Exhibit 529

Richard L Barbano, MD, Ph.D, PLLC
Diplomat of American Board of Psychiatry and Neurology
Diplomat of American Board of Electrodiagnostic Medicine
New York State License # 188639

Expert Witness Report: Edgar Peterson IV

I. Table of Contents

II. Personal Background/Qualifications

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, 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 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 at 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 physician 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 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

Following my review of Mr. Edgar Peterson's medical records, relevant deposition testimony, general causation reports, zoom interview of Mr. Peterson, medical literature review, and in conjunction with my personal neurological knowledge and experience, I offer the following core opinions:

1. Mr. Edgar Peterson suffers from Parkinson's disease. This opinion is stated to reasonable degree of medical certainty. The basis and support for this opinion is set out in detail below.

2. To a reasonable degree of medical certainty, Mr. Peterson's exposure to the water at Camp Lejeune is "at least as likely as not" a cause of his Parkinson's disease due to his exposure to TCE and PCE, which is the standard set forth in the CLJA. His exposure to known neurotoxic chemicals such as TCE is at least as likely as not the cause of his Parkinson 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 other potential causes of Parkinson's disease other than Mr. Peterson's exposure to the contaminated water at Camp Lejeune and is that none are substantial contributors.
3. Parkinson's Disease is a chronically progressive disease without any cure. Mr. Peterson'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 which 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. Peterson's Parkinson's disease and then, based on the strength of the evidence, excluded other potential causes to determine if the most likely cause or causes for his Parkinson's disease. In the present case, the exposure to the water at Camp Lejeune remained the only cause within my differential etiology. Mr. Peterson's exposure to the water at Camp Lejeune was substantial:

Exposure dates include May 16, 1975 to June 15, 1977.[1] His estimated total months of exposure of approximately 25 months from living and working at Camp Lejeune.

The substantial exposure to the water at Camp Lejeune combined with the lack of strength of any other substantial causal factor, allows me to conclude that Mr. Peterson's Parkinson's disease was at least as likely as not caused by his exposure to the water at Camp Lejeune.

The evidence I reviewed consists of the following:

A. Medical Records

[1] *01576_PETERSON_NARA_0000000085;01576_PETERSON_NARA_0000000230, 01576_PETERSON_NARA_0000000052; 01576_PETERSON_0000000621*

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:

- Joseph Allen, MD
- Tulio Bertorini, MD
- Veterans Administration
- Methodist Hospital
- Eugene C. Lai, MD
- Methodist Neurologic Institute
- Simpson, MD
- Lakeside Behavioral Health System
- Vanderbilt University
- Marc LeDoux, MD
- Semmes-Murphy Clinic
- Vishad Kumar, MD
- Shawn Hayden, MD

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 of Edgar Allen Peterson, 2/8/2024
Deposition of Lori Peterson, 4/16/2024

Deposition of Mr. Jerry Potter was reviewed in regard to Mr. Peterson's work history.

Deposition of Jerry Potter, 6/3/2024

Depositions of treating physicians, Dr. Tulio Bertorini, Dr. Joseph Allen and Dr. Vishad Kumar 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 of Tulio Bertorini, MD, 5/1/2024
Deposition of Vishad Kumar, MD, 6/4/2024

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 or parkinsonism and exposure, causative factors, etiology, toxins, head trauma, genetics, or 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 from Drs. Boehme, DeMiranda, Miller, Cannon, Costa, Freeman, and Bird were reviewed, along with an exposure report from Dr. Reynolds. This information was considered as to whether it supports or does not support the opinion of causation of Parkinson Disease in the case of Mr. Peterson.

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 and CNN New Interview

I conducted a zoom interview with Mr. Edgar Peterson on January 14, 2025. He was at his home. He was interviewed in the presence of his wife, Lori, with his permission. He used an electronic voice producer for communication. Several points of clarification regarding his medical records were reviewed, with note of possible Parkinson's contributors during his lifetime. The following information was obtained via the Zoom interview:

In regard to his family history, there is no family history of Parkinson's disease in his nuclear family, nor any in his extended family that he is aware of. His father had prostate cancer. One brother died of liver failure as a secondary complication of HIV; one sister died

of leukemia. He has two paternal uncles, one maternal uncle and two maternal aunts; none of whom have Parkinson's disease.

He reports that prior to the peri-onset period of Parkinson's disease, Mr. Peterson had no history of prior depression.

He denies any significant paint exposure, with no commercial paint and only residential use of paints after 1978.

Mr. Peterson is able to give details regarding the head injury he had while playing football at 26 years old during officers training. He reports that he hit his head against a cinder block wall. He reports that he blacked out for 15 to 30 seconds but immediately afterwards was walked to the sick bay where he was instructed to drive himself 10 miles to a hospital and back. He reports that he had no problems doing this and had no headaches after the incident. This indicates that head injury was not substantial and not a contributing factor in Mr. Peterson developing Parkinson's Disease.

Mr. Peterson was observed in his motorized wheelchair. He had taken his Parkinson's medications 5 minutes prior to our zoom interview and he considered himself on the "cusp of OFF." During this time, his exam was notable for mild truncal dyskinesias and severe dysarthria to the point of being unintelligible. He had extreme hypomimia with decreased expression, decreased blinking, and his mouth was open at rest. There was no resting tremor. He had sialorrhea and needed to frequently wipe his mouth.

Approximately 20 minutes after taking his medications he had significant truncal dyskinesias, including his head, with significant movement in his wheelchair. Examination of his arm movements were notable for bradykinetic and this rhythmic small finger taps, left more so than right. Hand opening speed was slow bilaterally with decrementing within the first 2 movements on the right and within the first 5 on the left. Pronation supination was slowed bilaterally left more so than right. There was no ataxia, and he was able to use his keyboard for communication.

A CNN interview dated 8/11/2023 was also reviewed on the Internet. His speech was more clear and less impacted by dyskinesia at that time. This is consistent with the progression of Parkinson's Disease and is evident that Mr. Peterson is in late-stage Parkinson's and will continue to deteriorate.

F. Additional Records

The following additional records were reviewed:

Military Records of Edgar Peterson
ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases, 1/13/2017

VI. Discussion of Opinions

Diagnosis

Mr. Peterson has Parkinson's disease. Mr. Peterson has a history of cervical spine spondyloarthropathy with laminectomy which affects gait and balance, but these comorbidities do not cause the constellation of symptoms consistent with the diagnosis of Parkinson's disease. Accordingly, his medical records confirm, and are consistent with, his diagnosis of Parkinson's disease.

