his exposure to primarily TCE, but also PCE, in the water at Camp Lejeune. I am able to rule out TCE or PCE exposure at any other location as there is no evidence documenting exposure to TCE or PCE outside of Camp Lejeune, either in his other jobs or recreational activities.

The latency period between Mr. McElhiney's exposure to the contaminated water at Camp Lejeune and the development of Parkinson's disease is to be expected given the

known mechanism in which this toxin causes the disease. His development of Parkinson's disease in or about 2016 is entirely within the scope of what is known about Parkinson's disease. It is more likely than not that he was showing symptoms even earlier. When he is examined by a neurologist on 7/27/12 (*McElhiney_VA_0000002767*), he is noted to have slowed rapid alternating movements. This deficit is an early manifestation of Parkinson's disease, and there is no more likely reason for Mr. McElhiney to display this than early Parkinson's disease. By the time Parkinson's disease is clinically manifested, it is estimated that the underlying death of dopaminergic neurons has been progressing over the prior 15-20 years. That indeed may be an underestimate as recent studies of Parkinson's disease prodrome symptom of REM behavior disorder can precede the diagnosis by even more years. On top of that, TCE induced cell death is not instantaneous, and therefore one would need to add on the time it takes for the toxin to *start* to cause the death of neurons.

Assessing the evidence addressed in this report, and based upon my education, training, and experience as a neurologist and specialist in movement disorders, it is my opinion that TCE is at least as likely as not a cause of Mr. McElhiney's Parkinson's disease. Although this diagnosis/etiology opinion can be based on clinical findings and judgment alone, it can also be supported by an application and consideration of the Bradford Hill criteria, not all of which need to be met, but individually:

1. Temporal relationship: In my opinion, as above, given the known decades-long prodrome for the development of clinical Parkinson's disease, the latency between the Mr. McElhiney's exposure to contaminated water in Camp Lejeune and the development of his Parkinson's disease meets this criterion.
2. Consistent positive associations: There is consistency between epidemiologic studies of exposure to TCE and the development of Parkinson's disease in other settings outside of the Camp Lejeune exposure. Another study in humans also showed an increased risk of Parkinson's disease in the general population exposed to TCE (*Exposure to industrial Solvent Linked to 24% Higher PD Risk- Medscape- April 26, 2024*). In my opinion, this criterion is met.
3. Magnitude of the effect estimate: It is more likely than not that Mr. McElhiney's estimated exposure to TCE exceeds those exposure levels linked to disease. (*Miller, General Causation report)*
4. Exposure-response relationship
5. Biological plausibility: TCE is a known neurotoxin that causes several dysfunctions in neurons that lead to the development of parkinsonism.
6. Coherence: there is clear coherence between the disease produced in laboratory animals and human Parkinson's disease. An animal model using this specific neurotoxin reproduces of all the key features that are seen in human Parkinson's disease (*Liu M et al J Neurochem 2010, 112:773* ; *De Miranda, General Causation report*).

7. Strength: the Goldman study comparing Marines at Camp Lejeune with those from Camp Pendelton was a large study with a significant effect size

I considered the other two criteria of analogy and experimentation (human), but I did not feel the former applied in this situation, and the latter is clearly not feasible.

Other Factors Considered

In conducting my differential diagnosis, I have also considered other possible causes of Mr. McElhiney's Parkinson's disease. The potential causes I considered are necessarily limited to the possible causes to which Mr. McElhiney was exposed given his work and life history. The other factors that I considered were: (1) Head Trauma; (2) Genetics/Family History; and (3) exposure to other neurotoxins known to cause Parkinson's disease.

Head trauma:

While there is an association between head trauma and Parkinson's disease, in my opinion Mr. McElhiney has not had the significant head trauma often considered as a risk factor. Obviously, head trauma severity is a continuum. My review of the evidence does not support any type of finding or opinion that Mr. McElhiney suffered the type of head trauma that would be necessary to cause Parkinson's disease.

On VA Mental Health intake, 9/7/16 (*McElhiney_VA_0000003405)*, reference is made to two events: one in 1987 with history at the time of this documentation (*medical records, 9/7/16 McElhiney_VA_0000003405*) indicating that he reports having felt dizzy and disoriented, and a second event where he was 'knocked out' after a collision playing softball. The initial note of Dr Heather Koons on 2/5/19 (*McElhiney_VA_0000001886)* reports "recurrent head trauma with one associated with loss of consciousness." Dr. Koons testifies in her deposition of 5/14/24 that she did not think that Mr. McElhiney had "the volume or time course that would suggest they were the cause of his Parkinson's." Based on the actual historical records, I would agree with that opinion.

It is important to note that retrospective histories, especially over decades, are prone to inaccuracies. In my opinion, it is therefore important to review the contemporaneous records.

In reviewing Mr. McElhiney's head trauma history, records indicate that these were few and relatively mild.

On 12/25/88 (*McElhiney_VA_0000001163)*, records indicate that he was punched in the nose in the setting of an assault. He sustained a nasal fracture and records indicate being "dizzy for a little while". No loss of consciousness or alteration of consciousness was recorded. No diagnosis of concussion was given. His 1/10/25 description of the event would

also not be consistent with a concussion. In my opinion, one cannot say that he sustained a concussion from this event.

On 8/28/91 (*McElhiney_VA_0000001107)*, he had another traumatic event when he ran into another softball player, fracturing his wrist. There is no contemporaneous mention of head trauma, and no documentation of any alteration of consciousness. While history obtained many years later that he was 'knocked out' from this event, it is not corroborated by contemporaneous medical records. In my opinion, head trauma with loss of consciousness would likely have appeared in the records had it occurred. His description on 1/10/25 of the event included no loss of consciousness but feeling 'dazed'. This would be consistent with if anything, a minimal concussion at best, for which he had no significant post-concussive sequelae and was able to return to full duty.

These events would not have reached the inclusion criteria in the large retrospective study evaluating the risk of Parkinson's disease among military veterans with mild traumatic brain injuries (*Gardner et al.,l Neurology 2018; 90:e1771)*. While this study shows an associated increased risk of Parkinson's disease with mild traumatic brain injury, the inclusion criteria (that is, what was required to qualify as mild TBI) required symptoms of posttraumatic amnesia, alteration of consciousness, or loss of consciousness. None of these criteria appear in the contemporaneous medical records of the events of Mr. McElhiney. His 1/10/25 description of the event of 8/28/91 would barely reach the minimal criteria used by the above retrospective study (he had no loss of consciousness, no post traumatic amnesia, and a brief feeling of being dazed), with the criteria for this category accepting up to 24 hours of alteration of consciousness.

Furthermore, the veteran's administration itself did not consider his prior head traumas to be significant enough to have considered him having a diagnosis of traumatic brain injury. See for example the VA mental health record of 6/10/16 (*McElhiney_VA_0000000544)* noting that "TBI was not shown in the records review and there was no diagnosis of TBI."

Additionally, Mr. McElhiney denies exposure to explosions. (*McElhiney Deposition at 127)*.

Finally, his lack of abnormalities on brain MRI scans makes Parkinson's disease due to significant head trauma less likely.

Overall, my opinion to a reasonable degree of medical certainty, is that Mr. McElhiney did not sustain head trauma significant enough to be considered as a significant or substantial contributing factor to the development of his Parkinson's disease. In my professional medical opinion, to consider Mr. McElhiney's head trauma, in light of his substantial Camp Lejeune water exposure, would be speculative as a potential cause of Mr. McElhiney's Parkinson's disease.

Family History:

As noted above, research in twins indicates that genetic factors, even if present, are unlikely to effectively mitigate against the increased risk of developing Parkinson's disease in the setting of TCE exposure.

Mr. McElhiney has no known family history of Parkinson's disease. He has no known genetic risk factors, which, given his lack of a family history, in a cohort of substantial size, including 5 siblings, children and other known relatives, would not be suspected. Lack of Parkinson's disease in his siblings, also diminishes the likelihood that any possible early life exposure played a role in Mr. McElhiney's development of Parkinson's disease.

He has 5 siblings, for a total sibling size of 6, including him. During depositions, questions have been raised about this as a risk factor for Parkinson's disease. Dr. Koons testified that his sibling size is not significant to his diagnosis of Parkinson's disease (Koons *deposition p 36*). A recent study of over 3.5 million people looking at early life risk factors found an association with parental occupation as farmers (distinction of type of farming was not made; see below), male sex and family history of PD, but not overall sibship size (*Liu B, et al. PLoS One. 2016; 11(4):e0152841*).

I agree with his treating doctor, Dr. Koons, and the literature, that Mr. McElhiney's family history is not a likely contributor to his Parkinson's disease and there is no indication of a genetic component to his disease.

Other potential toxins:

First and foremost, it must be stressed that it is irrelevant whether the water that Mr. McElhiney was exposed to at Camp Lejeune contained toxins other than TCE. Toxic exposures can be additive, but he was clearly exposed to levels of TCE high enough to be considered a clear risk factor or potential cause for his Parkinson's disease. Any further exposures to neurotoxic chemicals, such as PCE, vinyl chloride, and benzene, in the water at Camp Lejeune would only further increase the risk. Again, his exposure to TCE, alone, was sufficient to cause his Parkinson's disease and, in my opinion, is the cause of his Parkinson's disease.

Mr. McElhiney had no documented exposure, other than his time on Camp Lejeune, to other potential neurotoxins that might be contributors to being causative for Parkinson's disease.

The Supplemental Interrogatory response (5/15/2024, p 17) notes a 14-year exposure to asbestos. While he had exposure to asbestos, a clear toxin, asbestos is not considered a substantial causative agent for PD. Further, the VA did not find his exposure to asbestos significant (*see note of 9/16/85*). He had no exposure to Agent Orange.

The Supplemental Interrogatory response (5/15/24, p 17) also notes that he grew up on a farm. Although he grew up on a farm, it was a pig farm and not one that involved the use of widespread herbicides (*see Deposition by Mr. McElhiney 3/5/24, P 33*). His denies using pesticides for their home use. While it was alluded to in several depositions that Mr. McElhiney grew up on a farm, the evidence shows that he was not exposed to pesticides, such as Paraquat.

