Exhibit 556

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Date: 8 May 2025

RE: *Richard Sparks v. United States, 7:23-cv-00682*

I, Dr. Michael Young, M.D. am a board-certified neurology physician. This independent medical report is being conducted for the purpose of independent neurologic medical legal review and assessment. This report is strictly for independent review purposes only and no medical treatment or clinical recommendations will be made. All of the opinions herein are offered based on a reasonable degree of medical certainty. A review of prior medical documentation was carried out, as was an independent medical examination of Mr. Sparks.

A copy of my curriculum vitae, which includes a list of publications, is attached hereto as Exhibit A.

I have not testified as an expert in the last four years.

My rate is $525 per hour.

1. Overview of Report

This report provides a review of Mr. Richard Spark's pertinent medical records, deposition testimony, and current scientific literature, with a focus on the diagnosis, progression, and etiologic factors of Parkinson's disease. Also incorporated is a summary of an independent medical evaluation (IME) of Mr. Richard Sparks, conducted on April 24, 2025. The following sections detail his clinical history, diagnostic evaluation, and an assessment of potential risks, culminating in my independent neurologic opinion regarding his condition and underlying risks.

2. Expert Opinion

Based on my training, clinical experience, a thorough review of the records provided to me, and IME, it is my medical opinion, to a reasonable degree of medical certainty, that Mr. Richard Sparks has Parkinson's disease. Additionally, it is my opinion that there is insufficient evidence to conclude to a reasonable degree of medical certainty that his condition is definitively caused by exposure to contaminated water at Camp Lejeune.

I reserve the right to modify my opinions based on the acquisition of additional information that might arise in the future.

3. Methodology

In formulating this opinion, I reviewed Mr. Sparks' medical records and deposition testimony and all records referenced in this Report, as well as relevant and up-to-date medical literature concerning the diagnosis and potential etiologies of Parkinson's disease. I also performed an IME of Mr. Sparks on April 24, 2025. I formulated a differential diagnosis and comprehensive evaluation to come to my

opinion on causation, which includes an evaluation of all potential risk factors and etiologies, including idiopathic.

4. Medical Records and Other Records Reviewed

Prior medical and legal documentation was reviewed, including the following documents:[1]

- Records from:
  - UCHealth
  - Department of Homeland Security (DHS)
  - NARA
  - VHA
  - VBA
- Treater Depositions, including by:
  - Dr. Sabina Schickli
  - Dr. Maureen Leehey
  - Dr. Christen Epstein
  - Dr. Barrie Schmitt
- Depositions of:
  - Maria Sparks
  - Chris Lewis
  - Richard Sparks
- Radiology from UCHealth
- Specific Causation Expert report by Dr. Schwarz, February 7, 2025
- Life Care Planning report of Michael Fryar, February 7, 2025
- Economic expert report of Chad Staller, February 7, 2025
- Expert Report on Richard Sparks of Judy LaKind, May 8, 2025
- Expert Report on Richard Sparks of Lisa Bailey, May 8, 2025
- Discovery Pool Profile Form
- Short Form Complaint
- Administrative claim form
- Social Security Administration records
- Mr. Sparks' responses to the United States' Requests for Admission

5. Background on Parkinson's Disease

What is Parkinson's Disease (PD)?

Parkinson's disease (PD) is a gradually progressive neurodegenerative disorder that impacts both motor and non-motor functions.[1] James Parkinson first described the condition in 1817 in *"An Essay on the Shaking Palsy"* and in 1872, Jean-Martin Charcot dubbed the condition Parkinson's disease.[2,3] PD involves the loss of dopamine-producing neurons in the subtantia nigra (a part of the midbrain, in the upper part of the brainstem), that results in a dopamine deficiency.[4] Dopamine is a neurotransmitter – a chemical substance that plays a key role in nervous system signaling.[5] Disruptions of dopamine signaling in PD is associated with a range of motor and non-motor

[1] All materials considered are provided in the attached Appendix A.

symptoms.[6] Abnormal aggregates of proteins including a-synuclein have been identified in the central, autonomic and enteric nervous systems of individuals with PD, and have been associated with cell dysfunction and cell death,[7] though the exact pathophysiologic mechanisms in PD are not fully understood, and are being actively researched.[8] Prototypical symptoms of PD include motor symptoms (e.g., bradykinesia, muscle rigidity, rest tremor, gait disturbance)[9] and non-motor symptoms (e.g., constipation, REM-sleep disorders, impaired sense of smell, autonomic changes).[10,11] While PD was initially considered a disorder of the brain alone, there is increasing recognition that PD affects multiple systems throughout the body, and as evidenced by identification of a-synuclein aggregates not only in the brain but also in the gut, peripheral autonomic nervous system, skin, olfactory system, and salivary glands of individuals with PD.[12-15]

How Common is PD?

PD is considered the second most common neurodegenerative disease worldwide.[16] It has been estimated to affect over 6 million people globally, and over 900,000 people in the United States.[16,17] The Global Burden of Disease (GBD) project found that between 1990 and 2016, the number of people diagnosed with PD increased by approximately 76%.[18,19]

How is PD Diagnosed?

There is no single test that can definitively diagnose PD.[20-23] Diagnosis of PD relies principally on comprehensive medical history and meticulous neurological examination.[1,20-23] It is a clinical diagnosis, and no single laboratory or neuroimaging test has been identified that can definitively diagnose PD with 100% sensitivity and 100% specificity, though research efforts are underway to identify biomarkers that may be used to diagnose, subclassify and track disease progression in the future.[24-27]

In 2015, the Movement Disorder Society (MDS) published Clinical Diagnostic Criteria that provided a more detailed method for diagnosing PD based on movement symptoms, response to medication, and other clinical factors.[28-30] The first essential requirement for a diagnosis is parkinsonism, which is defined as bradykinesia (slowness of movement) in combination with at least one of the following: resting tremor or muscle rigidity.[28] Once parkinsonism is established, the diagnostic process determines whether the patient meets criteria for Clinically *Established* PD or Clinically *Probable* PD. This determination requires assessment of supportive criteria, exclusion criteria, and red flags.[28]

Supportive criteria increase confidence in a Parkinson's diagnosis.[28] A clear and dramatic response to dopaminergic therapy is one of the strongest indicators, where the patient returns to near-normal function with medication.[28] If detailed records of the initial response are unavailable, a dramatic response can still be confirmed by marked improvement with dose increases or significant worsening with dose decreases.[28] Additional supportive criteria include the presence of levodopa-induced dyskinesia; rest tremor documented in a clinical exam; and either olfactory loss (loss or diminution of smell) or cardiac sympathetic denervation on MIBG scintigraphy.[28]