At about 52 years old, Mr. Peterson was noted to have tremors of his left leg *(see note 10/26/00; Peterson_0000002312)*, prompting Dr. Allen to refer Mr. Peterson to a neurologist for a second opinion. (*Peterson_0000002312*). On 5/22/01 he was seen by neurology. (*Peterson_0000002571-0000002573*). At that time, history was obtained that he had been 'slower', initially felt to be due to depression. He was also noted to have had rest tremor of his left hand and foot, a monotonous voice, a 'masked' face with decreased eye blink with a positive glabellar reflex, decreased mobility of his left arm, rigidity of his right arm with contralateral reinforcement, and decreased arm swing while walking. (*Peterson_0000002571-0000002573*). In retrospect, it was reported that he may have had symptoms as early as 6 years prior, although those symptoms are not specified (*Cooper, 4/28/09*). (*Peterson_0000002668-0000002679*).

The symptoms mentioned in 2000 and 2006 are evidence of Parkinson's disease. The differential etiology producing this constellation of the sum of these signs is extremely limited to other 'atypical parkinsonism' and drug-induced parkinsonism. Subsequent history goes on to confirm this diagnosis of Parkinson's disease and makes the atypical parkinsonisms unlikely. This subsequent history includes a notable response to a trial of low dose carbidopa levodopa. This was noted by Dr Bertorini in follow-up on 7/6/01: "I told him that he most likely has Parkinson's disease." (*Peterson_0000002574*). This diagnosis was reported as agreed with in a second opinion by Dr Pfeiffer (*see note of 2/15/02; Peterson_0000002578*). Mr. Peterson was subsequently evaluated by several other neurologists (Drs Lai, Cooper, LeDoux, and Kumar), all of whom agreed with the diagnosis. (See Medical Records, generally).

None of the atypical parkinsonisms fit the medical records of the ensuing 20-plus years of Mr. Peterson's history. The average lifespan of patients with the atypical parkinsonisms that could be confused with Mr. Peterson's current syndrome is significantly less than his current 20-plus years of disease. Furthermore, he had a robust and sustained response to levodopa therapy, eventually inducing dyskinesias, which the atypical parkinsonisms do not. On examination, he continued with persistent side-to-side asymmetry of his symptoms, another feature that would be inconsistent with the atypical

parkinsonisms. Based upon the above, and upon my education, training and experience, I can rule out atypical parkinsonisms as the cause of Mr. Peterson's problems.

In regard to drug-induced parkinsonism, based upon my review his medical records, depositions, and my discussion with Mr. Mr. Peterson, he was not taking, nor does he have a history of taking, any of the medications associated with this Parkinson's disease or parkinsonism. Based upon my investigation and review of the above referenced materials, and upon my education, training and experience, I can rule out drug induced parkinsonism in this case.

The zoom interview with Mr. Peterson on January 14, 2025 evidenced his continued symptoms consistent with the diagnosis of Parkinson's disease. I observed that Mr. Peterson had severe dysarthria, hypophonia, hypomimia, asymmetric bilateral bradykinesia and clear time-linked levodopa induced dyskinesia. Given his history over 20 years this is unlikely to be anything but Parkinson's disease. (*See* Medical Records, generally).

Mr. Peterson meets the International Movement Disorders Society criteria for clinically probable Parkinson's disease (*Postuma, R, et al. Movement Disorders 2015, 30: 1591- 601*). It must be stressed that these criteria are the typically more stringent criteria for use in clinical research.

Per the MDS 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. MDS Clinical Diagnostic Criteria for PD—Executive Summary

(Postuma et al, 2015)

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 (e.g., 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 y of disease.*
4. *Parkinsonian features restricted to the lower limbs for more than 3 y 5. Treatment with a dopamine receptor blocker or a dopamine-depleting agent in a dose and time-course consistent with drug-induced parkinsonism.*
5. *Absence of observable response to high-dose levodopa despite at least moderate severity of disease.*
6. *Unequivocal cortical sensory loss (i.e., 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 y 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 years*
4. *Inspiratory respiratory dysfunction: either diurnal or nocturnal inspiratory stridor or frequent inspiratory sighs*.
5. *Severe autonomic failure in the first 5 years of disease. This can include:*
    a) *Orthostatic hypotension—orthostatic decrease of blood pressure within 3 minutes 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 years 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 years 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 y 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)*
9. *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:

Mr. Peterson meets the first essential criterion of parkinsonism on his first visit with Dr. Bertorini on 5/23/2001 (*Peterson_0000002573*) who finds the combination of bradykinesia ('slowness", decreased spontaneous blink) plus resting tremor (left hand and foot) and rigidity (right arm).

From there, Mr. Peterson meets the criteria for Probable Parkinson Disease, at a minimum as follows:

1. Mr. Peterson has none of the above absolute exclusion criteria.
2. Mr. Peterson has no potential red flags.

Additionally, Mr. Peterson has 3 supportive criteria: clear response to dopaminergic therapy (notes of Drs. Bertorini (*7/06/01, Peterson 0000002574*), Lai (*5/14/04; Peterson_0000002588*), among others), presence of levodopa-induced dyskinesias (*See* LeDoux (*12/15/10; Peterson_0000002639*), among others), and rest tremor of a limb *(Bertorini, 5/23/01; Peterson 0000002574*). All of these supporting criteria are amply verified throughout his medical records.

Thus, based on the medical records documenting clinical signs and symptoms of Parkinson disease and its associated symptoms, Mr. Peterson's treating providers-including at least 4 independent neurologists-, and the International Movement Disorder Society's more stringent research criteria, it is more likely than not that Mr. Peterson has Parkinson Disease.

Causality:

Mr. Peterson's Parkinson's disease is at least as likely as not caused by his exposure to the contaminated water at Camp Lejeune. Stated differently, Mr. Peterson 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 just 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, with no other known causative factor, 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. Peterson, given our expanding knowledge of causative etiologies including in his case, exposure to contaminated water at Camp Lejeune (with hazardous levels of trichloroethylene(TCE)), in my opinion, one can conclude that his diagnosis of Parkinson's disease is at least as likely as caused by his exposure to these toxins.

The concept of 'multifactorial' also needs to be discussed. Dr Bertorini (*Bertorini Dep. 33, generally*) agrees that it is 'likely so' that the cause of Parkinson disease is multifactorial. Parkinson's disease is known to be associated with a number of risk factors. 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.

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.

Therefore, in my opinion, whether Mr. Peterson has other risk factors for the development of Parkinson disease is irrelevant unless they were so overwhelming that his exposure to the contaminated water at Camp Lejeune was non-contributory. In my opinion, that clearly was not the case.

Before discussing potential risk factors for the Mr. Peterson's development of Parkinson disease, it is important to note that the standard for this opinion is "at least as likely as not."

His treating neurologist, Dr. Bertorini, wrote in a letter on 11/09/12 that "it is my opinion, within a reasonable degree of medical certainty, that Mr. Peterson's PD could have been caused by his presence at Camp Lejeune and his exposure to, ingestion of, and/or contact with, that contaminate water." (*Peterson_0000000600*). During testimony, Dr Bertorini reported that in an amendment letter he offered the opinion that "more likely than not there is an indication that Mr. Peterson's condition of Parkinson disease resulted from his exposure to the contaminated water at Camp Lejeune." (*Bertorini Dep. Exhibit 3*).