Thus, he had substantial exposure to TCE while at Camp Lejeune and no clear exposure to other toxins associated with Parkinson's disease other than his exposure to the contaminated water he encountered at Camp Lejeune.

VII. Diagnosis and Causation Conclusion

In my opinion, to reasonable degree of medical certainty, Mr. McElhiney is suffering from Parkinson's disease. It is also my opinion, to reasonable degree of medical certainty that his exposure to the contaminated water at Camp Lejeune is at least as likely as not to be a cause of Mr. McElhiney's Parkinson's disease.

VIII. Prognosis and Impact on Quality of Life

Parkinson's disease is a progressive neurodegenerative disorder. It is inexorable and uncurable. The effect on a person's quality and potential length of life cannot be understated. The debilitating symptoms of Parkinson's disease will progress and worsen in every patient as they age with the disease – a fact known to the patient and a cause of extraordinary mental distress regarding their future life and health. The typical Parkinson's disease patient faces a life of declining health and an inability to care for themselves. As the disease progresses with age, if the patient lives long enough, they will progress to requiring full-time care. This adds to the mental distress as the patient, including Mr. McElhiney, have a reasonable fear that they will become a burden to their family.

The impact of Parkinson's disease on quality of life of an individual is substantial and frequently leads to hospitalizations. Parkinson's disease is a multi-systemic disease, that is, it affects many functions of the body, and it is not simply a "movement disorder". There are both "motor", that is to say movement, manifestations and "non motor manifestations." These most often develop gradually, such that exact dates of onset are rarely possible to say and worsen over time.

In regard to the movement symptoms, certainly the resting tremor can impact quality of life by both being distracting to other people and socially embarrassing. Social embarrassment is not trivial as maintaining an active social life is important for overall cognitive health as we age. The resting tremor can also interfere with some activities such as trying to fall asleep. But the resting tremor is not the motor problem that has the most impact

on quality of life. Other cardinal features of Parkinson's disease include slowness of movements (bradykinesia)and stiffness (rigidity) of movements. These significantly affect quality of life and have an early impact on the ability to do fine hand manipulations with the classic symptom being an inability to button buttons. However, many activities are impacted by the loss of dexterity. The bradykinesia and rigidity also impair walking. Patients with Parkinson's disease develop substantial and significant difficulty with walking and frequently become wheelchair bound. Walking is also impacted by two other manifestations of the disorder including freezing of gait, a situation where a person stops in their tracks and can potentially fall over, and loss of balance. Loss of balance is particularly dangerous in Parkinson's disease as falls are a significant contributor to morbidity and mortality.

Non motor manifestations can be even more impactful. These are wide-ranging and they include but are not necessarily limited to: sleep disturbances; loss of sense of smell; autonomic disturbances such as low blood pressure leading to syncope (blacking out); R.E.M. behavior disorder; or acting out one's dreams which can cause injury to the patient or their bedmate; constipation, actually to the point of needing disimpaction; urinary dysfunction (including urge incontinence , nocturia, and incomplete bladder emptying), skin changes; Joint pains; vision changes; sexual dysfunction and of course, cognitive changes including dementia. Mood disorders, specifically anxiety and depression are also associated with Parkinson's disease and are considered non-motor symptoms.

Mr. McElhiney is already suffering from a number of these symptoms, which will likely worsen as the disease progresses. Furthermore, he is likely to suffer from even more symptoms and complications in the future as his disease progresses. As set out below, many symptoms can be temporarily treated with medications. His medical records report temporary improvements with medication, but this will inevitably change, and he will either need increased dosing, or the medication will stop working for him. It is important to realize, however, that while symptoms can be temporarily held at bay with medications, progression of the disorder is inexorable and the benefit of many of these treatments run their course. Furthermore, many of the medications used to treat Parkinson's disease come with their own side effects, as is seen in Mr. McElhiney's medical records.

He is already suffering from the following problems that are more likely than not substantially or wholly related to his Parkinson's disease. Some of the problems will continue to worsen as a direct result of, or by a significant contribution from, his Parkinson's disease.

He has already been diagnosed with Parkinson tremor, first evaluated in 2018 (*Huffnagle 6/7/18; McElhiney_0000000216*). He exhibited early loss of manual dexterity for activities such as buttoning. (*Huffnagle 10/11/18; McElhiney_0000000202*). Manual dexterity is needed for many activities of daily life, not just buttons, but zippers and buckles. As such, patients with Parkinson's disease have increasing difficulties getting dressed on their own and require increasing help from others, limiting their independence. Switching to 'slip-on' clothes serves as a temporary work-around, but this also limits choice of clothing

and in my experience, patients often feel less socially appropriately dressed, leading to further embarrassment and social isolation. Mr. McElhiney's dexterity has declined, as expected, to include difficulty with silverware (*Koons 5/10/23; McElhiney_0000000436*). As in all cases of Parkinson's disease, he will eventually develop slowness of movement (bradykinesia) and stiffness (rigidity) that will come to affect his life, as noted by Dr. Koons that he had five years of "functionally impactful bradykinesia and rigidity (*Koons, 10/25/19; McElhiney_VA_0000001822*).

Toe dystonia and foot cramping are painful manifestations of Parkinson's disease, frequently presenting early on the disease. Mr. McElhiney has suffered from these since 2017 (*Huffnagle 6/7/18; McElhiney_0000000216*). While there are other potential causes for his foot pains, such as his neuropathy and lumbar radiculopathy, the toe curling is more common in Parkinson's disease and is temporally associated with his Parkinson's disease rather than his neuropathy and radiculopathy (as he has had both for years). In my opinion, it is more likely than not that his painful toe curling is part of his Parkinson's disease and not his other comorbidities.

Another manifestation of the motor deterioration in Parkinson's disease is the loss of strength of speaking, known as hypophonia. This results in a soft voice which becomes increasingly difficult for others to understand, frustrating the sufferer and caretakers alike, and increasing a sense of isolation. Mr. McElhiney has exhibited this as well (*Koons 2/5/19; McElhiney_VA_0000001886*).

The impact of Parkinson's disease on gait cannot be understated. Walking becomes increasingly slow, difficult, and unsteady with increasing risk of and actual falls. This leads many patients to require assist devices and eventually wheelchairs. Mr. McElhiney has other comorbidities that clearly affect his gait, started with his long-standing lumbar spine orthopedic issues from the 1990s, his neuropathy, and possibly from Vitamin B12 deficiency. However, while one cannot say that his Parkinson's disease is the only reason for his gait difficulties, Parkinson's disease worsens this baseline and is clearly additive to his difficulties. Unsteadiness of his gait is noted by early 2019 at which time he is noted to have a positive 'pull test' (*Koons 2/5/19; McElhiney_VA_0000001886*). The 'pull test' is a demonstration of a person's inability to reflexively maintain their center of gravity and prevent themselves from falling and is considered an ominous sign in Parkinson's disease and portends an increased risk for future falls, as Mr. McElhiney has already begun to manifest by tripping (*Huffnagle 1/18/21; McElhiney_0000000066*).

In my opinion, Mr. McElhiney is more likely than not to have increasing need for assist devices and eventually a wheelchair.

Depression is a common comorbidity of Parkinson's disease and is a manifestation of the disorder, occurring in 40-50% of patients (*Marsh, L 2013, Curr Neurol Neurosci Rep 13:409*). While depression has been attributed to Mr. McElhiney's diagnosis of PTSD and adjustment in his records, it is important to recognize that anxiety and depression can be

harbingers of the onset of Parkinson's disease. In his case, he is diagnosed with these in 2016. It is impossible to say with certainty whether his depressive symptoms are caused by his Parkinson's disease, but it is more likely than not that the pathology of Parkinson's disease contributes to its severity. Mr. McElhiney's PTSD contributes to emotional distress, but so does the diagnosis of Parkinson's disease. These are not mutually exclusive and in fact may be additive in their emotional burden. The effect of depression on quality of life is self-evident.

Sleep disturbances, including REM behavior disorder (RBD), are also part of the Parkinson's disease effect on quality of life. Mr. McElhiney is noted to have RBD (*Koons 2/5/19; McElhiney_VA_0000001886*). RBD causes patients to act out their dreams, sometimes violently and is known to cause injuries to the patient and their bed partner. For Mr. McElhiney, this was considered significant enough to warrant treatment with a benzodiazepine medication (*Koons 4/28/20; McElhiney_VA_0000001787*). RBD is not his only sleep disorder issue; he is noted to have had sleep fragmentation and insomnia for years and becoming increasingly problematic (*Koons 5/10/23; McElhiney_0000000436*). Lack of restorative sleep leaves people feeling tired, less alert, and less cognitively sharp.

He has a loss of sense of smell since approximately 2016 (*Koons 2/5/19; McElhiney_VA_0000001886*). Smell and taste work together and loss of sense of smell leads to diminished enjoyment of eating.

Further complicating eating is the problem of dysphagia, or difficulty/ maladaptive swallowing. Mr. McElhiney is having this problem several times per month (*Koons 5/10/23 McElhiney_0000000436*). This was evaluated 8/23/23 (*McElhiney_0000000687)* and rated as mild,but is still a significant risk for morbidity and mortality via choking and aspiration leading to pneumonia.

The autonomic nervous system also degenerates as Parkinson's disease progresses.

Orthostatic hypotension (sudden drop in blood pressure) with syncope (loss of consciousness) is a complication of both Parkinson's disease as well as its treatment. On 12/20/22 (*McElhiney_0000000722)* Mr. McElhiney passed out while driving. While he was not injured in the event, it is clearly dangerous to the patient, passengers, other drivers, and pedestrians, and is a significant cause for losing one's driving privileges. Even when not driving, blacking out is associated with falls and injuries, including hospitalizations. Mr. McElhiney was noted to be weak and dizzy with low blood pressure (*Huffnagle 7/12/22; McElhiney_0000000060*).