Absolute exclusion criteria are features that definitively rule out a Parkinson's diagnosis.[28] These include cerebellar abnormalities (e.g., cerebellar gait, limb ataxia, or cerebellar oculomotor abnormalities); downward vertical gaze palsy or significantly slowed downward eye movement; a diagnosis of probable behavioral variant frontotemporal dementia or primary progressive aphasia within the first five years of disease; Parkinsonian symptoms that are restricted to the lower limbs

for more than three years; treatment with dopamine receptor blockers or dopamine-depleting agents at doses that could cause drug-induced parkinsonism; and a lack of an observable response to high-dose levodopa despite significant disease severity.[28] Additional exclusions include unequivocal cortical sensory loss (e.g., loss of graphesthesia or stereognosis with intact primary sensory modalities), clear limb ideomotor apraxia, progressive aphasia, or normal presynaptic dopaminergic neuroimaging, which suggests the absence of dopaminergic degeneration.[28] If another condition known to cause parkinsonism better explains the patient's symptoms, or if an expert physician determines that an alternative syndrome is more likely, PD is ruled out.[28]

Red flags are warning signs that suggest another condition may be responsible for the patient's symptoms.[28] A major red flag is rapid progression, where the patient develops significant gait impairment requiring regular wheelchair use within five years of symptom onset.[28] A complete absence of motor symptom progression over five or more years, unless the stability is due to treatment, also raises concerns.[28] Other red flags include early bulbar dysfunction, such as severe speech impairments (dysphonia or dysarthria) or swallowing difficulties (dysphagia), within the first five years, as well as respiratory dysfunction, including inspiratory stridor or frequent sighing.[28] Severe autonomic failure in the first five years is another red flag; this may present as orthostatic hypotension, defined as a drop in blood pressure of at least 30 mmHg systolic or 15 mmHg diastolic within three minutes of standing, in the absence of dehydration or medications that could explain it.[28] Severe urinary retention or incontinence within the first five years is also concerning, unless it is part of a long-standing or mild stress incontinence history or attributable to prostate disease.[28] Recurrent falls (more than one per year) due to balance issues within three years of onset is another red flag.[28] Additional red flags include disproportionate anterocollis (dystonic neck posture) or contractures of the hands or feet within the first ten years, as well as the absence of common non-motor features of Parkinson's despite five years of disease duration, such as sleep dysfunction (insomnia or symptoms of REM sleep behavior disorder), autonomic dysfunction (constipation, urinary urgency, symptomatic orthostasis), reduced sense of smell, or psychiatric symptoms (depression, anxiety, or hallucinations).[28] Unexplained pyramidal tract signs, such as pathologic hyperreflexia or pyramidal weakness, is also detailed as a red flag, as is bilateral symmetric onset, where both sides of the body are equally affected from the beginning with no side predominance.[28]

The diagnostic thus process follows a systematic approach. If a patient meets the basic movement symptom criteria (bradykinesia plus either rest tremor or rigidity), the neurologist first checks for absolute exclusion criteria.[28] If any are present, Parkinson's is ruled out.[28] If no exclusion criteria are found, the neurologist then assesses the number of red flags and supportive criteria.[28] If the patient has at least two supportive criteria and no red flags, they meet the criteria for Clinically Established PD.[28] If there are no more than two red flags, but the number of supportive criteria equals or outweighs them, Clinically Probable PD can be diagnosed.[28]

A diagnosis of Clinically Established PD requires three conditions: the absence of absolute exclusion criteria, the presence of at least two supportive criteria, and no red flags that could indicate an alternative condition.[28] Clinically Probable PD, which allows for some uncertainty, also requires the absence of absolute exclusion criteria, but it permits the presence of red flags, provided that they are counterbalanced by supportive criteria. If one red flag is present, at least one supportive criterion is needed to offset it.[28] If two red flags are present, at least two supportive criteria are required.[28]

Why Might Someone Develop PD?

A person's risk of developing PD is influenced by a complex interplay of risk factors.[6,31-33] Risk factors are characteristics or conditions that may independently or in combination increase the likelihood of (but do not necessarily guarantee) a person developing PD. Risk factors can be extrinsic (e.g., environmental factors, lifestyle behaviors, physical factors) or intrinsic (e.g., genetic vulnerability, medical comorbidities that may place one at higher risk of PD).[16,34] Some risk factors are modifiable (such as physical inactivity), whereas others are nonmodifiable (such as genetic mutations).[35]

General examples of PD risk factors include genetic vulnerabilities (e.g., pathogenic mutations in SNCA, LRRK2, GBA, parkin, PINK1, DJ-1, ATP13A2, PLA2G6, FBXO7; VPS35; ATP13A2; PLA2G6; FBXO7; SMPD1; APOE; ATP1A3, C19orf12, CSF1R, DCTN1, DNAJC6, FTL, GCH1, GRN, LYST, MAPT, OPA3, PANK2, PRKRA, PTRHD1, RAB39B, SLC30A10, SLC39A14, SLC6A3, SPG11, SPR, SYNJ1, TH, TUBB4A, VPS13A, and WDR45)[36-38]; environmental factors (e.g., exposures to air pollution/particulate matter,[39] certain chemicals, micro/nanoplastics,[40-42] pesticides);[43] medical comorbidities (e.g., history of head trauma,[44,45] certain infectious diseases[46-49] and autoimmune conditions,[50,51] diabetes/prediabetes,[52] cardiovascular disease,[53] PTSD,[54-56] upper gastrointestinal mucosal damage as may occur in GERD)[57,58]; and lifestyle factors (e.g., physical inactivity, certain dietary choices).[59] Also among non-modifiable risk factors are older age (PD is rare in individuals under age 50, with increase in incidence after age 60[60]), male sex,[61] and positive family history.[62,63]

While the presence of risk factors may increase the likelihood of developing PD, no single risk factor is categorically deterministic. This means that no risk factor's presence absolutely guarantees disease onset; many individuals with known risk factors never develop PD, and conversely, some individuals without identifiable risk factors nonetheless develop PD. Most cases of PD (estimated at around 70-80%) are considered idiopathic, meaning that they arise without a definitive, singular known cause.[64,65] Even when a specific cause is uncovered, the presentation can vary considerably across individuals, underscoring the multifactorial and heterogeneous nature of PD and its progression.[65,66]

Notably, risk factors do not necessarily constitute mechanisms of causation. While epidemiology frequently identifies risk factors through the observation of statistical associations with a disease on a population level, risk factors themselves do not inherently carry sole causal power to drive disease pathogenesis.[67] For example, while being male is a risk factor for PD (insofar as male sex is associated with increased risk of developing PD), being male does not cause PD. Risk factors alone, derived from population-level statistical associations, neither provide mechanistic explanations nor establish individual-level disease causation.[68] I defer to Dr. Goodman's meticulous report discussing PD causation more generally.