In an independent VA compensation exam on January 29, 2014, Dr. Bronstein reviewed several articles from the medical literature available at that time. (*Peterson_0000002498- 0000002504*). She noted that the most recent Up-To-Date review of the topic that included "evidence is inconclusive for other putative risk factors which include exposure to hydrocarbon solvents, particularly trichloroethylene." (*Peterson_0000002499*). Based on the totality of her readings in the report, she still comes to a determination that "it is at least as likely as not (50-50 probability) that Vet's Parkinson's disease was caused by or a result of exposure to contaminated water at Camp Lejeune." (*Peterson_0000002503*). It is important to note that opinion was 10 years ago and that the research linking TCE with Parkinson's disease has substantially grown over the ensuing decade (*see below)*. The most

recent Up-To-Date review, by the same author, while still writing that the "cause still unknown in the majority of cases" has elevated TCE exposure to a risk factor.

The Department of Veteran Affairs itself has concluded that Mr. Peterson's Parkinson disease is related to his exposure to water at Camp LeJeune. In a 2/11/14 VA Rating Decision Letter, the VA notes disability due to balance impairment, right arm tremor, left arm tremor, and leg rigidity. These are clearly all part of his Parkinson's disease and are almost pathognomonic for that entity. The VA concludes that "Service connection for balance impairment due to Parkinson's disease…Service connection for imbalance impairment has been established as directly related to military service. Upon review of all the evidence of record, the VA subject matter expert opined it is at least as likely as not (50-50 probability) that veterans Parkinson's disease was caused by or result of exposure to contaminated water at Camp Lejeune."

The standard to be met in the attribution of Mr. Peterson's Parkinson disease to his exposure to toxins in Camp Lejeune is 'at least as likely as not'. This level of certainty is provided by prior medical professionals. Dr Kumar simply testifies that he "does not know' if Mr. Peterson's Parkinson disease was caused by his exposure to water at Camp Lejeune, but clarifies that he "did not investigate" *(Kumar Dep. 81:24-25)*. On the alternative side, there is no evidence provided by Mr. Peterson's many physicians, including at least 3 neurologists, that Mr. Peterson's Parkinson disease was not, or unlikely to have been, caused by his exposure to the contaminated water at Camp Lejeune. From the records I reviewed, I did not see where Mr. Peterson's physicians engaged in a differential etiology to evaluate the cause of his Parkinson's Disease, nor would I have expected them to do so.

The possibility of alternative causes or contributing factors for Mr. Peterson's 'illnesses and conditions' has been raised (*US Supp Resp.to Plfs' First Set of Interrogatories, p 24*). 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. Peterson, like the majority of people his age, have more than one medical condition. The issue is his Parkinson's disease. Mr. Peterson's 'family history of strokes' is entirely irrelevant as he has Parkinson's disease and not cerebrovascular disease (strokes). It was also noted that prior to his diagnosis of Parkinson's disease he was being treated for 'mental illness, numbness, a hernia, and cervical pathology.' With the exception of 'mental illness' (mood disorder), which is specifically addressed below, the other conditions simply have nothing to do with Parkinson's disease and can therefore be ruled out in the differential etiology analysis.

TCE and PCE Exposure [1]

[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 those 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 can cause Parkinson's disease.

In my opinion, Mr. Peterson had substantial exposure to PCE and TCE from his time at Camp Lejeune.

TCE and PCE have been long known to cause health risks to humans such that the US Environmental Protection Agent has recently banned their use. These two chemicals have been known to be 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 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; De Miranda, General Causation report)*. 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 2012; 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 significance 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 water at Camp Lejeune, is clearly an appropriate factor to consider when conduction a causality assessment using a differential diagnosis for Parkinson's Disease.

Mr. Peterson does not have any documented exposure to TCE outside of this location, either in his other jobs or recreational activities.

Mr. Peterson was exposed to the water at Camp LeJeune from 5/16/1975 to 6/15/1977; with an estimated total exposure 25 months of exposure from living or working at Hadnot Point, Mainside Barracks, and Tarawa Terrace. (*See* 01576_PETERSON_NARA_0000000085; 01576_PETERSON_NARA_0000000230; 01576_PETERSON_VBA_0000000161; 01576_PETERSON_NARA_0000000052)

Mr. Peterson was exposed to the water at Camp Lejeune via water buffalo, during meals and from the water fountain during the day, and in the shower (*Peterson Dep. 16-19*). Mr. Peterson classified himself as a "water drinker" who would "probably have three to four [c]ups of water" in addition to other beverages made with water on base. (*Peterson Dep. 19:9-12*).

ATSDR Assessment (*Jan 13, 2017*), "A marine in training at Camp Lejeune 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 2024; 132:10*). 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."

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)*. Using available data, Gary Miller, PhD (*General Causation toxicology report)* estimates 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. 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*).

Mr. Peterson far exceeds that minimum exposure.

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

Dr Reynolds charts are found below:

	Chart 1: 1L	Chart 2: ATSDR civilian worker RME (3.092 L consumption per day) Scenario 1	Chart 3: ATSDR civilian worker CTE (1.227 L consumption per day) Scenario 2	Chart 4: FM 1957-1983 light activity (desk work, guard/KP duty), moderate day, desert/tropical <80oF (5.2049 L consumption per day) Scenario 3
	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 ATSDR exposure assumptions)**	**Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions)**
TCE	155,319	480,246	190,576	747,330
PCE	5,190	16,047	6,368	25,015
VC	7,264	22,460	8,913	35,175
BZ	1,528	4,725	1,875	7,209

As noted above, it has been calculated based on 3 different scenarios that Mr. Peterson had a cumulative exposure range of 190 mg to 747 mg of TCE, or about 1.3 to 5 times the conservative minimum amount associated with an increased risk of Parkinson's disease. Another way to assess the likely causality is to also see that he was exposed to levels greater than 366 mcg/L for 6 of the months that he was at Camp Lejeune, twice the minimum exposure estimated to potentially cause Parkinson's disease.

It must also be noted that this estimate does not include inhalation and dermal sources of exposure. Animal models indicate that inhalation exposure to TCE is equally if not even more toxic. Therefore, his total exposure is even higher than the above numbers. It also does not include his cumulative PCE exposure, which, as noted above, is likely to be of similar toxicity and additive in its effect, as PCE breaks down to TCE (PCE and TCE both breakdown to Vinyl chloride, another known contaminant in the water at Camp Lejeune) (*Michael Freeman, MedDR, PhD, GC Expert report*). Mr. Peterson's estimated cumulative dose of PCE exceeds an additional range of 6 to 25 mcg. These would only add to the already established toxicity of TCE alone.

Given his substantial exposure, it is my opinion that Mr. Peterson's Parkinson's disease was at least as likely as not caused by his exposure to primarily TCE, but also lesser amount of PCE, vinyl chloride and benzene in the water at Camp Lejeune.