Urinary issues are common in Parkinson's disease and are documented as part of Mr. McElhiney's problems (*Huffnagle 10/11/18; McElhiney_0000000202*). Nocturia causing insomnia is documented (*Koons 2/5/19; McElhiney_VA_0000001886*), as is frequent urination (*Koons 5/14/19; McElhiney_VA_0000001849*), likely due to incomplete bladder emptying, a complication of Parkinson's disease. This leads to poor sleep as well as limiting

social interactions and extended activities to locations with convenient toileting. Sexual dysfunction is also common in Parkinson's disease, causing significant distress to the patient with an obvious effect on their relationship with their partner, and an important quality of life issue. Mr.McElhiney notes that he has been having this problem for at least 7 years (deposition 3/5/24, p 144). While the deposition of 4/11/24 (p 38) mentions that he had no sexual dysfunction, this was in the context of his spine issues and thus strengthens the connection between his sexual dysfunction and his Parkinson's disease.

Constipation is another common symptom of Parkinson's disease and yet another one from which Mr. McElhiney suffers (*Koons 10/25/19; McElhiney_VA_0000001822*).

Cognitive problems are one of the most negatively impactful complications of Parkinson's disease. Mild cognitive impairment overall affects about half of all patients with Parkinson's disease. Mr. McElhiney is noted to be having some issues with memory, such as losing things (*Koons 2/5/19; McElhiney_VA_0000001886*). SLUMS testing indicates mild cognitive disorder (*Sherwood 8/22/23; McElhiney_Sherwood_0000000064*).

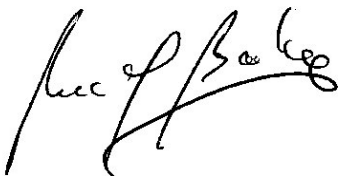
He is also noted to be hallucinating, such as seeing cats and people, either as part of his Parkinson's disease or as a side effect of its treatment. In either case, these do not portend well for his future. It is estimated that the probability of developing frank dementia is 27% at 10 years of disease duration and 50% at 15 years of disease duration (*Gallagher, J, et al. 2024, Neurology 103;e209699*).

While many of the symptoms of Parkinson's disease can be controlled with medications, the efficacy of many of these medications generally declines over time even after significant dose titration. Further, there are also significant side effects of the medications needed to treat Parkinson's disease, including but not necessarily limited to nausea, dropping blood pressure, confusion, sleepiness, and hallucinations. As responsiveness to medications declines, side effects worsen to the point where further increases are more detrimental to quality of life than the symptom the medication is indicated to control.

Because of the involvement of multiple body systems and the need to constantly monitor the benefits of the medications, Parkinson's disease is a challenging disease for doctors to control. Mr. McElhiney's treatment is no different. The treatment of Mr. McElhiney's Parkinson's disease is complicated by his increasing need for medication. By 5/1/24, he was on Parkinson's disease related medications including for his low blood pressure and passing out, and depression, as well as1600 mg levodopa and entacapone for his movement problems. All treatments have potential side effects. In addition to their possible role in his hallucinations, side effects from his medications have included drowsiness and nausea from ropinirole (*Huffnagle 12/13/18; McElhiney_0000000189*), fluctuations in his ability to move (*Koons,12/09/20; McElhiney_VA_0000001719*), and nausea and vomiting described as 'an ongoing struggle" (*Sherwood 2/22/24; McElhiney_Sherwood_0000000056*).

Parkinson's disease is progressive. Despite best medical management, disabilities accumulate, and quality of life suffers. For certain patients, aggressive therapy such as deep brain stimulation is an appropriate albeit a non-curative and invasive approach to control symptoms and maintain quality of life for as long as possible. Deep brain stimulation requires drilling burr holes through skull, placing electrodes deep into the brain itself and channeling wires under the skin to a subcutaneous battery pack in the chest. There are obvious attendant risks associated with the surgery including hemorrhaging into the brain and serious brain infections. Furthermore, the patient has to commit to many trials of adjusting stimulation parameters to optimize the therapy and to undergo surgery for periodic battery replacement. The decision to undergo this procedure is not taken lightly but is an acknowledgement of the seriousness and negative impact of the underlying progressive Parkinson's disease. Mr. McElhiney has recently undergone this surgery.

In sum, in my opinion, Mr. McElhiney's Parkinson's disease was at least as likely as not caused by his substantial exposure to TCE in the water while at Camp LeJeune. Recent research indicates that Parkinson's disease progression may be faster in people exposed to TCE with shorter periods of time until the development of psychosis, bone fractures and falls (*Goldman 2024, Movement Dis 39: 1732*). Mr. McElhiney has suffered significantly already from his Parkinson's disease, a condition which only progresses over time. His quality of life is currently diminished and will only be increasingly so over the ensuing years, and probably more rapidly so. And as addressed above, while medications may assist temporarily, ultimately – and with reasonable likelihood – medications will cease to relieve Mr. McElhiney's symptoms and are accompanied by potential side effects. The effect on quality of life is true for his wife as well, as the caretaker burden for a patient with Parkinson's disease is immense and increases commensurate with the progression of the disease.



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February 7, 2025

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Ye Q, Wen Y, Al-Kuwari N, et al. Association between Parkinson's disease and melanoma: Putting the pieces together. Front Aging Neurosci 2020; 12: 60

Publications (Last 10 years):
(for full list, see CV)

Abstracts/ Posters/ Platform Sessions

Globus pallidus oscillatory activity correlates with symptom improvement in patients with Parkinson's disease and dystonia. Amudhan A, Sell D, Barbano RL, et al. Finger Lakes Neuromodulation Conference. Rochester Oct 2024.

A Phase 2b Multicenter, Randomized, Double-Blind, Placebo-Controlled Study of OnabotulinumtoxinA for the Treatment of Upper Limb Essential Tremor: ELATE Trial in Progress. Barbano R, Simpson D,Patterson K, Alibhai N, James L. International Neurotoxin Association, 7th Annual Meeting, Berlin, Germany Jan 2024.

Incidence of dysphagia and comorbidities in patients with cervical dystonia, analyzed by botulinum neurotoxin treatment exposure. Sadeghi M, Ukah A, Yue EX, Ifantides KB, Huang NY, Lee, J Barbano R. International Neurotoxin Association, 7th Annual Meeting, Berlin, Germany Jan 2024

Dry cleaning chemicals and Parkinson's Disease. Pawlik ME, Lettenberger SE, Zafar M, et al. International Congress of Parkinson's Disease and Movement Disorders. Copenhagen, DE Aug 27-31, 2023

Efficacy of DaxibotulinumA for injection over successive treatments in adults with isolated cervical dystonia in the phase three ASPEN-1 and ASPEN-OLS trials. Comella C, Barbano R, Rudzinska M, et al. American Association of Physiatry Annual Meeting. Anaheim , USA Feb 21-24, 2023

Head tremor jerkiness in cervical dystonia: clinical and computer vision assessments. Cisneros E, Le L, Vu JP, et al. 2nd International Tremor Congress. NY, USA May 18-19, 2023

Clusters of Parkinson's Disease may be linked to widely used dry cleaning solvents. Pawlik ME, et al. International Congress of Parkinson's Disease and Movement Disorders. Madrid, Spain Sep 14-18, 2022

Impact of disease severity on presentation subtype and Onabotulinum toxinA utilization on patients with cervical dystonia: Results from the CD Probe completer population. International Neurotoxin Association Annual Meeting, New Orleans, LA, Jul 2022

Benefits of treatment with Onabotulinum toxinA in naïve and non-naïve pateints with cervical dystonia are sustained over time in CD-PROBE. P Agarwal, M Schwartz, A

Zuzek, et al. Canadian Neurological Sciences Federation Congress, Toronto, Canada Oct 25-28, 2021

Impact of Disease Severity on Presentation Subtype and Onabotulinum ToxinA Utilization in Patients with Cervical Dystonia: Results from the CD PROBE Completer Population. Agarwal P. Barbano R, Moore H, et al. International Parkinson and Movement Disorder Society. Virtual, Sep 17-22, 2021

Head Tremor in Cervical Dystonia: the effect of postural maneuvers. E Cisneros, JP Vu, Q Chen, et al. Society for Neuroscience 2020 Annual Meeting. Washington, DC, Oct 2020

Validation of Fox Insight Cohort via Virtual Research Visits. TL Myers, RB Schneider, M Daeschler, et al. American Academy of Neurology 72nd Annual Meeting, Toronto, CA , April 2020

Stiff limb syndrome masquerading as a focal limb dystonia. PE Morrison, RL Barbano. International Congress of Parkinson's Disease and Movement Disorders. Nice, FR Sep 22-26, 2019

Genome-wide association study identifies common genetic variants associated with cervical dystonia. GK Berkman, R Barbano, Y Sun, H Jinnah. International Congress of Parkinson's Disease and Movement Disorders. Hong Kong, Oct 4-8, 2018

Dystonia, Tremor and Dystonic Tremor. AG Shaikh, AR Rosen, LM Scorr, A Cotton, RL Barbano, C Testa, HA Jinnah for the Dystonia Coalition Investigators . International Congress of Parkinson's Disease and Movement Disorders. Hong Kong, Oct 4-8, 2018

Cervical dystonia and substance abuse. A Mahajan, N Patel, J Jankovic, L Marsh, H Jinnah, C Comella, R Barbano, J Perlmutter. American Academy of Neurology 70th Annual Meeting, Los Angeles, April 2018

Levodopa-carbidopa intestinal gel therapy (LCIG) choices and clinical outcomes in DBS-eligible versus palliative stage PD patients. M.A. Burack, A. Santiago, K. Biglan, R. Barbano. 30th Annual Symposium on Parkinson Disease and Other Movement Disorders. Portland, OR Sept 19, 2016

Assessing Vestibular Function in Individuals with Cervical Dystonia and the Effects of Botulinum Toxin Treatment. Kelly L. Andrzejewski, *et al*. 20th International Congress of Parkinson's Disease and Movement Disorders. Berlin, Germany June 19-23,2016

Effectiveness of OnabotulinumtoxinA in Patients with Cervical Dystonia Naïve to Botulinum Toxin Treatment. C. Singer, R Barbano, M Schwartz, *et al*. 19th International