Prognosis and Management of PD

The prognosis of PD typically involves progressive neurodegeneration, leading to increasing motor and nonmotor symptom burden; however, the tempo and nature of symptom progression varies considerably among individuals.[69] Even as PD leads to deterioration in neurological function, quality of life may be maintained through multifactorial symptom management involving multidisciplinary care and therapeutic management.[70,71] Dopaminergic pharmacotherapy, lifestyle interventions (e.g., exercise-based strategies), and careful symptomatic treatment, are among main strategies to manage

PD, and neurotechnological approaches such as deep brain stimulation (DBS) may also play a role in some cases.[72,73]

6. Independent Medical Examination

On April 24, 2025, I performed an independent medical examination of Mr. Richard Sparks. Mr. Sparks was alert, appropriately groomed, seated in an office chair, and breathing comfortably on room air. Mr. Sparks shared that he was born in 1953 at St Mary's Hospital in Kansas City, Missouri. He grew up in Shawnee Mission, Kansas, in what he described as an unfinished basement "with no running water," where the family borrowed water from relatives, bathed "once a week in the pond," and used an outhouse. He had 7 siblings growing up, 4 sisters and 3 brothers. He recalled that his parents divorced in 1967, and he continued to live with his mother. He attended De Soto schools, earned excellent grades, but left two weeks into the 11th grade because he "wanted to get away." He then lived and worked with his father for a short period of time before enlisting in the Marines. Prior to enlisting in the Marines, Mr. Sparks worked with his father for approximately 1-1.5 months, and recalled that his primary responsibilities included fueling up tractors and heavy equipment, pumping gas into vehicles. He recalled wearing gloves but did not wear a mask. Mr. Sparks met a recruiter in a nearby town and "within a week" was told to "pack his bags." He recalled that boot camp began in March 1971 at the San Diego Recruit Depot and lasted thirteen weeks. Later, at Camp Pendleton he was assigned to the Truck Company where he recalled his responsibilities included driving trucks. In 1972 Mr. Sparks completed Embassy Marine Security Guard School in Washington, D.C., and was posted to Rio de Janeiro, Brazil. Mr. Sparks described a motor-vehicle collision resulting in head injury when in Brazil: while riding in "an old-fashioned Suburban" on "wet cobblestones," the vehicle "sideswiped several parked cars," and his head struck the dashboard. He was "probably not wearing a seat belt," recalled spending about an hour in the emergency department, and required sutures to the right eyebrow and chin.

Separately, upon his return to the United States, he recalled being at a Naval Hospital possibly near Bethesda. Mr. Sparks was thereafter sent to Camp Lejeune, where he arrived in March 1974, where he served as a truck driver, disbursement specialist, and motor-pool gunnery sergeant, and was eventually promoted to E-5. Mr. Sparks married, and his daughter was born in March 1975. Mr. Sparks received an honorable discharge in June 1975.

Following military service Mr. Sparks returned to Kansas, and recalled working twelve-hour shifts at a Kansas gasoline service station, pumping fuel, and maintaining inventory. Mr. Sparks estimated that he worked there for 6-7 months. In the years that followed he lived with his family intermittently in Brasília, Brazil doing miscellaneous work including teaching English, consulting on Embassy security, and doing some construction work such as installing perimeter barriers. Mr. Sparks recalled working for around 2 years at Greyhound during an intercurrent return to the U.S. before returning to Brazil. Mr. Sparks returned with his family to the U.S. and then moved to Albuquerque where he worked at a used car dealership before joining the U.S. Customs Service in Laredo, Texas, in 1991, where activities included monitoring and inspecting transfers/shipments at the Southern border.

Mr. Sparks described a 1996 incident while on duty in which he "inhaled some dust that came through a shipment": during a night shift there was a need to inspect cargo on a shipment, a forklift

punctured a container, releasing dust. "My job was to keep it isolated… we treat everything as if it was hazardous," he said, estimating he remained within the area for twenty minutes and "apparently inhaled it." Mr. Sparks recalled that it was not an illicit substance but rather dust that was released from a container that was primarily filled with "plastic pellets". The next morning, Mr. Sparks developed constant abdominal cramps that kept him out of work. He went to his primary care doctor, in Laredo Texas, Dr. Benavides, who diagnosed a gastric ulcer, and required of diet of soft foods. He resigned within a week. "When this happened to my stomach I put two and two together and figured it could have been hazardous material". He then took university courses with his son, and also worked as a police dispatcher during this period. Mr. Sparks recalls re-entering federal service in 2001 with U.S. Customs and Border Protection in Denver, later receiving a Commissioner's Award, and in 2008 becoming Homeland Security attaché in Lisbon, Portugal, overseeing radiographic and radiation screening of maritime cargo. No inadvertent radiation exposure incidents occurred to his knowledge while there.

While in Lisbon, Mr. Sparks noticed a tremor of the right hand that later extended to the right leg; a Portuguese physician monitored the tremor. After returning to Denver he noticed the tremor had worsened as well as his balance. This later led to a diagnosis of PD. Mr. Sparks noted that initial response to levodopa-carbidopa was satisfactory, but over time dosage frequency increased and effectiveness has diminished; he now takes two tablets every two to three hours while awake. He reports progression to bilateral tremor, rigidity, bradykinesia, freezing of gait and speech, drooling, anosmia ("sense of smell nonexistent"), urinary urgency, constipation, vivid sleep dream enactment behaviors, and cognitive decline he calls "a jumble." Sparks notes that he has frequent falls. On one occasion he broke three ribs following a fall, describing a persistent sensation "like an entity is physically shoving me." He reported sometimes seeing dark shadows in the corner of his eye. Mood is described as a "constant battle, and it is getting worse and worse." Lower back pain limits prolonged standing and is relieved by walking. Mr. Sparks describes that two vocal-fold surgeries performed at the University of Colorado Anschutz "did not help". Mr. Sparks retired in 2017. Current physical activities include performing stationary cycling up to thirty minutes per week "if I'm lucky." Mr. Sparks no longer drives, negotiates stairs slowly, manages bathing and finances but needs help with socks and shoe-tying, and limits outings to places within fifteen minutes of a restroom. Mr. Sparks related that he started painting 3 years ago, which he did for about 1-2 years, and has made over 100 paintings, but now finds that he has a harder time with energy and focus to paint. He does not use assistive devices. Current medications are carbidopa-levodopa, sertraline each morning, mirtazapine at bedtime, vitamin B12, and vitamin D3. The family history that Mr. Sparks recalled include a brother who died in 2021 or 2022 with "dementia," a father who died of lung cancer at sixty-nine, and a mother who died of a "brain infection" at seventy. Mr. Sparks resides with his wife and an adult grandson who assists with yard maintenance. Mr. Sparks is not currently engaged in active physical therapy (PT), occupational therapy (OT), or speech and language therapy (SLP).

During the examination, Mr. Sparks was in no apparent distress, breathing comfortably on room air. Mr. Sparks remained cognitively engaged during the ~3.5 hour interaction, demonstrating insight, cooperative demeanor, and preserved and fluent communication. His speech was not dysarthric though mildly hypophonic with reduced blink rate. Extraocular movements were intact, facial sensation was symmetric, and he had no difficulty turning his head or shrugging his shoulders. Hypomimia was evident. There was no pronator drift. There was a mild R>L resting tremor. Rapid alternating movements revealed mild motor block R>L, and finger-to-nose testing was intact. Praxis and higher-level sequencing (Luria) were preserved. Drawing was constructionally intact but mildly

micrographic (including clock and cube). Serial 7s were performed correctly. Abstraction was intact. Mr. Sparks transitioned from a seated to standing position independently and ambulated cautiously with reduced arm swing without assistive devices, and en bloc turns.