Mr. Peterson likely developed his first symptoms of Parkinson's disease between 1995 and 2000 (*Peterson_0000002668-2671; 0000002318*). The latency period between Mr. Peterson's exposure to TCE and the development of his Parkinson disease, is expected given the mechanism of the disease. By the time Parkinson's Disease is clinically manifested, it is estimated that the underlying dying 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. Reports in humans show a latency from exposure to symptom development to range from 10-40 years (*Goldman, et al Ann Neurol 2012; 71:776-784)*.

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. Peterson's Parkinson's disease. Although this diagnosis/etiology opinion can be based on clinical findings and judgment alone, it can also be supported by and 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. Peterson'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.

Demographics

Mr. Peterson likely started having symptoms of his Parkinson disease around age 50 (range 47-51) and diagnosed with the disease at age 52. This is a relatively young onset. Younger onset of disease is felt to be more likely with genetic causes, however, Mr. Peterson has no known family history of Parkinson disease. This makes considering other risk factors relatively more important, including toxin exposure (in this case TCE/PCE). While he did not smoke, Mr. Peterson did consume caffeine, a small but mitigating factor *against* the development of Parkinson disease. Furthermore, Mr. Peterson was a vigorous exerciser which also tends to mitigate against the risk of Parkinson's. Therefore, he developed Parkinson disease despite these mitigating factors.

Mood disorders:

Mr. Peterson had the onset of a mood disorder in the time frame immediately preceding his diagnosis of Parkinson disease. On 1/14/25, he denied any antecedent issues with depression. Rather than this being the cause of his symptoms, which was an original consideration, the onset of his mood disorder was likely part of his development of Parkinson's disease. Depression has been associated with Parkinson's disease, and studies have shown that the incidence of depression increases in the immediate time frame prior to receiving a diagnosis of Parkinson's disease and data suggest that depression may indeed be an early symptom (*Jacob EL, et al 2010, Parkinsonism Relat Disorder 16:576; Badenoch, et al.2024, J Neurol Neurosurg Psychiatry 95:966)*. Given the timing of its onset and lack of a preceding history of significant depression, the development of depression by Mr. Peterson was likely an early symptom of his Parkinson disease and not a risk factor of any significance.

Head trauma:

While there is an association between head trauma and Parkinson's disease, the risk increases with more severe head trauma and with multiple episodes of trauma.

Medical records indicate that Mr. Peterson had 1 concussion while playing football. This is reported to have occurred in 1974 (*Peterson_0000002588; Deposition, Lori Peterson, 62*) This single episode of concussion was not notable enough to be reported (*Peterson_000002489*) or considered as a high enough risk factor for his other treating neurologists (Drs Bertolini, Lai, LeDoux, and Sillay) to mention this lone episode of trauma in their formulation of his condition (*Peterson_0000002489; Peterson_0000002571; Peterson_0000002588; Peterson_0000002639; and Peterson_0000000595-598*.) His

description of the event on 1/14/2025 would be consistent with a mild concussion with 15-20 second loss of consciousness but immediately able to drive and function afterwards and with no evidence of post-concussive symptoms nor any other sequelae.

Mr. Peterson has not had the significant head trauma often considered as a risk factor. Not only was his head trauma an isolated incident and of a severity that would be considered 'mild' by standard criteria with no post-concussive symptoms, but it also did not prevent him from pursuing a cognitive-intense career and becoming a successful attorney. In my opinion, this isolated, single mild event is not as substantial as his exposure to the toxic water at Camp Lejeune, and can be ruled out as cause of his Parkinson's disease.

In my professional medical opinion, to consider Mr. Peterson's head trauma, in light of his substantial Camp Lejeune water exposure, would be speculative as a potential cause of Mr. Peterson's Parkinson's disease.

Family History / Genetics:

As noted above, research in twins would indicate 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. On the other hand, whether genetic factors predispose an individual to the risks associated with TCE/PCE exposure is irrelevant if the toxin exposure would be a necessary condition for the development of Parkinson's disease. That is, one could only say that the toxin exposure is irrelevant if a person had the genetics that made them more likely than not to develop Parkinson's disease regardless of toxin exposure.

It is clear that Mr. Peterson is not in that category.

Mr. Peterson 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 moderate size, including 3 siblings, children and other known relatives (parents and 5 aunts and uncles), would not be suspected. Lack of Parkinson's disease in his siblings, also diminishes the likelihood that early life exposure played a role in Mr. Peterson's development of Parkinson's Disease.

Mr. Peterson has been diagnosed with melanoma. It is known that there is a bidirectional association between Parkinson's disease and melanoma. A common underlying mechanism has been postulated as both are considered to "result from complex interactions of genetic and environmental factors" (*Ye, Q, et al 2020 Front Aging Neurosci)*. However, given Mr. Peterson's lack of family history makes the toxin exposure, at least as likely, if not more likely, than a genetic cause.

Other potential toxins:

Exposure to pesticides and herbicides are recognized as risk factors for the development of Parkinson's disease. He has no known exposure to either of these toxins.

There are questions regarding his exposure to paint. This exposure enters the discussion because of blood tests to rule out heavy metal exposure as a cause of his symptoms early on in his medical evaluation. However, this exposure is neither quantified nor qualified other than being homeowner related. Mr. Peterson reports that he had no commercial painting employment, and his painting involved his own residences and occurred after buying his home in 1978. Of note lead paint was banned in 1978. Not only is 'paint' itself not a notable risk factor for Parkinson's disease but his blood test was negative for heavy metals.

Other than his time on Camp Lejeune, Mr. Peterson had no clear exposure to other potential neurotoxins that might be contributors to or causative for Parkinson's Disease.

VII. Diagnosis and Causation Conclusion

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

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"- i.e., 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 substantial contributor to morbidity and mortality.

Non motor manifestations can be even more impactful. These are wide-ranging and they include but 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; and of course, cognitive changes including dementia. Mood disorders, specifically anxiety and depression are also associated with Parkinson disease and are considered non-motor symptoms.

Mr. Peterson is already suffering from a number of these symptoms, which are likely to worsen. Furthermore, he is likely to suffer from even more symptoms and complications in the future as his disease progresses. Many symptoms can be temporarily treated with medications or in his case, deep brain stimulation. 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 treatment, 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 disease come with their own side effects, as is seen in Mr. Peterson's medical records. 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.