Congress of Parkinson's Disease and Movement Disorders. San Diego, CA June 14-18,2015

Inter-rater reliability of the severity subscale of the revised Toronto Spasmodic Torticollis Rating Scale (TWSTRS-2). C Comella, MD *et al*. Platform Session. American Academy of Neurology 67th Annual Meeting. Washington, DC. April 18-23, 2015

Reliability and validity of the revised TWSTRS psychiatric module (TWSTRS-PSYCH) of the Comprehensive Cervical Dystonia Rating Scale (CCDRS). Mateusz Zurowski, MD, *et al*. 18th International Congress of Parkinson's Disease and Movement Disorders. Stockholm, Sweden. June 8-12, 2014

Convergent validity of the revised TWSTRS modules of the Comprehensive Cervical Dystonia Rating Scale (CCDRS). Cynthia L. Comella, M.D., *et al*. 18th International Congress of Parkinson's Disease and Movement Disorders. Stockholm, Sweden. June 8-12, 2014

Clinimetric testing of the modules of the Comprehensive Cervical Dystonia Rating Scale (CCDRS). C. Comella, *et al*. Platform presentation. American Academy of Neurology 66th Annual Meeting, Philadelphia, PA April 26-May3, 2014

Peer Reviewed Publications

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Chunga, N, Barbano R, et al. A novel PDE8B gene variant associated with autosomal dominant striatal degeneration. Mov Disorders Clin Pract. 2024; accepted

Dorsey ER, Kinel D, Pawlik ME, et al.. Dry-Cleaning chemicals and a cluster of Parkinson's disease and cancer: A retrospective Investigation. Mov Disorders 2024; 39: 606-613. Doi 10.1002/mds.29723. PMID: 38389433

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Scorr LM, Choo HJ, Kilic-Bermen G, et al. Clinical features and evolution of blepharospasm: A multi-center international cohort and systemic literature review. 2021 Dystonia 2022; 1: 10359. doi:10.3389/dyst.2022.10359. PMID: 36248010

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Andrzejewski K, Ma S, Owens A, et al. Alterations in Vestibular Function in Individuals with Cervical Dystonia and the Effects of Botulinum Toxin Treatment. Basal Ganglia 2018. *Accepted MS ID: BAGA_2017_18_R2*

Klimkowicz-Mrowiec, A, Kasprzyk-Galon K, Barbano R. Isolated hemifacial spasm presenting as unilateral, involuntary ear movements. Parkinsonism & Related Disorders. 2018. 49:106-107. PMID 29329937

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Andrzejewski KL, Barbano R, Mink J. Cannabinoids in the treatment of movement disorders: a systematic review of case series and clinical trials. Basal Ganglia. 2016; 6(3):173-181

Comella CL, Perlmutter JS, Jinnah HA, et al. Clinimetric testing of the comprehensive cervical dystonia rating scale. Mov Disord 2016; 31: 563-9 PMID:26971359

Barbano, R. Botulinum Toxins in clinical practice: Gaps in Knowledge. Neurol Clin Pract 2016; 6:206-208. PMID: 27347437

LeDoux MS, Vemula SR, Xiao J, et.al. Clinical and genetic features of cervical dystonia in a large multicenter cohort. Neurol Genet 2016; 2(3) e69. PMID: 27123488

Viollet L, et al. Alternating Hemiplegia of Childhood: Retrospective genetic study and genotype-phenotype correlations in 187 subjects from the US AHCF registry. PLOS ONE 2015; 10(8):e0137370 PMID: 25996915

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Barbano RL. Standard strategies for diagnosis and treatment of patients with newly diagnoses Parkinson disease. Neurol Clin Pract 2013; 3(6):475-476. PMID 30107015

Testimony (Last 4 years)

Caption/Plaintiff	Jurisdiction	Case/Date	Deposition	Testimony
Hedaa, Hazel (fact witness)	Rochester	11/29/21	Yes	No

Compensation/Rate Sheet:

My fee to cover record review, literature search, patient interview, manuscript/expert report preparation, and court preparation as necessary is $580/hr.

CV (attached)

CURRICULUM VITAE

Richard L. Barbano, MD, PhD, FAAN

Addresses

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University of Rochester Neurology
919 Westfall Rd ., Bldg. C Suite 100
Rochester, NY 14618

University of Rochester
265 Crittenden Boulevard
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Rochester, NY 14642

Positions

2020- Present	Consulting Neurologist, Rochester Psychiatric Center, Rochester, NY
2014- 2018	Associate Chair, Community Outreach and Regional Development, Dept of Neurology, University of Rochester
2012- 2020	Chief, Movement Disorders Unit, Dept of Neurology, University of Rochester
2009- 2012	Chief of Physical Medicine and Rehabilitation, Rochester General Hospital, Rochester, NY
2008- Present	Professor of Neurology, University of Rochester, Rochester, NY
2007- 2014	Chief of Neurology, Rochester General Hospital, Rochester, NY
2007- 2014	Attending Neurologist, Rochester General Hospital
2001 -Present	Attending, Movement Disorders Division
1997- Present	Director, Botulinum Toxin Clinic
1999 -2008	Associate Professor of Neurology, University of Rochester
1993- 1999	Assistant Professor of Neurology, University of Rochester
1993- Present	Attending Neurologist, Strong Memorial Hospital, Rochester, NY
1992- 1993	Chief Resident in Neurology/Graduate Management Training Program. Columbia-Presbyterian Medical Center, New York, NY
1990 - 1993	Resident in Neurology, Columbia-Presbyterian Medical Center, New York, NY
1989 - 1990	Internship in Internal Medicine, Montefiore Medical Center, New York, NY

Education

1985 - 1989	MD	Albert Einstein College of Medicine, Bronx, NY
1979 - 1985	PhD	Northwestern University, Evanston, IL Light and Ultrastructural Immunohistochemistry of EAE and Theilers Virus Induced Demyelination
1975 - 1979	BS	State University of New York at Albany, Albany, NY Biology Major, Physics Minor

Awards

Senior Faculty Award, Department of Neurology, University of Rochester, 2020
Patient Satisfaction Award, Strong Memorial Hospital 2013
Neurology Faculty Residency Teaching Award, 1994-95
Andrew Doyle Memorial Award for Excellence in Neurology, 1989
International Health Fellowship Award recipient to study health care in Chile, SA, 1989

Honors

Magna Cum Laude, State University of New York at Albany, 1979
Fellow, American Academy of Neurology, 2005

Professional Membership

1991 - Present	American Academy of Neurology
1995 -2007	American Association of Neuromuscular and Electrodiagnostic Medicine
1997 – 2007	Peripheral Neuropathy Association
2001 – Present	Dystonia Study Group
2002- Present	Member, International Neurotoxin Association
2005 – Present	Fellow of the American Academy of Neurology
2006- Present	Member, Movement Disorders Society
2007-2011	Dystonia Study Group, Secretary-Treasurer
2009- 2020	Member, Classification of Evidence Committee, American Academy of Neurology
2009- 2015	Member, Guideline Development Subcommittee of the American Academy of Neurology
2012- 2023	American Neurological Association

Medical Licensure

1992-Present	New York #188639

Board Certification

1990	National Board of Medical Examiners #379936
1994	American Board of Psychiatry and Neurology #40412; Recertification 2014
1998	American Board of Electrodiagnostic Medicine #2355; Recertification 2008

Clinical Activities

1993 - 2021	Attending, Neurology Consultation Service . Evaluate neurologic patients in the emergency room and consultation on inpatients. Resident and medical student supervision.
1993 - 1999	Electromyographer, University of Rochester Neurology.
1993 – 1997	Co-Director, Adult Attention Deficit Disorder Clinic. With three colleagues, developed this clinic for evaluation and treatment of patients, as well as research on the illness.
1994 - 1998	Attending and Preceptor, HIV Neurology. Service dedicated to both patient care and Implementation of research protocols. Supervised one full-time fellow and resident. 4 months/year.
1995 - 2010	Attending, Neurology, Stroke Service. Evaluation and treatment of patients with strokes, as well as implementation of research protocols. Supervision of residents.
1995- Present	Attending, Neurologist, Botulinum Toxin clinic. Treatment of patients with botulinum toxin for targeted therapy.
1997 - Present	Director, Botulinum Toxin Clinic. Treatment of dystonic and spastic patients; supervision and training of 1-3 fellows/yr.
1999- 2007	Electromyographer, Dept of Neurology EMG Lab. Training of Fellows
2006- 2014	Attending, Deep Brain Stimulation (DBS) Program
2007- 2014	Attending, Neurology Consultation Service, Rochester General Hospital. Inpatient and outpatient consultation; supervision of residents and medical students
2019-2020	UR/Bassett Health Telemedicine consultation service
2020- Present	UR/Bassett Health Telemedicine Movement Disorder Subspecialty
2020- Present	Neurology Consultant, Rochester Psychiatric Center General neurologic care for hospitalized and imprisoned patients

2023- Present	St Joseph's Community Clinic. Neurology Attending Volunteer, Supervising medical student run clinic for the uninsured