7. Mr. Spark's Relevant Medical History

Review of the available medical records reveal the following additional details. Mr. Richard Sparks is a 71-year-old, right-handed male, born in Kansas City, Missouri (one of eight children) served in the U.S. Marine Corps from 1971-1975, rising to Sergeant, trained as a truck driver and disbursing clerk, and posted as an Embassy Security Guard in Rio de Janeiro, then worked variously in fuel service, Greyhound terminal operations, and, for more than two decades, as a U.S. Customs and Border Protection officer (ultimately Homeland Security attaché in Lisbon) until retiring in 2017; his medical history includes remote head injury, bilateral retinal tears (repaired), right-eye cataract, sensorineural hearing loss, hyperlipidemia, pre-diabetes, GERD, ventral hernia, obstructive sleep apnea, granulomatous lung disease, basal cell carcinoma, melanoma, actinic keratosis, benign prostatic hypertrophy, cervicalgia, left knee pain, tubular adenoma, mood and sleep disorders, prior throat and spine surgeries, vitamin B12 deficiency, and a constellation of gradually progressive neurological symptoms beginning in the late 2000s with asymmetric tremor, bradykinesia, rigidity, motor fluctuations, and mild cognitive impairment that led to a diagnosis of PD.

From 1971 to 1975, Mr. Sparks served in the United States Marine Corps, where he was initially trained as a truck driver and disbursing clerk. During his service, he was selected for specialized State Department training in diplomatic security and was assigned as an Embassy security guard in Rio de Janeiro, Brazil.

From 10 October 1973 to 16 November 1973 Mr. Sparks was treated at the National Naval Medical Center in Bethesda Maryland. He was referred to National Naval Medical Center for psychiatric evaluation and disposition. On admission the general and neurological examinations were normal, and "[a]ll indicated laboratory examinations, including electroencephalography [(EEG)], were normal with the exception of a stool examination which revealed trichuris trichuria ova." Mr. Sparks was placed on appropriate antiparasitic medication for the trichinuria discovered on admission and subsequent stool examinations were normal. The narrative summary of the clinical record chronicles "several aggressive episodes:"

> One night, while he and other marines were in a local bar, a friend became involved in a fight with a Brazilian. The patient came to his friend's aid, but he had to be restrained from seriously harming the Brazilian. Later, while driving in a car with other marines, the patient found the road blocked by a Brazilian's car. The patient became enraged and had to be again restrained by the other marines lest he harm the Brazilian. Two weeks prior to admission, the patient was riding with his girlfriend and another marine in a government vehicle. The patient claimed he became unaware of his surroundings, and in that state was involved in a multi-car accident, hitting two cars and a tree. After each of these episodes, the patient related that he had "gone blank" and had no conscious reason for his behavior at those times. His command became concerned about these episodes and his overall conduct and referred him for psychiatric evaluation. He was first seen by a Brazilian general practitioner, but because of security reasons he was transferred to Panama to see a military psychiatrist. After a brief evaluation, the patient was tentatively diagnosed as having a Chronic Anxiety

> Syndrome and Acute Dissociative Reaction.…He was preoccupied with anger at the Marine Corps and repeatedly stated, "I'm tired of being told what to do all the time." He also expressed the firm wish to be out of the Marine Corps "psychiatrically or any other way."

The narrative summary also addresses Mr. Sparks's early homelife and military career:

> The patient's early homelife was characterized by constant fighting between his parents. His father had a brain tumor which was excised but left him in a chronically irritable condition. The patient's parents eventually divorced when he was 14. At present the patient is quite concerned about his mother's economic well-being. The patient was considered a chronic disciplinary problem in school. He dropped out at age 15 and returned to school at age 16. He dropped out again because he "was tired of being told what to do." The patient impulsively left home when his mother gave him an ultimatum of going to school or getting a job. He joined the Marine Corps in March 1971 to prove he could do it. He immediately disliked Marine Corps regimentation. He sought and achieved a placement in Rio as a security guard at the U.S. Consulate in order to escape from some of the Marine Corps regulations. At present he is rated as a truck driver.

Mr. Sparks's hospital stay at the National Naval Medical Center in Bethesda was:

> initially a calm one with passive acceptance of hospital routine. At first he displayed some anger toward the Marine Corps for passing over him in promotion and he minimized the impact of his aggressive episodes. He remained socially isolated, contributing little to ward interactions. Although he was initially preoccupied with his desire to leave the Marine Corps, after a period of time his anger toward the Marine Corps abated. He then tried to arrange to return to duty, but only if he could return to his previous post. When this did not seem feasible he became angry, dejected, and explosive. He acquired no insight into his feelings and gained no control over his explosive behavior in spite of intensive therapeutic efforts in that direction. He required no medications and at no time did he exhibit any dissociative symptoms. Psychological testing revealed a generally immature personality pattern with evidence of passive-aggressive traits, authority conflicts, and poor impulse control.

While at the Naval Medical Center in Bethesda, Mr. Sparks was diagnosed with:

> Passive-Aggressive Personality #3018, EPTE, manifested by excessive passivity and lack of self-assertion, followed by angry, violent outbursts, marked resentment of authority, manipulative behavior, and obstructionism. Predisposition marked (chaotic home environment, inconsistent parenting, history of disciplinary problems in school); precipitating stress none (routine military duties); impairment severe (unsuitable for further military service)."

(00682_SPARKS_NARA_0000000004-6). Mr. Sparks was subsequently transferred to Camp Lejeune in March 1974 where he worked as a truck driver and disbursement clerk. On June 1, 1975, Mr. Sparks was honorably discharged with the rank of Sergeant (E-5).

After leaving the Marine Corps, from 1975 to 1978, Mr. Sparks taught English as a foreign language in Brasília, Brazil. Between 1976 and 1977, he worked as a bilingual (Portuguese) sales agent for Travelodge Corporation, providing telephone information services to tourists at the Travelodge

phone center in Shawnee Mission, Kansas. From 1977 to 1978, Mr. Sparks managed a full-service gas station in Independence, Missouri. Between 1978 and 1984 Mr. Sparks worked as a terminal employee at Greyhound Bus Corps, where responsibilities included "ticket counter, baggage, freight, and telephone information. Spent 3 months as terminal manager and dispatcher, supervising approximately 50 employees." (00682_SPARKS_DHS_0000000200). Mr. Sparks later worked as a Customs Inspector on the Southern Border. Mr. Sparks described that between 5/1995 and 3/2000, "[w]hile recovering from injuries suffered while on duty as a Customs Inspector, I lobbied in Washington for more money for the Customs Service for better training, more personnel and safety equipment. I was credited with the approval of an additional $120 million in funding." (00682_SPARKS_DHS_0000000144). Later writing that "In June of 1995 I was "nondisciplinarily discharged" from the U.S. Customs Service due to "injuries suffered in a chemical spill while on duty." These injuries have since resolved." (00682_SPARKS_DHS_0000000149). Mr. Sparks later returned to work with U.S. Customs and Border Protection and Homeland Security, and between 2008 and 2013 Mr. Sparks lived and worked in Portugal. Mr. Sparks was responsible for maritime cargo inspections in Lisbon.