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

Mr. Peterson was diagnosed with Parkinson's disease in 2001 at which time he was already manifesting several symptoms which were affecting his ability to work as a trial attorney. Hypophonia (low voice volume with inability to project one's voice), with decreased facial movements make projecting emotion and emphasis on spoken words difficult or even ineffective. Tremor can be a distraction to an audience. His doctor noted that "when he in is in court he starts shaking more and his speech becomes slower". (*Peterson_0000002577*)

By 2002 and 2003, he was exhibiting a change in personality and compulsive gambling (*see Peterson_0000002582)*. Compulsive gambling is one of the most common Impulse Control Disorders more common in younger -onset Parkinson's disease and the use of dopamine agonists, such as ropinirole (*Santangelo, G. et al. 2013; Park Related Disord; 19:645)*. Both of these pertain to Mr. Peterson. His episode of psychosis and delusions were clearly brought on by his Parkinson's disease and its treatment, and in his case severe enough to warrant a seven-day hospitalization (*Peterson_0000000006-116)*. Psychosis and delusions can be caused by Parkinson's disease itself, but more commonly are caused by the interaction with medication, which in his case was the most likely culprit given that with appropriate medications, his symptoms did not return. Ultimately, it is more likely than not that the burden of his disease and its treatment led to his termination at work.

Cognitive problems are one of the most negatively impactful complications of Parkinson disease. Mild cognitive impairment overall affects about half of all patients with Parkinson disease. 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)*. Given his episode of psychosis, Mr. Peterson is at higher risk for developing frank dementia (*Factor et al 2003, Neurol. 60:1756-1761*).

Mr. Peterson opted to get deep brain stimulation, which was a reasonable decision. Placement of a deep brain stimulator is an invasive procedure involving the drilling of burr holes through the skull and pacing electrodes in the brain. It also requires the surgical placement of a battery pack under the skin and running wires under the skin to the electrodes. Furthermore, the batteries need to be surgically replaced every several years. All of these involve hospital stays and visits for procedures.

Mr. Peterson was involved in an MVA on 8/11/20 (*Peterson_0000000395*). This occurred when he fell asleep while driving, which more likely than not was related to his treatments for his Parkinson's disease. As a result, 'given his severe Parkinson's disease, MVA and high risk for MVA, he was told not to drive' (*Peterson_0000000519)*). Syncope (loss of consciousness) is a complication of both Parkinson's disease as well as its treatment. Further episodes of syncope ('blacking out') resulted in a 5-day hospitalization (*Peterson_0000002193-2232)*.

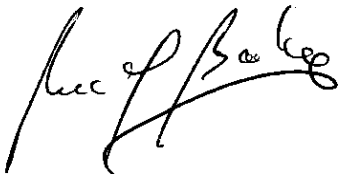
Mr. Peterson's Parkinson's disease has caused a significant deterioration in his gait and mobility. He started having increasing falls by 2011 (*Peterson_0000002645)*. By 2013, not only had his gait deteriorated further, but he was having freezing episodes along with uncontrollable excessive movements (dyskinesias) (*Peterson_0000000003)*. By 2015, further complications of his Parkinson's disease included sleep difficulties, fainting, urinary frequency, muscle pain, depression, and anxiety (*Peterson_0000000591-594*). The combination of freezing, dyskinesias, and generalized impairment of balance all progressed and resulted in increasing falls and even an inability to sit still (*Peterson_0000000571-574*). By 2021, his wife was requesting that he have a wheelchair.

Another manifestation of the motor deterioration in Parkinson 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. In my experience this is even more devastating to people whose professional career revolved around speaking. The effect is evident during his testimony (*Deposition, Peterson, 2/8/24*), the transcripts of which make clear that he was at best difficult to understand and needed to use text to speak software. My zoom interview with him on 1/14/25 confirmed that the vast majority of his speech is unintelligible and he almost entirely relied on his electronic speech assist device.

In regard to the overall effect of the disease on his quality of life, Dr Joseph Allen, his primary care doctor who has known him for decades summed it up as "[he was a] jocular, fun-loving guy, loving life, always pleasant to be around. And now he's a shell of his former self, both his capabilities and also his mood is not as good either." (*Allen Dep. 34:1-6*).

Depression is a common comorbidity of Parkinson disease and is considered manifestations of the disorder, occurring in 40-50% of patients (*Marsh, L 2013, Curr Neurol Neurosci Rep 13:409*). As above, it is more likely than not that Mr. Peterson's depression is a manifestation of his Parkinson's disease, and that the pathology of Parkinson's disease contributes to its severity. The effect of depression on quality of life is self-evident.

In sum, in my opinion, Mr. Peterson's Parkinson disease was at least as likely as not caused by his prolonged exposure to TCE while at Camp LeJeune. Recent research indicates that Parkinson disease progression may be faster in people exposed to TCE with shorter periods of time until the development of psychosis, bone fractures and falls (*Goldman2024, Movement Dis 39: 1732*). Mr. Peterson has suffered significantly already from his Parkinson 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. This is true for his wife as well, as the caretaker burden for a patient with Parkinson's disease is immense.



Richard L Barbano, MD, PhD

2/7/2025

Reference Materials

ATSDR Assessment of the evidence for the drinking water contaminants at Camp Lejeune and specific cancers and other diseases. Jan 13, 2017.

Badenoch JB, Paris A, Jacobs BM, et al. Neuroanatomical and prognostic associations of depression in Parkinson's disease. J Neurol Neurosurg Psychiatry 2024; 95: 966-973

Bove FJ, Greek A, Gatilba R, et al. Cancer incidence among marines and navy personnel and civilian workers exposed to industrial solvents in drinking water at US Marine Corps Base Camp Lejeune. Environ Health Perspect 2024; 132:107008. Doi 10.1289/EHP14966

Factor SA, Feustel PJ, Friedman JH, et al. Longitudinal outcome of Parkinson's disease patients with psychosis. Neurol 2003; 60:1756-1761

Gallagher, J, Gochanour C, Caspell-Garcia C, et al. Long-term dementia risk in Parkinson's disease. Neurology 2024;103; e209699

Gardner, R, Byers AL, Barnes DE, et al. Mild TBI and risk of Parkinson disease. A chronic effects of neurotrauma consortium study. Neurol 2018; 90: e 1771-e1779

Gash DM, Rutland K, Hudson NL, et al. Trichloroethylene: Parkinsonism and complex 1 mitochondrial neurotoxicity. Ann Neurol 2008 63:184-92

Goldman SM, Quinlan PJ, Ross GW, et al. Solvent exposures and Parkinson disease risk in twins. Ann Neurol 2012;71(6):776-784. doi:10.1002/ana.22629

Goldman S. Environmental toxins and Parkinson's disease. Ann Rev Pharmacol Toxicol 2014; 54:141-64.

Goldman SM, Weaver FM, Stroupe KT, et al. Risk of Parkinson disease among service members at Marine Corps base Camp Lejeune. JAMA Neurol 2023; 80:673-681.

Goldman SM, Weaver FM, Gonzalez B, et al. Pakinson's disease progression and exposure to contaminated water at Camp Lejeune. Movement Disord 2024; 39: 1732-1739. doi:10.1002/mds.29922

Jacob EL, Gatto NM, Thompson A, et al. Occurrence of depression and anxiety prior to Parkinson's disease. Parkinsonism Relat Disorders 2010;16: 576-581.