Intramural Teaching and Mentoring

1993 – Present	Intramural Lectures: Medical Student, Neurology Residency, Psychiatry Residency, Neuroscience Graduate Program and Electrophysiology Fellowship: "Headache"; "Peripheral Neuropathy"; "Neuropathic Pain"; "Diabetic Neuropathies"; "Back and Neck Pain"; "Botulinum Toxin Therapy"; "Dystonia"; "Ataxias"
1993- Present	Examiner: UR residency annual Mock Boards
1993 –1995	Attending, Residency Longitudinal Firm
1993 – 2021	Inpatient Resident and Medical Student supervising Attending
1998- 2007	Preceptor, Chief Resident Clinic. Individual supervision 2x week.
2000 – 2001	Clinical Mentor, MD,PhD Clinical Scientist Program. One-on-one mentorship of MD, PhD candidates during their research years: Mark Dubin, 6/2000 – 5/2001 Robert Burch 12/2000 – 12/2001
2002 – 2005	PhD Thesis Committee Member: Kuei Lim
2006 - 2007	Clinical Mentor, Laura Western, graduate student , Biomedical Engineering
2010	Invited Judge: Internal Medicine Resident Citywide Research Poster Award, Rochester General Hospital
2010-2014	Clinical Mentor, Rochester Institute of Technology/Rochester General Hospital Alliance - Title: Human Motion Tracking System Faculty Supervisor: Dr. Elizabeth DeBartolo Team Members: Brittany Bochette, Lindsey Clark, Michael Ostertag, Maya Ramaswamy, Andrei Stihi
2010-2014	Clinical Mentor, Rochester Institute of Technology/Rochester General Hospital Alliance - Title: Design of a Robotic Assist Device for Patients with Footdrop Faculty Supervisor: Dr Kathleen Lamkin-Kennard, Dr Elizabeth DeBartolo Student: Christopher Sullivan, MS student RIT 2011 RIT&RGHS Alliance Award $20,000
2010-2013	Community Career Advisor. Career Internship Program, Pittsford High Schools
2014	Clinical Mentor, Anette Stark, MD. Visiting Movement Disorders Resident, Denmark
2017	Clinical Mentor: Mika Naor. Sackler School of Medicine New York State/American Program of Tel Aviv University (Class of 2022)

Invited Lectures / Additional Teaching Activities

Aug 2024	Dystonia Medical Research Foundation, 'Ask the Experts' Teleconference National distribution.
10/2023, 6/2024	Movement Disorder Society Center-to-Center Program. Training program with University of Peru, Arequipa, Peru. Training of Peruvian neurologists
Jul 2022	"Unlocking Movement: Addressing Key Challenges and Strategic Solutions in the Use of Botulinum Toxin for Spasticity and Dystonia", with Katharine Alter, MD, and David Simpson, MD. CME Online panel discussion
Jan 2021	Lecture: "Guidance, Localization and Optimal Placement of Botulinum Toxin: Cervical Dystonia." International Neurotoxin Association: Toxins 2021. Virtual, Jan 16-17, 2021
Apr 2019	Lecture: 'An Overview of Existing and New Drug Therapies for Parkinson's Disease"; Parkinson Foundation of Greater Rochester Annual Symposium
Jun 2018	CME Lecture "Update in Movement Disorders", Ithaca, NY
Nov 2017	CME Lecture "Medical Treatment of Movement Disorders" Batavia, NY
Nov 2017	Living with Cervical Dystonia. Informational program for Patients, sponsored by Dystonia Medical Research Foundation and Allergan , NY, NY
Jun 2016	CME Lecture: "Update in Movement Disorders" Auburn Community Hospital

Jun 2016	Invited Lecture: “Parkinson’s Disease” Warsaw County Hospital Medical Staff Rounds
May 2016	Invited Lectures: “Dystonia” and “Case Presentations”. Symposium on the Update in Movement Disorders, Jagiellonian University, Krakow, Poland
Feb 2016	“Hemifacial Spasm and Bell’s Palsy” . Neurosurgery Board Review Series
Jan 2015	Invited Workshop Director: Practical Applications for Cervical Dystonia and Blepharospasm. International Neurotoxin Association, Lisbon, Portugal Jan14-17,2015
Apr 2014	Invited Lecture: “Parkinson’s Disease: Current Therapies” for Parkinson’s Disease Foundation Symposium for patients and families. Rochester, NY
Mar 2014	Invited Lecture: "Introduction to Dystonia and Its Treatment” for "Neurobiology and Neurology of Highly Skilled Motor Performance in Musicians" Symposium co-sponsored by the Schmitt Program on Integrative Brain Research and the University of Rochester Provost's Multidisciplinary Award Program
Jan 2013	Lecture: “ Movement Disorder Emergencies” . Rochester General Hospital, Department of Medicine Grand rounds
Oct 2011	Invited Lecture: “Musician’s Dystonia”. Rochester Institute of Technology, Osher Learning Center
Oct 2009	Invited Panelist: “H1N1: Special Edition” *Second Opinion*, WXXI Public Broadcasting ; * This episode won a Silver Communicators Award and a Bronze Telly Award
Jun 2009	CME lecture: "Current Management Strategies for Parkinson's Disease- A Case Based Approach." University of South Florida sponsor, Rochester NY
Apr 2009	Update in Neuropathic Pain, University of Rochester CME Course. Invited lecture: “Botulinum Toxins and Pain.”
Jan 2009	CME Lecture: “Neurology in the ED”. Rochester General Hospital Department of Emergency Medicine
Sep 2008	Lecture: “Pharmacology of Botulinum Toxins” Annual Meeting Rochester Area Society of Health-Systems Pharmacists.
May 2008	CME Lecturer: “Cranial Neuropathies” in *Advances in Neuromedicine*. Rochester Academy of Medicine
Mar-Jun 2008	Preceptor: Botulinum Toxin Cervical Dystonia Injection Center. Training extramural neurologists in botulinum toxin injection therapy. Briomed, Inc
Mar 2008	Grand Rounds, Dept of Medicine, Rochester General Hospital: “Clinical Uses of Botulinum Toxins”
Sep 2007,2008, 2011	Lecturer: “Critical Care Neurology” FCLS course: core curriculum for residents in medicine
Apr 2007	Grand Rounds, Dept of Neurology, University of Rochester: “Forensic Neurology.”
Mar 2007	Course Director: “Update in Movement Disorders”. University of Rochester CME. Lecture: “Botulinum toxin therapy for Movement Disorders”
Nov 2006	Instructor: Neuro Rehab Preceptor Program. Training extramural PM&R residents in botulinum toxin injections. Annenberg Center for Health Sciences, and Cognimed, Inc.
Apr 2006	Pain Management Update for the Primary Care Provider Invited Lecture: “Neuropathic Pain”
Nov 2005	Advances in the Management of Neuropathic Pain. CME Regional Lecture Series. Indianapolis, IN Invited Lecturer: “Understanding and Diagnosing Neuropathic Pain.”
Sep 2005	Director AAN/MDS Dystonia/Spasticity Workshop: *Advanced Treatment of Dystonia and Spasticity Workshop Demonstrating the Use of Botulinum Toxin.*
Sep 2005	CME Lecture: “Botulinum Toxin Type A- Headache Update 2005”
8-9 2005	Invited Lecturer: Joslin Diabetic Center CME: “Impacting Diabetic Neuropathies”. Washington, DC, Philadelphia, New York, Short Hills
Jun 2005	Treatment of Dystonia: Workshop Demonstrating the use of Botulinum Toxin. Invited Lecture: “Spasticity and Other Uses of Toxin.”
Nov 2004	Eleventh Annual International Diabetes Teaching Day: Diagnosis and Management of Diabetes. Invited Lecture: “Office Evaluation of Diabetic Peripheral Neuropathy”
Nov 2004	CME Lecture: “Evidence Based Medicine Review of Botulinum Toxin Therapy for Headache”
Oct 2004	National Spasmodic Torticollis Association Annual Meeting, Ypsilanti, MI Invited Lecture: “Maximizing Botulinum Toxin Therapy”
Sep 2003	Irish Institute of Clinical Neuroscience, Galway, Ireland. Update in Neurology

	Invited Lecture: "Neuropathic Pain: Pathophysiology and Management"
Jun 2003	Course Director: Migraine Update for Primary Care Providers: Practical Issues in Management. Lecture: "Migraine Preventive Strategies"
Oct 2002	Grand Rounds, Dept Of Physical Medicine and Rehab, University of Rochester. "Botulinum Toxin Therapy in the Management of Spasticity"
Sep 2002	Grand Rounds, Dept of Medicine, Jones Memorial Hospital "Clinical Uses of Botulinum Toxin Therapy"
Mar 2002	Grand Rounds, Dept of Medicine, Canandaigua VA Hospital "Clinical Uses of Botulinum Toxin Therapy"
3–6 / 2001	Invited Lecturer, Joslin Diabetes Center CME. "Impacting Diabetic Neuropathies"
Oct 2000	Invited Lecturer, Neuropathic Pain Conference, Beth Israel Medical Center CME. "Neuropathic Pain: Clinical features, pathophysiology and assessment"
Oct 2000	Section Director, Mind-Brain –Behavior Integration Course, University of Rochester School of Medicine. "Headaches"
Jun 2000	Grand Rounds, Dept. of Medicine, Canandaigua VA Hospital. "Migraine Management"
Apr 2000	Grand Rounds, Dept. of Geriatric Medicine, Monroe Community Hospital. "Common Botulinum Toxin Responsive Conditions in the Elderly"
Apr 2000	Invited Lecturer: "Low Back Pain" Greater Rochester Association of Neurologic Nursing
Mar. 2000	Grand Rounds, Dept of Medicine. Jones Memorial Hospital "Migraine Diagnosis & Management"
July 1999	Invited Lecturer: "Headaches". Wellness Lecture Series, Corning Glassware Co.
Feb. 1999	Invited Lecturer: "Diabetic Peripheral Neuropathy" Diabetes Support Group, Strong Memorial Hospital
Sept. 1998	Invited Speaker, "A Primer on Whiplash, Back Injury, and Carpal Tunnel Syndrome" NYSBA Annual Torts and Compensation Meeting
Oct. 1997	Invited Lecturer, "Neurology in the Courtroom" NY Bar Assoc.
1994, 1996	Invited Lecturer, The Charles E. Henry Society of Neurodiagnostic Technicians, Rochester, NY Chapter
Mar. 1995	Grand Rounds. Dept of Neurology, SMH "Attention Deficit - Hyperactivity Disorder"
1996	Grand Rounds, Department of Oral Surgery. "Evaluation of Headaches".
Dec. 1995	Course Lecturer, Office of Continuing Professional Education, University of Rochester. Neurology for the Primary Care Provider. "Low Back Pain."