The following is a recitation of certain pertinent entries from Mr. Sparks' medical records:

On 12/18/2013, shortly after repatriating from his Homeland Security post in Portugal, Mr. Sparks presented to Dr. Julia Clemons at UCHealth Internal Medicine to establish primary care. He reported a one-week viral-syndromic illness, non-productive cough, rhinorrhea, congestion, fever with sweats, myalgias, and diarrhea, for which supportive therapy was advised. More significantly, Dr. Clemons documented a five-year history of an asymmetric resting tremor that began in the right hand and had recently spread to the right leg. It was noted that Mr. Sparks had previously undergone an EMG and had been treated for four years with ropinirole-LP 4 mg and trihexyphenidyl 2 mg three to four times daily, yet he noted worsening tremor, occasional "stumbling into doorways," and a gait change observed by his wife, although he denied falls or depressive symptoms. Dr. Clemons thereupon initiated a neurology referral. (00682_SPARKS_UCH_0000003234).

On 1/2/2014, Mr. Sparks was evaluated by fellow Dr. Jessica Hedeman at UCHealth neurology clinic with Dr. Leehey attending. At that it was noted that Mr. Sparks:

> First symptoms of PD were about five years ago with shaking in the right hand at rest. Then he developed problems with dexterity and problems with writing and doing buttons. A couple of years ago he developed tremor in the right leg. Was living in Portugal at the time and saw a neurologist who started him on Requip which has helped with tremor. Also tried Propranolol with no benefit. Artane was also added early which has been very helpful for tremor. Just moved from Portugal 2 weeks ago and thinks Portuguese Artane was more effective. Also feeling stress right now with the move, time change, starting a new job, etc. His tremor has been much worse in these past two weeks. Tremor has been present throughout the day and quite bothersome. He works in customs and his new position is at the airport in border control and needs to carry a weapon. He is concerned about passing an exam next week in which he needs to hold a weapon. Biggest problems now are writing, eating soup with a spoon, doing little buttons. No symptoms at all on the left side of his body. Has cramps with "aches and pains" in the right arm. Other symptoms include quieter and mumbling speech. No loss of sense of smell. Posture has become a little stooped. Sleeps well at night. No changes in bowel or bladder function. Memory is a little changed in that he

> repeats questions to his wife. He is under more stress right now with a job change, moving across seas and getting back in uniform." Exam was notable for MOCA 25/30 with points deducted for language and delayed recall; mild hypophonia; R>L cogwheel rigidity, bradykinesia; tremor; reduced arm swing with gait. Plan included increasing "Ropinirole to 6 mg XR from 4 mg. Side effects discussed. New prescription for non-generic form of Artane. Will discuss possibility of getting Artane from Portugal if new medication not beneficial. Trial of Alprazolam 0.25 mg PRN tremor. Can increase to 1 mg slowly if needed. MRI Brain rule out structural process given strictly unilateral disease after 5 years. Referral to Dermatology for skin check with increased risk of melanoma in PD. Gave information on PD support groups and resources locally. He will call next week with response to above. Consider addition of Levodopa. Addition of MAO-I in future visits. We will plan to have Mr. Sparks return to clinic in 6 weeks with Dr. Hedeman, with Dr. Leehey attending.

(00682_SPARKS_UCH_0000003200-1).

On 2/12/2014, Mr. Sparks returned to the UCHealth Neurology clinic for follow-up with Dr. Maureen Leehey. His Parkinson's trajectory was described as characterized by strictly right-sided manifestations, resting tremor, rigidity, and bradykinesia of the upper and lower extremities, and reported marked tremor improvement on the regimen instituted six weeks earlier (trihexyphenidyl, ropinirole XR 6 mg, and intermittent alprazolam). Examination confirmed persistent unilateral signs without new motor or non-motor complications. Dr. Leehey advanced disease-modifying therapy by increasing ropinirole XR to 8 mg daily, with instructions to introduce rasagiline (Azilect) 1 mg each morning after two weeks. Dr. Leehey also advised gradual tapering of alprazolam and trihexyphenidyl as higher-dose ropinirole and rasagiline took effect, encouraged continued range-of-motion and aerobic exercise, and deferred levodopa initiation. Follow-up was scheduled for three months. (00682_SPARKS_UCH_0000003165).

On 11/5/2014 ENT specialist Dr. Clary evaluated progressive hoarseness beginning December 2013. Parkinson's disease was "well controlled," but Mr. Sparks described a voice that tired and became rough over the day, plus throat-clearing without dysphagia or reflux. Voice therapy, LSVT (Lee Silverman Voice Treatment) and possible vocal-fold augmentation were advised, with review at three months.

On 11/6/2014 Mr. Sparks was re-evaluated by Dr. Leehey in the UCHealth Neurology Clinic. At that visit it was noted that:

> First symptoms of PD were about 2008 with shaking in the right hand at rest. Then he developed problems with dexterity, writing and doing buttons. In 2012 he developed tremor in the right leg. Was living in Portugal at the time and saw a neurologist who started him on Requip which has helped with tremor. Also tried Propranolol with no benefit. Artane was also added early which has been very helpful for tremor. Moved from Portugal in 2013 and thinks Portuguese Artane was more effective. Has to take every 3–4 hours, when working takes 5 per day, when not working takes 3 per day. He works in customs at the airport in border control and needs to carry a weapon. On current therapy has been able to pass exams given every 3 months which he needs to hold a weapon. Without the Artane the tremor is persistent and interferes at work. Denies side effects from Artane, including memory problems. Tremor interferes with writing, eating soup with a spoon, doing little buttons. Other major issue is that he is lightheaded when stands up and sometimes after standing for

> a while. No CP or other cardiac symptoms. He often eats poorly and goes for many hours without eating while at work. No symptoms at all on the left side of his body. Has cramps with "aches and pains" in the right arm.

This exam was notable for increased tone with cogwheeling R>L; mild resting tremor; bradykinesia; reduced arm swing with gait. Tremor was noted to be "adequately controlled on current regimen of Artane (6-10mg per day), Ropinorole and Xanax." MRI brain was noted to be "unremarkable". Recommendations were made for dietary optimization, speech therapy, PT, exercise routine. (00682_SPARKS_UCH_0000003060).

On 11/13/2014 Mr. Sparks underwent voice evaluation and therapy with Juliana Litts, MA, where it was described that Mr. Sparks experienced:

> sudden onset of voice problems about 11 months ago. Precipitating factors included: moved back to Colorado in December 2013 from Portugal. He complains of decreased volume and he complains of hoarseness or a "frog" feeling in his throat. He occasionally gets normal voice, mostly in the morning. His voice tends to get worse during the day, and he is almost aphonic. He is a customs officer at the airport. He is short of breath most of the time, but he believes he is still adjusting to the altitude. He does not have any problems swallowing. He has a diagnosis of early onset Parkinson's that he received about 5 years ago. His symptoms include weakness in his right hand and leg as well as some tremors, but his symptoms are controlled by medication.

Impressions included "muscle tension dysphonia, bilateral vocal cord paresis" with recommendations for voice therapy which he subsequently received. (00682_SPARKS_UCH_0000003056).