Liu M, Yang CS, Chen MC, et al. Trichloroethylene induces dopaminergic neurodegeneration in Fisher 344 rats. J Neurochem 2010, 112:773-83.

Marsh L. Depression and Parkinson's disease; current knowledge. 2013 Curr Neurol Neurosci Rep 13:409

Medscape. Exposure to industrial Solvent Linked to 24% Higher PD Risk - April 26, 2024.

Postuma, R, Berg D, Stern M, et al. MDS clinical diagnostic criteria for Parkinson's disease. Movement Disorders 2015, 30: 1591- 601

Santangelo G, et al. Pathological gambling in Parkinson's disease. A comprehensive review. 2013 Parkinsonism Relat Disorders 19: 645-653.

Shaw CA and Hoglinger GU. Neurodegenerative diseases: Neurotoxins as sufficient etiologic agents? Neuromolecular Med 2008; 10:8016-8

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

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, 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

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

Tarolli CG, Barbano R. Quality measures in Parkinson disease: What do the outcomes show? Neurol Clin Pract 2020; 10:5-6. Doi:10.1212/CPJ0000000000756. PMID: 3218501

Giacino JT, Katz DI, Schiff ND, et al. Comprehensive Systematic Review Update Summary: Disorders of Consciousness: Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology; the American Congress of Rehabilitation Medicine; and the National Institute on Disability, Independent Living, and Rehabilitation Research. Arch Phys Med Rehabil. 2018 Aug 7. pii: S0003-9993(18)30447-7. doi: 10.1016/j.apmr.2018.07.002. PMID:30098792

Giacino JT, Katz DI, Schiff ND, et al. Practice Guideline Update Recommendations Summary: Disorders of Consciousness: Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology; the American Congress of Rehabilitation Medicine; and the National Institute on Disability, Independent Living, and Rehabilitation Research. Arch Phys Med Rehabil. 2018 Aug 7. pii: S0003-9993(18)30446-5. doi: 10.1016/j.apmr.2018.07.001. PMID:30098791

Giacino JT, Katz DI, Schiff ND, et al. Practice guideline update recommendations summary: Disorders of consciousness: Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology; the American Congress of Rehabilitation Medicine; and the National Institute on Disability, Independent Living, and Rehabilitation Research. Neurology.

2018 Aug 8. pii: 10.1212/WNL.0000000000005926. doi: 10.1212/WNL.0000000000005926. PMID:30089618

Giacino JT, Katz DI, Schiff ND, et al. Comprehensive systematic review update summary: Disorders of consciousness: Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology; the American Congress of Rehabilitation Medicine; and the National Institute on Disability, Independent Living, and Rehabilitation Research. Neurology. 2018 Aug 8. pii: 10.1212/WNL.0000000000005928. doi: 10.1212/WNL.0000000000005928. PMID: 30089617

Mahajan, A, Jankovic J, Marsh L, Patel A, Jinnah HA, Comella C, Barbano R, Perlmutter J, Patel N. Cervical Dystonia and substance abuse. J Neurol. 2018 265(4): 970-975. PMID 29569175

Junker, J, Brandt V, Berman B, et al. Predictors of Alcohol Responsiveness in Dystonia. Neurology. 2018; 91(21): e2020-2026. PMID: 30341158

Hoffmann C, Schweighardt A, Conn KM, Nelson D, Barbano R, Marshall F, Brown J. Enhanced Adherence in Patients Using an Automated Home Medication Dispenser J Healthc Qual. 2018 Jul/Aug;40(4):194-200. doi: 10.1097/JHQ.0000000000000097. PMID:28749791

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

Korn RE, Shukla AW, Katz M, et al. Virtual visits for Parkinson Disease: a multi-center non-controlled cohort. Neurol Clin Pract 2017; 7(4):283-295. PMID:2884091

Saran JS, Barbano RL, Shult R, Wiegand TJ, Selioutski O. Chronic diphenhydramine abuse and withdrawal: diagnostic challenge. Neurol Clin Pract 2017 ; 7(5):439-441. PMID 29569175

Pirio Richardson S; Wegele AR; Skipper B; Deligtisch A; Jinnah HA; Dystonia treatment: Patterns of medication use in an international cohort. Neurology 2017;88(6):543-550. PMID 28077492

Norris SA, Jinnah HA, Espay AJ, Klein C, Bruggemann N, Barbano RL, et al. Clinical and demographic characteristics related to onset site and spread of cervical dystonia. Mov Disord. 2016; 31:1874-1882. PMID 27753188

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

Evidente VG, Truong D, Jankovic J, Comella CLGrafe S,Hanschmann A. IncobotulinumtoxinA (Xeomin) injected for blepharospasm or cervical dystonia according to patient needs is well tolerated. J Neurol Sci 2014; 346:116-120. (site PI)

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 (see attached)

CURRICULUM VITAE

Richard L. Barbano, MD, PhD, FAAN

Addresses

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

University of Rochester
265 Crittenden Boulevard
Box MIND
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

Tarolli CG, Barbano R. Quality measures in Parkinson disease: What do the outcomes show? Neurol Clin Pract 2020; 10:5-6. Doi:10.1212/CPJ0000000000756. PMID: 3218501

Giacino JT, Katz DI, Schiff ND, et al. Comprehensive Systematic Review Update Summary: Disorders of Consciousness: Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology; the American Congress of Rehabilitation Medicine; and the National Institute on Disability, Independent Living, and Rehabilitation Research. Arch Phys Med Rehabil. 2018 Aug 7. pii: S0003-9993(18)30447-7. doi: 10.1016/j.apmr.2018.07.002. PMID:30098792

Giacino JT, Katz DI, Schiff ND, et al. Practice Guideline Update Recommendations Summary: Disorders of Consciousness: Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology; the American Congress of Rehabilitation Medicine; and the National Institute on Disability, Independent Living, and Rehabilitation Research. Arch Phys Med Rehabil. 2018 Aug 7. pii: S0003-9993(18)30446-5. doi: 10.1016/j.apmr.2018.07.001. PMID:30098791

Giacino JT, Katz DI, Schiff ND, et al. Practice guideline update recommendations summary: Disorders of consciousness: Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology; the American Congress of Rehabilitation Medicine; and the National Institute on Disability, Independent Living, and Rehabilitation Research. Neurology. 2018 Aug 8. pii: 10.1212/WNL.0000000000005926. doi: 10.1212/WNL.0000000000005926. PMID:30089618

Giacino JT, Katz DI, Schiff ND, et al. Comprehensive systematic review update summary: Disorders of consciousness: Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology; the American Congress of Rehabilitation Medicine; and the National Institute on Disability, Independent Living, and Rehabilitation Research. Neurology. 2018 Aug 8. pii: 10.1212/WNL.0000000000005928. doi: 10.1212/WNL.0000000000005928. PMID: 30089617