Other Professional and Administrative Activities

2023- Present	MDS Center-to Center Training Grant. Dr Karlo Lizarraga, PI. Eight 1 week faculty visits for training, travel to Rochester NY and Arequipa, Peru
2021- present	Virtual Injector Trainer, ELATE: A clinical trial of botulinum toxin in Essential Tremor, Abbvie
2021- present	Ad Hoc reviewer, Neurology Clinical Practice
2020- present	Consultant, Abbvie/Allergan. Designing clinical trials of botulinum toxin for essential tremor
2019- 2023	Consultant, Oscine Corporation. Designing clinical trials of progenitor cells in Huntington Disease
2019-present	Ad hoc reviewer, Clinical Parkinsonism & Related Disorders
2019- present	Ad hoc reviewer, Toxins
2019	Group Leader, American Academy of Neurology Course: "Advanced Leadership Training: Preparing for Your Career's Insurmountable Opportunities"
2018- present	Ad hoc reviewer, Tremor and Other Hyperkinetic Disorders
2017-2021	Research Administrator: Lawler Foundation for Huntington Disease Research
Sep 2018	MDS-PAS Neuromodulation for Movement Disorders Course, Ottawa, CA
2018-present	Ad hoc reviewer, Journal of Neurological Sciences
2014- 2018	Strong Memorial Hospital Regional Planning Committee

2014 – 2018	URMFG Regional Operations Committee
2014-2016	Senior Leadership Education and Development Program. Certificate Awardee, June 2016. Project: Development and Implementation of Regional Network: Department of Neurology
2014-present	Neurology Section Editor, Visual Diagnostics. Online tool for clinicians
2014	Invited Movement Disorders Roundtable Member: Partnership to Improve Patient Care (PIPC) and the American Academy of Neurology (AAN) on issues specific to the dissemination and implementation agenda of the Patient-Centered Outcomes Research Institute (PCORI).
2014	Participant: RITE cadaver course, New York City
2013-2015	Fellowship Director, Movement Disorders Division, University of Rochester
2012- 2021	Associate Editor, Neurology: Clinical Practice. *winner of an Eddie Award for Editorial Excellence, category of Healthcare/Medical/Nursing, Full Issue for Neurology Clinical Practice, 3(2), 2013
2011-2020	Level of Evidence Rater, Neurology
2010-2011	Member, General Neurology Task Force, American Academy of Neurology
2010- present	Ad hoc reviewer, Movement Disorders
Oct 2009	Participant: AAMC Executive Leadership Course
2009-2015	Member, Guidelines Development Subcommittee (*former* Quality Standards Subcommittee) of the American Academy of Neurology
2008-2014	Interviewer, Medicine Residency Program, Rochester General Hospital
2007-2014	Quality Council, Rochester General Hospital
2007-2014	Medical Board, Rochester General Hospital
2007-2019	Executive Steering Committee, Department of Neurology, SMH
Mar 2007	American Academy of Neurology Leadership Development Program
2007	Ad hoc reviewer, Clinical Neurophysiology
2005-2011	Consultant, Allergan, Inc. Advisor on clinical trial protocol for new drug development.
2005	Ad hoc reviewer, Clinical Journal of Pain
2004-5	Ad hoc Reviewer, J Pain
2002	Invited Reviewer: American College of Physician Journal Club
2002 –2003	Research Grant Review Panel Member: American Diabetes Association. Biannual review of grant applications, neurologic complications of diabetes
2001-2002	Consultant, Pfizer Inc. Function as an external advisor for clinical trial protocol development.
2000-2007	Ad hoc Book Reviewer, Clinical J Pain
1999-2000	Medical Monitor, New York State Office of Professional Medical Conduct
1999- 2005	Ad hoc Book Reviewer, Neurology
1999	Internal Editor, Continuing Medical Education publication, Migraine Pain: Management of Migraine With The Triptans
1998 -1999	Examiner, American Board of Psychiatry & Neurology
1998	Ad hoc Book Reviewer, Journal of Neuro-Ophthalmology
1998	Member, Neurovascular Center Planning Committee
1997 – 2003	Member, Clinical Steering Committee, Department of Neurology
1997 -Present	Ad hoc reviewer for Neurology
1996 -Present	Forensic Neurology: Expert Witness. Independent Medical Examinations and Reviews of neurological cases involved in litigation.
1996-1998	Departmental Representative, Practice Management Committee
1996-1997	Departmental Representative, Council of the Medical Staff
1994-2004	Director, University of Rochester Neurology (faculty practice)

1993-1995 Interviewer, Office of Admissions, UR School of Medicine

Funded Research

Patient Centered Outcome Project for patients with cervical dystonia, blepharospasm and laryngeal dystonia. Site PI Sponsored by Dystonia Coalition and Rare Diseases Clinical Research Network. 2023-present

A Phase 2 Multicenter, Randomized, Double-blind, Placebo-controlled Study of BOTOX (Botulinum Toxin Type A) Purified Neurotoxin Complex for the Treatment of Upper Limb Essential Tremor (ELATE). Independent Rater. Funded by Abbvie, Inc. 2022-present

Assessing health Risks and Outcomes for Attorneys: Cluster PD. Site Investigator. E Ray Dorsey, MD PI. Funded by University of Rochester. 2021-2024.

Parkinson's Progression Marker Initiative. Site Investigator. RB Schneider, MD, Site-PI. Funded by Michael J Fox Foundation 2019- present.

Registry for the Advancement of Deep Brain Stimulation in Parkinson's Disease (RAD-PI). Site PI. Funded by NeuroPoint Alliance. 2019-2021

A Phase 3, Randomized, Double-Blind, Placebo-Controlled, Parallel Group,Multi-Center Trial to Evaluate the Long Term Safety and Efficacy of a Single Treatment of DaxibotulinumtoxinA for Injection in Adults with Isolated Cervical Dystonia (ASPEN-1). Protocol 1720302. Site PI. Funded by Revance Therapeutics, Inc: 2018-2021

Validation of Fox Insight Cohort via Virtual Research Visits (FIVE). Site Sub-I. Funded by Michael J Fox Foundation: 2018-2021

A Phase 3, Open-Label, Multi-Center Trial to Evaluate the Long Term Safety and Efficacy of Repeated Treatments of DaxibotulinumtoxinA for Injection in Adults with Isolated Cervical Dystonia (ASPEN-OLS). Protocol 1720304. Site PI. Funded by Revance Thearpeutics, Inc: 2018-2021

Management of Parkinson's Disease psychosis in actual practice (The INSYTE study). Protocol ACP-NIS-001. Site PI. Funded by Acadia Pharmaceuticals Inc. 2018 - 2021

Clinician-input study: how the Fox Insight mobile application can influence treatment and care (CIS-PD). Site SubI. Funded by Michael J Fox Foundation: 2016-2018

A Phase 3, Multicenter, Randomized, Double-Blind, Placebo-Controlled Study with an Open-Label Phase to Determine the Efficacy and Safety of Tozadenant as Adjunctive Therapy in Levodopa-Treated Patients with Parkinson's Disease Experiencing End-of-Dose "Wearing-Off" (TOZ-PD). Site PI. Funded by Biotie Therapies. Protocol TOZ-CL05. 2015-2017

A Phase 2 multi-center, randomized, double-blind, placebo-controlled study in subjects with late prodromal and early manifest Huntington's disease (HD) to assess the safety, tolerability, pharmacokinectics and efficacy of VX15/2503 (SIGNAL). Site PI. Funded by Vaccinex, Inc. Protocol VX15/2503-N-131. 2015-2021

Vestibular Changes in Cervical Dystonia and the Potential Effects of Botulinum Toxin. Mentor to K Andrzejewski, PI. Dystonia Medical Research Foundation Young Investigator Award. $75,000. 7/1/15-6/30/16.

Remote Access to Care Everywhere (RACE-PD). Site Rater. Ray Dorsey, PI. Funded by Davis Phinney Foundation Clinicaltrials.gov ID:NCT02144220. 2014-2016

Dystonia Coalition Projects: Funded by National Institute of Health (NIH), National Institute of Neurological Diseases and Stroke (NINDS), and Office of Rare Disorders Research (ORDR)

(1) Natural History and Biospecimen Repository for Dystonia. Site PI. Perlmutter, J, Project Leader
2012-present

(2) Comprehensive Rating Tools for Cervical Dystonia. Site PI. Comella, C, Project Leader
2012-2013

Recruitment and Sample Collection for Antecedent Biomarker Discovery in Parkinson's Disease. Sub Investigator. Funded by Department of Defense (DOD) Telemedicine and Advanced Technology Research Center (TATRC). Kurlan, R, and Federoff, H, PIs. 2011- 2013

Design of a Robotic Assist Device for Patients with Footdrop. Clinical Research Mentor.Faculty Supervisor: Dr Kathleen Lamkin-Kennard, Dr Elizabeth DeBartolo; Student: Christopher Sullivan, MS student RIT. 2011 RIT&RGHS Alliance Award $20,000. 2011- 2012

CD Probe: Cervical Dystonia Patient Registry for Observation of Botox Efficacy. Site PI. Funded by Allergan. Protocol Med Aff BTX 0718. 2009-2012

Longitudinal Studies of the Variable Phenotypic Presentations of Rapid-Onset Dystonia-Parkinsonism and Other Movement Disorders. Site Investigator. Funded by NIH to A Brashear, PI. 2009-2015

Prospective, double blind, placebo-controlled, randomized, multi-center trial with a double-blind parallel group Extension Period to investigate the efficacy and safety of different doses of NT 201 in the treatment of cervical dystonia. Protocol MRZ 60201-0408. Site PI. Funded by Merz. 10/2006-2010.

A Multi-Center, Double-Blind, Randomized, Parallel Group, Placebo-Controlled Trial of Ethyl-EPA (Miraxion) in Subjects with Mild to Moderate Huntington's Disease. Protocol AN01.01.0011. Sub-Investigator. Funded by Amarin Neuroscience Ltd. 2005-2009.

A 13-Week, Double-Blind, Placebo-Controlled Phase 4 Trial of Pregabalin (CI-1008, 600 mg/day) for Relief of Pain in Subjects with Painful Diabetic Peripheral Neuropathy. Protocol A0081060. Site PI. Funded by Pfizer. 2004-2006

An Open-Label Extension Safety Trial of Pregabalin (CI-1008) in Subjects with Painful Diabetic Peripheral Neuropathy. Protocol A0081036. Site PI. Funded by Pfizer. 2004-2006.