On 6/5/2015 Mr. Sparks was evaluated by Christen Epstein, NP in the UCHealth Neurology Clinic, with primary concerns involving "fatigue, progression of tremor and more difficulty with dexterity" as well as "some word finding and short term memory difficulties, which are likely related in part to Artane" as well as "occasional dizziness." At that visit it was recapitulated that:

> First symptoms of PD were around 2008 with right hand resting tremor. He is currently taking Artane 2 mg (5–6 times a day) for tremor. He does endorse some trouble with short term memory and word finding on this dose. Also taking Requip 8 mg XL in the morning. Denies ICD, hallucinations, pedal edema. Initially Artane was really helpful for tremor, however he notes progression primarily within the last year. Notes worsening right hand tremor and dexterity. Typing is much more difficult. Difficulty eating, especially with spoons. Doesn't like to go out in public as much. Has to concentrate more with walking. Had one fall since last seen, did not feel it was really PD related. Tripped while walking down stairs into his garage. Notices progressing hypophonia, people often ask him to repeat himself. Swallowing okay. Does cough when throat is dry, not related to eating or drinking (likely SE from Artane). Endorses lightheadedness at times. No syncopal episodes. Endorses fatigue, "feels exhausted all the time." Sleep is okay, occasionally has difficulty falling asleep (attributes this to tremor keeping him awake). No acting out dreams. Mood is okay, "feels frustrated." Has not taken Xanax for 3–4 months and feels anxiety is under control. Has not seen dermatology recently.

(00682_SPARKS_UCH_0000003010). Exam at that visit was notable for moderately increased tone with RUE cogwheeling and mildly increased tone in RLE, neck and LUE and LLE; R>L bradykinesia; resting tremor on R; reduced RUE arm swing; negative Romberg. Recommendations included initiation of amantadine; exercise; and orthostasis interventions.

10/26/2015 Mr. Sparks was evaluated by Christen Epstein, NP. It was reported that:

> Since his last visit he feels like he is having more bad days than good days. He is having increased tremors on his right side UE>LE. On the last visit he added Amantadine which seemed to help really well by decreasing fluctuations and decreasing tremor. This helped for about a month and then started to wear off. His tremor and his voice are the most bothersome feature at this time. His tremor is most responsive to Artane. Takes about 20 min for Artane to kicks in, and it wears off in a couple hours. Symptoms get worse at the end of the day. Voice: His voice has been decreasing in amplitude over the past year, tried LSVT but forgets to practice exercises at home. Tremor: His tremor is mainly on his right side and occurs mainly at rest. His fine motor skills have decreased, is unable to do buttons, can't write, can't eat soup or cereal. Has never seen OT. Balance: Has not fallen. But has had several near falls. Feels unsteady a lot of the time. Never uses assistive devices. Has never done PT. Not exercising formally. Does walk a lot at work and constantly stretches. Denies freezing of gait. Endorses right arm stiffness. Still having light headedness with position changes. Has been drinking a quart of water a day and changing positions slowly. Neither of these have been helpful. Swallow: No choking but often coughs while eating or drinking. Always feels like something is stuck in the back of his throat. Has not seen a dermatologist this year. Is having trouble with short term memory. Hard time focusing. Denies hallucinations. Denies compulsive behaviors. Always feels fatigued and groggy. Not taking naps during the day. Sleeps well at night, denies vivid dreams, denies acting out dreams. Has never triad Sinemet in the past…thinking about retiring soon. Would like to work one more year, but is unsure if he can with his symptoms.

Exam revealed MOCA 26/30; moderate hypophonia without dysarthria and mild hypomimia on cranial nerve examination. Motor strength was preserved at 5/5 in all extremities, moderately increased cogwheeling in the right upper extremity and mild cogwheeling in the left upper extremity, along with bradykinesia that was mild to moderate on the right and slight to mild on the left. There was a frequent resting tremor on the right side that was moderate in amplitude, accompanied by slight postural and mild action tremor on the right. An intermittent tremor of the right leg was also noted. No tremor was observed on the left side. With respect to gait, the patient demonstrated reduced right arm swing and tremor breakthrough during walking, though stride length and turning remained normal. The pull test was negative, and coordination testing (finger-to-nose) was intact without dysmetria. (00682_SPARKS_UCH_0000002966-68). Recommendations were made around orthostasis, increasing amantadine for tremor and fatigue, decreasing artane given memory concerns, voice exercises, and "discussed DBS as an option in the future."

5/5/2016 Mr. Sparks followed up with Christen Epstein, NP. Interim history at that time noted that Mr. Sparks was:

> Off amantadine, caused SE [side effect] of rash and extreme edema. Has since started Sinemet for management of tremor. Taking 1 tab at 8 am, 12 pm, 4 pm, and 8 pm. Notes more breakthrough at end of dose and when stressed. Tremor interferes with writing. Right

> leg tremor often occurs when sitting. Plans to retire next spring. Concerned he won't be able to qualify with reloading his weapon. Feels comfortable firing, but slow to reload due to worsening dexterity. Works for customs/border control. Denies dyskinesia. Taking Requip 8 mg XI in am. Denies ICD, hallucinations, sleep attacks. L ankle has been painful for last 3-4 months. No cramping or turning in of foot. Right toes sometimes curl down, not painful or impacting gait. Doesn't appear to respond to Levodopa. Taking Artane 2 mg BID-TID. Tries to limit this as he notes worsening short term memory on higher doses. No falls. If standing for prolonged periods, he will often reach out and hold on to something to steady himself. Had collagen injections with Dr. Clary and this was very helpful for hypophonia. Denies problems with swallowing. Medications make him sleepy. Sleeping well. He does act out dreams but this is very infrequent. No injury. Recently went to Brazil and hopes to retire there next year. Follows up with dermatology recently. Last exam negative 3 months ago. Not exercising but walks a lot at work.

Exam at that time was notable for mild hypophonia, no dysarthria, mild hypomimia, mild-moderate increased tone in R>L arms, slightly increased tone in neck and lower extremities; mild bradykinesia R>L; infrequent resting tremor of the right side mild in amplitude; slight postural and action tremor; normal rising from chair; arm swing slightly reduced on R; normal stride length; stable turn in 1 step; pull test negative. Sinemet was increased to 1.5 tabs QID, and consideration was given to melatonin for REM sleep behavior disorder. Recommendations for regular exercise were also made. (00682_SPARKS_UCH_0000002825).

On 1/9/2016 Mr. Sparks visited with ENT for "voice changes". It was noted that he had undergone bilateral vocal fold injections on 12/4/2015. He "had significant improvement in voice for approximately two weeks and then noticed a slow decline in the volume of his voice…effectively at pre-procedure baseline." (00682_SPARKS_UCH_0000002921). Plan was made for follow-up and awake injection laryngoplasty with long term agent.

On 8/5/2017 Mr. Sparks presented to IM clinic for "snoring and hearing changes" and it was also noted that "he was recently seen by Dr. Anderson for a hip fracture s/p fall." It was noted that "Mr. Sparks would like to be reevaluated for sleep apnea today. He was supposedly diagnosed in 2007 when he lived in Texas; he tried to wear a CPAP mask, but did not tolerate it." Referrals made to audiology and sleep study. (00682_SPARKS_UCH_0000002531).