Mahajan, A, Jankovic J, Marsh L, Patel A, Jinnah HA, Comella C, Barbano R, Perlmutter J, Patel N. Cervical Dystonia and substance abuse. J Neurol. 2018 265(4): 970-975. PMID 29569175

Junker, J, Brandt V, Berman B, et al. Predictors of Alcohol Responsiveness in Dystonia. Neurology. 2018; 91(21): e2020-2026. PMID: 30341158

Hoffmann C, Schweighardt A, Conn KM, Nelson D, Barbano R, Marshall F, Brown J. Enhanced Adherence in Patients Using an Automated Home Medication Dispenser J Healthc Qual. 2018 Jul/Aug;40(4):194-200. doi: 10.1097/JHQ.0000000000000097. PMID:28749791

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

Korn RE, Shukla AW, Katz M, et al. Virtual visits for Parkinson Disease: a multi-center non-controlled cohort. Neurol Clin Pract 2017; 7(4):283-295. PMID:2884091

Saran JS, Barbano RL, Shult R, Wiegand TJ, Selioutski O. Chronic diphenhydramine abuse and withdrawal: diagnostic challenge. Neurol Clin Pract 2017 ; 7(5):439-441. PMID 29569175

Pirio Richardson S; Wegele AR; Skipper B; Deligtisch A; Jinnah HA; Dystonia treatment: Patterns of medication use in an international cohort. Neurology 2017;88(6):543-550. PMID 28077492

Norris SA, Jinnah HA, Espay AJ, Klein C, Bruggemann N, Barbano RL, et al. Clinical and demographic characteristics related to onset site and spread of cervical dystonia. Mov Disord. 2016; 31:1874-1882. PMID 27753188

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

Evidente VG, Truong D, Jankovic J, Comella CLGrafe S,Hanschmann A. IncobotulinumtoxinA (Xeomin) injected for blepharospasm or cervical dystonia according to patient needs is well tolerated. J Neurol Sci 2014; 346:116-120. (site PI)

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

Truong DD, Gollomp SM, Jankovic J, LeWitt PA, Marx M, Hanschmann A, Fernandez HH; Xeomin US Blepharospasm Study Group. Sustained efficacy and safety of repeated incobotulinumtoxinA (Xeomin(®)) injections in blepharospasm. J Neural Transm. 2013; 120(9):1345-53 PMID 23435927

Barbano RL, Hill DF, Snively BM, Light LS, Boggs N, McCall WV, Stacy M, Ozelius L, Sweadner KJ, Brashear A. New triggers and non-motor findings in a family with mutation in ATP1A3. Parkinsonism and Related Disorders 2012; Jul;18(6):737-41. PMID: 22534615.

Jankovic J, Adler CH, Charles PD, Comella C, Stacy M, Schwartz M, Sutch SM, Brin MF, Papapetropoulis S. (as part of CD PROBE Study group). Rationale and design of a prospective study: Cervical Dystonia Patient Registry for Observation of OnaBotulinumtoxinA Efficacy (CD PROBE). BMC Neurology 2011,11:140

Comella CL, Jankovic J, Truong DD, Hanschmann A, Grafe S; US Xeomin Cervical Dystonia Study Group. Efficacy and safety of incobotulinumtoxinA (NT201,Xeomin, botulinum neurotoxin type A, without accessory proteins) in patients with cervical dystonia. J Neurol Sci 2011; 308:103-9. PMID 21764407

Perlmutter, DH, Petraglia, AL, Barbano, R, Schwalb JM. Microvascular Decompression in Patient With Atypical Features of Hemifacial Spasm Secondary to Compression by a Tortuous Vertebrobasilar System: Case Report. Neurosurg 2010; 66 (6):e1212 . PMID: 20495395

Dworkin RH, Barbano RL, Tyring SK, Betts RF, McDermott MP, Pennella-Vaughan J, Bennett GJ,Berber E, Gnann JW,Irvine C, Kamp C, Kieburtz K, Max MB, Schmader KE. A randomized, placebo-controlled trial of oxycodone and of gabapentin for acute pain in herpes zoster. Pain. 2009 Apr;142(3):209-17. PMID: 19195785

Simpson DM, Gracies J-M, Yablon SA, Barbano R, and Brashear A. Botulinum Neurotoxin vs Tizanidine in Upper Limb Spasticity: A Placebo-Controlled Study. J Neurol Neurosurg Psychiatry. 2009 Apr;80(4):380-5. PMID:18977811

Dorsey ER, Beck CA, Adams M, Chadwick G, de Blieck EA, McCallum C, Briner L, Deuel L, Clarke A, Stewart R, Shoulson I; Huntington Study Group TREND-HD Investigators. Communicating clinical trial results to research participants. Arch Neurol. 2008 Dec; 65(12):1590-5. PMID 19064746

Huntington Study Group TREND-HD Investigators. Randomized controlled trial of ethyl-eicosapentaenoic acid in Huntington disease: The TREND-HD Study. Arch Neurol 2008; 65(12): 1582-9. PMID 19064745

Swope D, Barbano R. Treatment recommendations and practical applications of botulinum toxin treatment cervical dystonia. Neurol Clin 2008; 26 Suppl 1:54-65. PMID 18603168

Herrmann DN, Pannoni V, Barbano RL, Pennella-Vaughan J, Dworkin RH. Skin Biopsy and Quantitative Sensory Testing Do Not Predict Response to the Lidocaine Patch 5% in Painful Neuropathies. Muscle Nerve 2006; 33: 42-48 PMID 16228968

Comella CL, Jankovic J, Shannon KM, Tsui J, Swenson M, Leurgans S, Fan W, and the Dystonia Study Group. Comparison of botulinum toxin serotypes A and B for the treatment of cervical dystonia. Neurology 2005; 65: 1423-1429.

Chai J, Herrmann DN, Stanton M, Barbano RL, Logigian EL. Painful Small Fiber Neuropathy in Sjogren Syndrome. Neurology 2005; 65: 925-927. PMID 16186536

Frank S, Barbano RL. Trauma-Induced Spinal Vascular Event Producing Hemipseudoathetosis. Mov Disord 2005; 20:1378-80.

Herrmann DN, Barbano RL Hart-Gouleau, S Pennella-Vaughan, J Dworkin, RH. An Open label Study of the Lidocaine Patch 5% in Painful Idiopathic Sensory Polyneuropathy, Pain Medicine 2005; 6: 379-384. PMID 16266359

Barbano, RL, Herrmann, DN, Hart-Gouleau, S, Pennella-Vaughan, J, Lodewick, PA, Dworkin, RH. Effectiveness, Tolerability, and Impact on Quality of Life of the 5% Lidocaine Patch in Diabetic Polyneuropathy. Arch Neurol 2004; 61: 914-918.PMID 15210530

Herrmann DN, Ferguson ML, Pannoni V, Barbano RL, Stanton M, Logigian EL. Plantar nerve AP and skin biopsy in sensory neuropathies with normal routine conduction studies. Neurology 2004; 63:879-885.PMID 15365140

Nodera, H, Barbano, RL, Henderson, D, Herrmann, DN. Epidermal reinnervation concomitant with symptomatic improvement in a sensory neuropathy. Muscle & Nerve 2003: 27(4): 507-09. PMID 12661055

Gilliam, FG, Veloso, F, Bomhof, MAM, Gazda SK, Biton V, Ter Bruggen JP, Neto W, Bailey C, Pledger G, Wu Sc, and the Topiramate EPMN 104 Study Group. A dose-comparison trial of topiramate as monotherapy in recently diagnosed partial epilepsy. Neurology 2003; 60: 196-202.