A Multicenter, Double-Blind, Randomized, Placebo-Controlled, Parallel Study of the Safety and Efficacy of Botulinum Toxin Type A Purified Neurotoxin Complex in Subjects with Postherpetic Neuralgia. Protocol 191622-066. Site PI. Funded by Allergan. 2004-2005.

Placebo Controlled Trial of Botox versus Zanaflex for the Treatment of Subjects with Post-stroke Upper Limb Spasticity. Site PI. Funded by Mt Sinai School of Medicine. 2002-2006.

A Multicenter, Randomized, Placebo-Controlled, Double Blind, Parallel-Group Trial to Evaluate Early Efficacy and Tolerability of Zolmitriptan Nasal Spray in the Acute Treatment of Adult Subjects with Migraine. Site Co-PI. Funded by AstraZeneca Pharmaceuticals. 2002-2003.

A Prospective, Open-Label Trial of Lidocaine Patch 5% (Lidoderm) in Painful Neuropathies. Co-Principal Investigator, with Robert H Dworkin, PhD. Funded by Endo Pharmaceuticals. 2001 -2003.

Botulinum Toxin Type A Compared to Botulinum Toxin Type B in Cervical Dystonia. A Randomized, Multicenter, Double-Blind, Parallel Group Study Comparing Botox (Botulinum Toxin Type A) and Myobloc (Botulinum Toxin Type B) in Cervical Dystonia (CD) Subjects Responsive to Botulinum Toxin Type A. (Protocol # DSG 2000R01). Site PI. Developed and Implemented by the Dystonia Study Group supported by an unrestricted grant from Allergan. 2001 – 2004.

Shingles Trial of Oxycodone to Prevent PHN (STOMP-PHN). A clinical trial planning grant.
Co-Principal Investigator. Funded by NIH, grant # R21 NS40685. 2001 –2005.

A Double-Blind, Placebo-Controlled, Parallel Group Study To Evaluate the Effect of Topiramate on Electrophysiological Parameters in Subjects with Diabetic Peripheral Polyneuropathy. (Protocol TOPMAT-NP-005).

Site PI. The RW Johnson Pharmaceutical Research Institute. 2001 – 2002.

A Double-Blind, Placebo-Controlled, Parallel Group, Dose Ranging Study to Evaluate the Efficacy and Safety of Topiramate versus Placebo in the Relief of Pain in Diabetic Peripheral Neuropathy. Site Principal Investigator. R.W.Johnson Pharmaceutical Research Institute TOPMAT-NP-001. 1999-2000.

A 7-Week, Randomized, Double-Blind, Placebo-Controlled, Parallel-Group Study of Pregabalin in Patients with Chronic Low Back Pain (Protocol 1008-032). Site PI. Parke-Davis. 1998.

Pregabalin Open-Label, Extension Safety Trial in Patients with Chronic Pain (Protocol 1008-033). Site PI. Parke-Davis. 1998- 2001.

Placebo controlled, randomized trial of amifostene in the prevention of paclitaxol induced neuropathy. Co-Investigator. Jennifer Griggs, MD, PI. Funded by Alza Pharmaceuticals. 1997-1999.

Topiramate Monotherapy Clinical Trial in Subjects with Recently Diagnosed Partial Onset Seizures. (RWJ-17021-000) Site PI. Sponsored by Robert Wood Johnson Pharmaceutical Research Institute. 1997-2003.

Trial of Recombinant Human Nerve Growth Factor in Diabetic Peripheral Neuropathy. Phase III Study. Site PI, funded by Genentech. 1997 - 1999

Trial of Recombinant Human Nerve Growth Factor in Diabetic Peripheral Neuropathy. Phase II Open Label. Site PI. Funded by Genentech. 1997- 1999

Recombinant Human Nerve Growth Factor in HIV Associated Neuropathy. ACTG 291 Sub-Investigator. NIH funded (PI: Karl Kieburtz, MD). 1996- 1998

Trial of Recombinant Human Nerve Growth Factor in Diabetic Peripheral Neuropathy; Double-Blind, Placebo-Controlled Phase II. Site Co-PI. Funded by Genentech. 1995-1996

Publications

Abstracts/ Posters/ Platform Sessions

Globus pallidus oscillatory activity correlates with symptom improvement in patients with Parkinson's disease and dystonia. Amudhan A, Sell D, Barbano RL, et al. Finger Lakes Neuromodulation Conference. Rochester Oct 2024.

A Phase 2b Multicenter, Randomized, Double-Blind, Placebo-Controlled Study of OnabotulinumtoxinA for the Treatment of Upper Limb Essential Tremor: ELATE Trial in Progress. Barbano R, Simpson D,Patterson K, Alibhai N, James L. International Neurotoxin Association, 7th Annual Meeting, Berlin, Germany Jan 2024.

Incidence of dysphagia and comorbidities in patients with cervical dystonia, analyzed by botulinum neurotoxin treatment exposure. Sadeghi M, Ukah A, Yue EX, Ifantides KB, Huang NY, Lee, J Barbano R. International Neurotoxin Association, 7th Annual Meeting, Berlin, Germany Jan 2024

Dry cleaning chemicals and Parkinson's Disease. Pawlik ME, Lettenberger SE, Zafar M, et al. International Congress of Parkinson's Disease and Movement Disorders. Copenhagen, DE Aug 27-31, 2023

Efficacy of DaxibotulinumA for injection over successive treatments in adults with isolated cervical dystonia in the phase three ASPEN-1 and ASPEN-OLS trials. Comella C, Barbano R, Rudzinska M, et al. American Association of Physiatry Annual Meeting. Anaheim , USA Feb 21-24, 2023

Head tremor jerkiness in cervical dystonia: clinical and computer vision assessments. Cisneros E, Le L, Vu JP, et al. 2nd International Tremor Congress. NY, USA May 18-19, 2023

Clusters of Parkinson's Disease may be linked to widely used dry cleaning solvents. Pawlik ME, et al. International

Congress of Parkinson's Disease and Movement Disorders. Madrid, Spain Sep 14-18, 2022

Impact of disease severity on presentation subtype and Onabotulinum toxinA utilization on patients with cervical dystonia: Results from the CD Probe completer population. International Neurotoxin Association Annual Meeting, New Orleans, LA, Jul 2022

Benefits of treatment with Onabotulinum toxinA in naïve and non-naïve pateints with cervical dystonia are sustained over time in CD-PROBE. P Agarwal, M Schwartz, A Zuzek, et al. Canadian Neurological Sciences Federation Congress, Toronto, Canada Oct 25-28, 2021

Impact of Disease Severity on Presentation Subtype and Onabotulinum ToxinA Utilization in Patients with Cervical Dystonia: Results from the CD PROBE Completer Population. Agarwal P. Barbano R, Moore H, et al. International Parkinson and Movement Disorder Society. Virtual, Sep 17-22, 2021

Head Tremor in Cervical Dystonia: the effect of postural maneuvers. E Cisneros, JP Vu, Q Chen, et al. Society for Neuroscience 2020 Annual Meeting. Washington, DC, Oct 2020

Validation of Fox Insight Cohort via Virtual Research Visits. TL Myers, RB Schneider, M Daeschler, et al. American Academy of Neurology 72nd Annual Meeting, Toronto, CA , April 2020

Stiff limb syndrome masquerading as a focal limb dystonia. PE Morrison, RL Barbano. International Congress of Parkinson's Disease and Movement Disorders. Nice, FR Sep 22-26, 2019

Genome-wide association study identifies common genetic variants associated with cervical dystonia. GK Berkman, R Barbano, Y Sun, H Jinnah. International Congress of Parkinson's Disease and Movement Disorders. Hong Kong, Oct 4-8, 2018

Dystonia, Tremor and Dystonic Tremor. AG Shaikh, AR Rosen, LM Scorr, A Cotton, RL Barbano, C Testa, HA Jinnah for the Dystonia Coalition Investigators . International Congress of Parkinson's Disease and Movement Disorders. Hong Kong, Oct 4-8, 2018

Cervical dystonia and substance abuse. A Mahajan, N Patel, J Jankovic, L Marsh, H Jinnah, C Comella, R Barbano, J Perlmutter. American Academy of Neurology 70th Annual Meeting, Los Angeles, April 2018

Levodopa-carbidopa intestinal gel therapy (LCIG) choices and clinical outcomes in DBS-eligible versus palliative stage PD patients. M.A. Burack, A. Santiago, K. Biglan, R. Barbano. 30th Annual Symposium on Parkinson Disease and Other Movement Disorders. Portland, OR Sept 19, 2016

Assessing Vestibular Function in Individuals with Cervical Dystonia and the Effects of Botulinum Toxin Treatment Kelly L. Andrzejewski, *et al*. 20th International Congress of Parkinson's Disease and Movement Disorders. Berlin, Germany June 19-23,2016

Effectiveness of OnabotulinumtoxinA in Patients with Cervical Dystonia Naïve to Botulinum Toxin Treatment. C. Singer, R Barbano, M Schwartz, *et al*. 19th International Congress of Parkinson's Disease and Movement Disorders. San Diego, CA June 14-18,2015

Inter-rater reliability of the severity subscale of the revised Toronto Spasmodic Torticollis Rating Scale (TWSTRS-2). C Comella, MD *et al*. Platform Session. American Academy of Neurology 67th Annual Meeting. Washington, DC. April 18-23, 2015

Reliability and validity of the revised TWSTRS psychiatric module (TWSTRS-PSYCH) of the Comprehensive Cervical Dystonia Rating Scale (CCDRS). Mateusz Zurowski, MD, *et al.* 18th International Congress of Parkinson's Disease and Movement Disorders. Stockholm, Sweden. June 8-12, 2014

Convergent validity of the revised TWSTRS modules of the Comprehensive Cervical Dystonia Rating Scale (CCDRS). Cynthia L. Comella, M.D., *et al.* 18th International Congress of Parkinson's Disease and Movement

Disorders. Stockholm, Sweden. June 8-12, 2014

Clinimetric testing of the modules of the Comprehensive Cervical Dystonia Rating Scale (CCDRS). C. Comella, *et al*. Platform presentation. American Academy of Neurology 66th Annual Meeting, Philadelphia, PA April 26-May3, 2014

Design of a Robotic Assist Device for Patients with Footdrop. Clinical Research Mentor. Faculty Supervisor: Dr Kathleen Lamkin-Kennard, Dr Elizabeth DeBartolo; Student: Christopher Sullivan, MS student RIT. RIT-RGH Alliance Poster Session Apr 2011

Tizanidine Improves Geriatric Depression Scale Scores in Stroke and Brain Injury Survivors with Spastic Hemiparesis: Post−Hoc Analysis from a Randomized, Double−Blind, Placebo−Controlled Trial. S Yablon, JM Gracies, DM Simpson, A Brashear, R Barbano, P Raghavan. American Academy of Neurology 62nd Annual Meeting, Toronto, ONT, Canada. Apr 2010.