On 9/5/2017 Mr. Sparks underwent evaluation in UCHealth Audiology clinic with Laura Campos AuD, who noted "bilateral tinnitus and decreased hearing for the past year….reports noise exposure (firearms, right-handed)". Mr Sparks was found to have hearing loss and was referred for amplification/communication consult. (00682_SPARKS_UCH_0000002520).

On 9/27/2017 Mr. Sparks was evaluated in the VA neurology clinic; increased muscle tone, R>L resting tremor, unsteadiness with pull test, slowed rapid alternating movements, narrow based gait with en bloc turning, unsteady with tandem gait, decreased sensation to vibration in BLE were noted. Rasagaline with added, and ropinorole was increased. Workup was also initiated for peripheral neuropathy. (00682_SPARKS_VBA_0000008985).

On 3/6/2018 evaluated by Dr. Armenia of UCHealth Neurology when it was noted that he had "been having progressive symptoms with a lot of wearing off – carbidopa/levodopa…only last 2.5

to 3 hours at best. Tremor is much worse at end of dose…when lying down he has some dyskinesia to R shoulder and leg." Sleep also noted to be fragmented with dream enactment behaviors, with daytime fatigue and low mood. "Described not to be suicidal…does not want to exercise…did not want to do the advanced treatment class as recommended last visit." MOCA at that visit was 26/30, and exam otherwise notable for bradykinesia, generalized dyskinesia, rigidity mild hypomimia, hypophonia, decreased R arm swing with slower gait. Plan made to start Rytary in lieu of carbidopa-levodopa; start Zoloft; start melatonin; exercise program; "see Dr. Kleptiskaya for advanced therapeutic options", brain MRI and education and treatment of orthostatic hypotension. (0682_SPARKS_UCH_0000002407)

On 5/10/2016 Mr. Sparks presented to Dr. Armenia at UCHealth ENT, for "voice changes that appear to be predominantly due to a combination of Parkinson disease as well as vocal fold atrophy" and was noted to be "doing well after awake bilateral Prolaryn Plus injection on 3/4/2016" with "subjectively improve voice and objectively mildly improved voice from pre, with improved glottic closure." Plan for follow-up was made in 3 months. (00682_SPARKS_UCH_0000002815)

On 8/8/2017 Mr. Sparks was evaluated by Dr. Anderson at UCHealth Internal Medicine for rib pain that followed a mechanical fall at home, prompting home safety evaluation, ambulatory referral to PT, Tylenol PRN and PA/L chest x-rays. (00682_SPARKS_UCH_0000002557).

On 8/15/2017 Dr. Clary of ENT wrote in response to question about whether vocal fold atrophy is a common result of PD that "technically vocal fold atrophy is from aging not necessarily from Parkinson's. It's the combination of the two that leads to significant voice limitations." (00682_SPARKS_UCH_0000002552).

On 11/29/2017 Mr. Sparks was seen by Christen Epstein in Neurology who described that Mr. Sparks had been:

> Taking Sinemet which helps with management of tremor. Taking 1.5 tabs at 9 am, 12 pm, 4 pm, and 8 pm. Plans to increase to 5 times per day (every 3 hours - 6 am, 9 am, 12 pm, 3 pm, 6 pm) – per VA recommendations, but hasn't done this yet. Lasts 2 hours. Tremor is much worse at end of dose and R foot dystonia is worse. When lying down he has some dyskinesia to R shoulder and leg. No urge to move, not relieved with movement. Denies hallucinations. Has sleepiness but no clear sleep attacks. Has continued Ropinirole XL 8 mg (thinks, not entirely clear). Endorses harder time stopping with gambling than in the past – may spend a few thousand dollars. Not going up to Black Hawk as much as he used to as a result. Taking Artane 2 mg in am only. Unless it's a rough day with tremor, he'll take 2 doses. Fell 3–4 months ago going down stairs and broke a rib. Veers to R when walking and bangs arm. Endorses dizziness with standing. Notes he reaches out for things more. Swallowing is "not bad." Denies difficulties. Has GERD – burning with swallowing. Denies constipation. Endorsing R foot dystonia. Stopped PT after 1 month – didn't feel it was helpful. Sleep is really fragmented. Endorses acting out dreams. No injuries. Endorses a great deal of fatigue. Had recent sleep apnea test. Rarely drives due to fatigue and trouble with depth perception. Harder to read because he's so sleepy during the day. Endorses memory concerns. Mood is a little down. He is currently living with his 19-year-old grandson which is working well as his grandson helps with driving to appointments, etc. His wife works for FEMA and is gone for long periods of time for work. Richard has retired as a customs and border protection agent at the airport since his last visit.

(00682_SPARKS_UCH_0000002472). At that visit plan was made to start Trazodone for depression and insomnia, and gradually increate entacapone for motor fluctuations, as well as limiting artane as "this medication appears to exacerbate mild cognitive difficulties." (00682_SPARKS_UCH_0000002477).

On 2/22/2018 Mr. Sparks began experiencing visual hallucinations. He wrote in a message to NP Epstein "I now have new friends visiting me. I see them as shadows out of the corners of my eyes. When I look at them, they disappear. For the most part it is a nuisance….they still startle me sometimes. This is not my failing vision, I see them as shadow people…I only leave the house when it is absolutely necessary…it is getting more and more difficult to function." (00682_SPARKS_UCH_0000002421).

On 4/6/2018 Mr. Sparks underwent cardiac exercise stress study with no evidence of ischemia or infarction; LVEF was 66% with mild LV hypertrophy and no regional wall motion abnormality. (00682_SPARKS_UCH_0000002347).

On 4/6/2018 a MRI of the brain revealed "stable mild volume loss and minimal burden of periventricular T2 hyperintense white matter lesions likely due to vascular risk factors." (00682_SPARKS_UCH_0000002124).

On 8/16/2018 Mr. Sparks wrote to Christen Epstein NP that he had been experiencing persistent depression and "I haven't seen a doctor in several months, and am pretty much self-medicating, not sleeping, not socializing, just marking time. I was prescribed CPAP, but cannot stand it on my face at night, so do not use it." (00682_SPARKS_UCH_0000002240).