Barbano R, S Hart-Gouleau, J Pennella-Vaughan, RH Dworkin. Pharmacotherapy of painful diabetic neuropathy.

Curr pain Headache Rep 2003; 7:169-77. PMID 12720596

Musser, WS, Barbano, RL, Berger AR, Thornton, CA, Moxley R, Herrmann DN, Logigian EL. Distal Myasthenia Gravis with a decrement, an increment, and denervation. J Clin Neuromusc Dis 2001: 3: 16-19. PMID 19078648

Apfel SC, Schwartz S, Adornato BT, Freeman R, Biton V, Rendell M, Vinik A, Giuliani M, Stevens JC, Barbano R, Dyck PJ. Efficacy and safety of Recombinant Human Nerve Growth Factor in patients with diabetic polyneuropathy: A randomized Controlled Trial. JAMA 2000; 284: 2215-2221. PMID 11056593

Counihan, TJ and Barbano, RL. Progressive micrographia in a migraineur. Neurology 2000; 54:2107.

Apfel, SC, Kessler, JA, Adornato, BT, Litchy, WJ, Sanders, C, Rask, CA, and the NGF Study Group. Recombinant Human Nerve Growth Factor in the treatment of diabetic polyneuropathy. Neurology 1998; 51:695-702.

Schifitto, G, Barbano, RL, Cohn, SE, Zwillich, SH, and Kieburtz, KD. HIV related vasculitic mononeuropathy multiplex: a role for IVIG? Journal of Neurology, Neurosurgery, and Psychiatry 1997; 63:255-56. PMID 9285470

Riggs, JE, Ketonen, LM, Wymer, JP, Barbano, RL, Valanne, LK, and Bodensteiner, JB. Acute and delayed cerebral infarction after wasp sting anaphylaxis. Clinical Neuropharmacology. 1994; 17:384-388. PMID 9316687

Dal Canto, MC, Barbano, RL, and Jubelt, B. Ultrastructural immunohistochemical localization of poliovirus during virulent infection of mice. Journal of Neuropathy Exp Neurology. 1986; 45:613-618. PMID 3021913

Dal Canto, MC and Barbano, RL. Antibody to myelin associated glycoprotein reacts with Plasma cells in mice. Journal of Neuroimmunology. 1986; 10:279-286.

Dal Canto, MC and Barbano, RL. Immunocytochemical localization of MAG, MBP, and PO protein in acute and relapsing demyelinating lesions of Theilers virus infection. Journal of Neuroimmunology. 1985; 10:129-140.

Barbano, RL and Dal Canto, MC. Serum and cells from Theilers virus infected mice fail to injure myelinating cultures or to produce in vivo transfer of disease. Journal of Neurological Science. 1984; 66:283-293. PMID 6085111

Dal Canto, MC and Barbano, RL. Remyelination during remission in Theiler's virus infection. American Journal of Pathology. 1984; 116:30-45. PMID 6742106

Chapters, Others:
Bartleson JD and Barbano RL. Mechanical and other lesions of the spine, nerve roots, and spinal cord. In: Cecil's Textbook of Medicine, 26th Edition. Elsevier. Philadelphia. 2019

Gronseth, GS et al., Clinical Practice Guideline Process Manual. Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology. 2015

Barbano, RL. Mechanical and other lesions of the spine, nerve roots, and spinal cord. In: Cecil's Textbook of Medicine, 25th Edition. Elsevier. Philadelphia. 2015

Barbano, RL. Commentary: Standard strategies for diagnosis and treatment of patients with newly diagnosed Parkinson disease. Neurology Clinical Practice 2013; 3(6):475-6.

Barbano, RL. Anatomic Principles for Botulinum Toxin Injections. In: Handbook of Dystonia. 2nd Edition M. Stacy, Ed. Informa Healthcare. NY 2011

Swope, D and Barbano, RL. Treatment Recommendations and Practical applications of Botulinum Toxin Treatment of Cervical Dystonia. Neurologic Clinics 2008; 26 (suppl 1):54-65.

Barbano, RL. Anatomic Principles for Botulinum Toxin Injections. In: Handbook of Dystonia. M. Stacy, Ed.

Informa Healthcare. NY 2007

Barbano, RL. Risks of erasing wrinkles. Buyer beware. Neurology. 2006 Nov 28;67(10):E17-8.

Barbano RL . *Invited Commentary* on "Surgery was associated with greater long term treatment success than wrist splinting in carpal tunnel syndrome." ACP Journal Club 2003; 138 (2): 47.

Counihan, TC, Plotkin, K, Deiparine, J, King, A, Cleland, J, Barbano, R and Rempe, D. Current Therapy. In, Baker's Clinical Neurology. RJ Joynt, RC Griggs, Eds. Lippincott-Raven, Philadelphia, 2001; update 2002.

Barbano, RL. Chemotherapy induced neuropathies. In**:** Neurobase. Gilman S, Goldstein GW, Waxman SG,Eds. Arbor Publishing. San Diego, USA. 2000; 2nd ed: 2002; update 2004.

Barbano, RL. Issues and Opinions, "Needle EMG Guidance for injection of Botulinum Toxin". Muscle & Nerve 2001; 24:1567-68.

Logigian, EL and Barbano, RL. Applied physiology of muscle. In: Disorders of Voluntary Muscle, 7th Edition. Karpati G, Hilton-Jones D, Griggs R, Eds. Cambridge University Press, Cambridge, UK. 2002.

Barbano, RL, Feigen,A, and Kieburtz,K. Neurologic Complications of Vitamin and Mineral Disorders. In, Baker's Clinical Neurology. RJ Joynt, RC Griggs, Eds. Lippincott-Raven. Philadelphia. 2000; Update 2002**.**

Barbano, RL and Griggs, RC. Metabolic Encephalopathy: In, Clinical Neurology, R Rosenberg, Ed. 2nd Edition. John Wiley. 1998.

Barbano, RL and Griggs, RC. Hepatic coma and portal-systemic encephalopathy: Current Therapy in Neurologic Disease. 5th Edition. Mosby Year Book, St. Louis. 1996.

Barbano, RL and Griggs, RC. Hepatic coma and portal-systemic encephalopathy: Current Therapy in Internal Medicine. Mosby year Book, St. Louis. 1996.

Revised 12/2024