Variable Phenotypic Expression of Rapid Onset Dystonia Parkinsonism in a Newly Discovered Italian Family. Barbano RL, Ozelius L, Hill DF, Brashear A. American Academy of Neurology 61st Annual Meeting, Seattle WA Apr 2009

Deep brain stimulation for tardive dyskinesia and akathisia. C Kenney, RL Barbano, JK Sheffield, J Jankovic. 11th International Congress of Parkinson's Disease and Movement Disorders. Istanbul, Turkey. June 3-7,2007

Botulinum Neurotoxin vs Oral Tizanidine in the Treatment of Upper Limb Spasticity: A Double-Blind, Placebo-Controlled Study. DM Simpson, JM Gracies, S Yablon, R Barbano, A Brashear, and the BoNT/Tiz Study Team. American Academy of Neurology Annual Meeting. Boston MA May 2007

Utility of Adjunctive Electromyography in Botulinum Toxin Injection for Cervical Dystonia.
Barbano, RL, Comella C, Fan W, Leurgans S and the Dystonia Study Group
The Movement Disorder's Society: 9th International Conference of Parkinson's Disease and Movement Disorders. New Orleans, LA, USA March 5-8, 2005

A Randomized, Multicenter, Double-Blind, Parallel Study Comparing Botulinum Toxin Type A (BOTOX®) and Botulinum Toxin Type B (MyoBloc™) in Cervical Dystonia. Cynthia L. Comella, MD,[1] Joseph Jankovic, MD,[2] Sue Leurgans, PhD,[1] Frederick Marshall, MD,[3] Kathleen M Shannon, MD,[1] Michael R. Swenson, MD,[4] Joseph Tsui, MBBS,[5] for the Dystonia Study Group*. American Neurological Association Annual Meeting. Toronto, October 2004

Effectiveness of Lidocaine Patch 5% in Diabetic Neuropathy Patients With or Without Allodynia. RL Barbano, DN Herrmann, BS Galer, AR Gammaitoni, S Hart-Gouleau, J Pennella-Vaughan, J Domingos, RH Dworkin. 6th International Conference on Mechanisms and Treatment of Neuropathic Pain. San Francisco, September 2003

Effectiveness of Lidocaine Patch 5% in Idiopathic Sensory Polyneuropathy. RL Barbano, DN Herrmann, BS Galer, AR Gammaitoni, S Hart-Gouleau, J Pennella-Vaughan, J Domingos, RH Dworkin. 6th Intrnational Conference on Mechanisms and Treatment of Neuropathic Pain. San Francisco, September 2003

Results of a Double-Blind, Placebo-Controlled Trial of Recombinant Human Nerve Growth Factor in Diabetic Polyneuropathy. SC Apfel, BT Adornato, PJ Dyck, JA Kessler, A Vinik, M Rendell, RC Griggs, RL Barbano, C Rask, and the NGF Study Group. Ann Neurology 1996, 40:T194.

Discriminant validity of self-rating scales and neuropsychological assessment in diagnosing adult ADD. National Academy of Neuropsychology Annual Conference. D Palumbo, J Porter, P Como, RL Barbano, D Giang. San Francisco, 1995

Clinical characteristics of adults referred to an attention deficit disorder clinic. D Palumbo, RL Barbano, P Como, D Giang, and S Silverstein. NY State Office of Mental Health Research conference, 1994

Peer Reviewed Publications

Thomsen M, Ott F, Loens S, et al. Genetic diversity and expanded phenotypes in hereditary dystonia: Insights from large-scale exome sequencing. medRxiv [preprint] 2024 Dec 5. PMID: 39677454

Chunga N, Minks K, sell DL, et al. A novel PDE8B gene variant associated with autosomal dominant striatal degeneration. Mov Disorders Clin Pract. 2024; 11(8): 1044-46. PMID: 38818539

Dorsey ER, Kinel D, Pawlik ME, et al. Dry-Cleaning chemicals and a cluster of Parkinson's disease and cancer: A retrospective Investigation. Mov Disorders 2024; 39: 606-613. Doi 10.1002/mds.29723. PMID: 38389433

Comella CL, Jankovic J, Hauser RA, et al. Efficacy and safety of DaxibotulinumtoinA for injection in cervical dystonia: ASPEN-1 Phase 3 Randomized control trial. Neurology 2024; 102;e208091. PMID: 38295229

Vu JP, Cisneros E, Zhao J, et al. From null to midline: changes in head posture do not predictably change head tremor in cervical dystonia. Dystonia 2022; 1:10684. Doi:10.3389/dyst.2022.10684. PMID: 37101941

Agarwal P, Barbano R, Moore H, et al. OnabotulinumtoxinA dosing, disease severity, and treatment benefit in patients with cervical dystonia: A cohort analysis from CD Probe. Front Neurol. 2022; 13:914486. Doi:10.3389/fneur.2022.914486. PMID: 35847221

Feigin A, Evans EE, Fisher TL, et al. Safety and efficacy of pepinemab antibody blockade of SEMA4D in patients with early Huntington's Disease: A randomized, placebo-controlled, multicenter, Phase 2 clinical trial (SIGNAL). Nature Med 2022; 8:1-11. Doi: 10.1038/s41591-022-01919-8. PMID: 35941373

Scorr LM, Choo HJ, Kilic-Bermen G, et al. Clinical features and evolution of blepharospasm: A multi-center international cohort and systemic literature review. 2021 Dystonia 2022; 1: 10359. doi:10.3389/dyst.2022.10359. PMID: 36248010

Zhang, Z, Cisneros E, Lee, HY, et al. Hold that pose: Capturing cervical dystonia's head deviation severity from video. Ann Clin Translation Neurol 2022; 9(5): 684-694. doi:10.1002/acn3.51549. PMID: 35333449

Vu JP, Cisneros E, Lee HY, et al. Head tremor in cervical dystonia: Quantifying severity with computer vision. J Neurol Sci 2022; 434: 120154. Doi:10.1016/j.jns.2022.120154. PMID: 35101766

Scorr LM, Factor SA, Parra SP, et al. Oromandibular Dystonia: A Clinical Examination of 2,020 Cases. Front Neurol. 2021 Sep 16;12:700714. doi: 10.3389/fneur.2021.700714. PMID: 34603182; PMCID: PMC8481678.

Myers TL, Tarolli CG, Adams JL, et al. Video-based Parkinson's disease assessments in a nationwide cohort of Fox Insight participants. Clin Park & Relat Disord 2021 doi:10.1016/j.prdoa.2021.100094. PMID: 3431667

Kilic-Berkman G, SP Richardson, JS Perlmutter, et al. Current Guidelines for classifying and diagnosing cervical dystonia: Empirical evidence and recommendations. Mov Disorders Clin Pract 2021 9(2): 183-190. DOI: 10.1002/mdc3.13376. PMID: 35146058

Wadon ME, Bailey GA, Yilmaz Z, et al. Non-motor phenotypic subgroups in adult-onset idiopathic, isolated, focal cervical dystonia. Brain Behav. 2021 Aug;11(8):e2292. doi: 10.1002/brb3.2292. Epub 2021 Jul 21. PMID: 34291595; PMCID: PMC8413761.

Sun YV, Li C, Hui Q, et al. A multi-center genome-wide association study of cervical dystonia. Mov Disord 2021 28July2021. 36(12):2795-2801. Doi.org/10.1002/mds.28732 PMID 34320236

Vu JP, Lee HY, Chen Q, et al. Head tremor and pain in cervical dystonia. J Neurol. 2021 May;268(5):1945-1950. doi: 10.1007/s00415-020-10378-5. Epub 2021 Jan 8. PMID: 33417005; PMCID: PMC8076053.

Shaikh AG, SB Beylergil, L Scorr, et al. Dystonia and Tremor: A Cross-Sectional study of the Dystonia Coalition

Cohort. Neurology 2021 Jan 26;96(4): e563-574. PMID: 33046615

Cisneros E, Vu JP, Lee HY, et al. Does Raising the Arms Modify Head Tremor Severity in Cervical Dystonia? Tremor Other Hyperkinet Mov (NY). 2021 Jun 23;11:21. doi: 10.5334/tohm.623. PMID: 34221696; PMCID: PMC8231450.

Cisneros E, Stebbins GT, Chen Q, et al. It's tricky: Rating alleviating maneuvers in cervical dystonia. J Neurol Sci 2020 Dec 15;419:117205. PMID: 33160248

Norris, SA, Hyder AJ, Klein C, et al. Clinical and demographic Characteristics of Upper limb Dystonia. Mov Disorders 2020. 35(11): 2086-2090. PMID: 32845549

Chen Q, Vu JP, Cisneros E, Benadof CN, et al. Postural Directionality and Head Tremor in Cervical Dystonia. Tremor Other Hyperkinet Mov (NY). 2020 Jan 20;10. doi: 10.7916/tohm.v0.745. PMID: 32015932; PMCID: PMC6988138.

Berman BD, CL Groth, SH Sillau, et al. Risk of spread in adult-onset isolated focal dystonia: a prospective international cohort study. J Neurol Neurosurg Psych 2020 Mar 91 (3):314-320. PMID 31848221

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