On 6/20/2018 Mr. Sparks was evaluated by clinical psychologist Sabrina Schickli, PsyD at the VA, who chronicled that "the veteran was diagnosed with anxiety disorder and passive-aggressive personality existing prior to service. He was referred to mental health due to anger problems and was hospitalized psychiatrically from October 10 until November 16, 1973." The results of neuropsychological testing on August 3, 2017:

> The veteran is a 63 year-old, Caucasian, Right-Handed male, with a diagnosis of Parkinson's Disease. In addition, the veteran has a history of obstructive sleep apnea (not treated with CPAP) and REM Sleep Behavior Disorder. Based on several areas assessed that are typically resistant to decline, his premorbid functioning was estimated to be within the high average range. This was largely consistent with his prior occupational functioning. His early educational history reflects the effects of familial discord, although his later educational achievements are more consistent with this estimate of premorbid functioning. As compared with his estimated average premorbid functioning, the veteran demonstrated largely intact cognitive functioning, with circumscribed areas of likely diminished functioning but not frank impairment. Visual reasoning was borderline, and graphomotor processing speed was low average – these tasks both involve visual processing and working memory. Abstract verbal concept formation, digit repetition and manipulation, visual processing, and mental arithmetic were average but below expected levels when compared to his premorbid status. Language, verbal and non-verbal immediate, delayed and recognition memory, and most aspects of executive functioning were intact. The subtle diminishments in veteran's cognitive functioning revealed by the evaluation are consistent with the effects of

> Parkinson's Disease. Frontal-subcortical system involvement is typically implicated in Parkinson's and is associated with declines in mental processing speed, visuospatial abilities, and executive functioning. While most aspects of executive functioning were intact, the veteran exhibited weakness in processing speed and aspects of executive functioning that involved visuospatial ability. Veteran's minor depressive symptoms and sleep apnea would not fully account for these deficits. He meets criteria for a Mild Neurocognitive Disorder due to Parkinson's Disease. The findings from this evaluation indicate that the veteran should continue to be monitored for changes in cognitive status over time. He may want to pursue consultation with neurology and/or geropsychology to implement compensatory strategies and approaches that might mitigate further decline. He would benefit from psychological interventions to improve his mood. He should be assessed for a CPAP and should begin using this device. The implications of this evaluation is that he may encounter some difficulty when faced with novel situations in which he is required to rely upon his own judgment or problem-solving abilities. He will likely perform better in highly routinized situations that require minimal new learning. Based on results of this evaluation, he retains capacity to manage his financial affairs at this time. This may need to be reassessed if there is evidence of further decline in the future." Despite his depressed mood, the veteran denies any active suicidal thinking… The veteran has chronic sleep disruption. He is in bed at 10 or 11 PM but is up at 2 or 3 AM and never later than 4 AM. … sees "my shadow friends" in his peripheral vision. He says these appear to be human shapes the color of smoke… The veteran is able to do the laundry and wash dishes. His wife primarily does the cooking. He limits his driving and he does not drive at all at night due to his impaired night vision. …He manages the money every morning. He looks up their accounts on the Internet. He does not describe any financial errors or difficulties…. OPINION: The veteran's depression is at least as likely as not (50% or greater probability) proximately due to or the result of Parkinson's. Rationale: The veteran reports no history of depression predating his Parkinson's disease. He describes his depression as being related directly to the functional and motor difficulties associated with his disease.

(00682_SPARKS_VHA_0000000703-707).

On 11/12/2018 Mr. Sparks was evaluated by Christen Epstein at UCHealth Neurology noting progression in his disease:

> specifically in regard to motor fluctuations and mild dyskinesia. Nonmotor complaints are also a key concern including fragmented sleep and untreated OSA which is likely causing worsening fatigue, attention/concentration, and depression. He also has symptoms consistent with REM sleep behavior, mild cognitive impairment (most recent Moca 26 out of 30, MRI brain unremarkable) and lightheadedness with position changes. His exam is also notable for decreased sensation to pinprick in right toes as compared to left toes. This could be in part related to progressing right foot dystonia, however labs checked to rule out reversible causes in clinic today.

Mr. Sparks also noted that "I think someone is standing on the side of me." Additional symptoms at that visit were described to include hypersexuality, online gambling, fatigue, urinary urgency, lack of motivation, gait instability, dream enactment, OSA not on sleep apnea, sense of "internal vibration", toe cramping. At that visit, Ropinorole was decreased, and carbidopa-levodopa was increased with plan for close monitoring. (00682_SPARKS_UCH_0000002118-2128).

On 3/18/2019 notice was sent by Dr. Hassett to Mr. Sparks indicating that "recent lab testing revealed a functional B12 deficiency" with elevated methylmalonic acid; oral supplementation was prescribed. (00682_SPARKS_VHA_0000000676)

On 5/4/2021 Mr. Sparks was evaluated by Dr Griffith for:

> evaluation of dyspnea on exertion associated with increased abdominal girth and granulomatous disease on chest imaging. Differential diagnosis includes history of histoplasmosis, tuberculosis, or sarcoidosis. He has spent a significant portion of his life in endemic histoplasmosis areas including Missouri, North Carolina and Brazil. He has also spent more than 10 years living in a country with endemic spread of TB (Brazil). He has no family history of sarcoidosis and is an older white male which makes this diagnosis less likely but remains a possibility. No evidence of latent TB on quantiferon. Normal PFTs and no evidenc[e] of tree-in-bud bronchiolitis or perilymphatic nodularity on CT scan that would suggest active pulmonary sarcoidosis. No prior chest imaging in JLV. EKG wnl [(within normal limits)]. Given totally normal PFTs and imaging findings that do not suggest active sarcoidosis I would not recommend a biopsy for pulmonary sarcoidosis at this time. …Also cannot use CPAP despite OSA diagnosis due to PTSD from an event while living in Portugal. Will refer to dental for evaluation of mandibular advancement appliance.

(00682_SPARKS_VHA_0000000569).

On 9/30/2021 Mr. Sparks presented to UCHealth Eye Center with decreased visual acuity where he was found to have left retinal detachment (00682_SPARKS_UCH_0000001701).

On 10/24/2023 Mr. Sparks presented to physical therapy for decreased mobility, decreased endurance, gait abnormality and low back pain worsened by exertion. A rehab plan was developed. (00682_SPARKS_UCH_0000001059-80).

During a Functional Assessment on 4/4/2024 it was noted that "Veteran reported difficulty with sleep onset due to physical discomfort; dx of OSA and received CPAP 4 years ago, but was unable to tolerate the sensation, discussed option of CPAP desensitization but Veteran declined and does not plan to resume CPAP use." (00682_SPARKS_VHA_0000001244).

On 3/12/2024 Mr. Sparks presented to the Emergency Department with "involuntary mouth movements that have been worsening over the last 1 month." Basic labs were checked and were unremarkable. There was no change in symptoms with diphenhydramine. It was requested that he decrease his sertraline dose to 25 mg and follow-up as outpatient with diagnostic considerations including "new extrapyramidal effects from SSRI that was recently started". Mr. Sparks followed up with Primary Care on 4/4/2024 when it was reported that "Per Neurology note dated 3/14/24, recommended to follow w/MH Med management to "address mood stabilization given limitations of increasing sertraline" pt reports only taking 1/2 tab PO Daily. Pt does not think it is his MH medication causing the increased jaw movements, was recently seen by UCH Neuro and recommended to take Sinemet Q3H, which pt was not compliant on previously d/t fear or "tolerance" to medication, reports was educated by Neuro that one can not build tol[erance] to medication and encouraged to take it as prescribed. Since starting to take med as directed, he reports has noticed sxs have improved. Pt also reports he continues to struggle with anhedonia, which wife

is also concerned about "he does not want to do anything, just stay in the bedroom". Pt denies SI/HI or plan." (00682_SPARKS_VHA_0000001256).

On 4/2/2024 Mr. Sparks was seen by attending physician Dr. Trevor Hawkins at UCHealth who noted that Mr. Sparks "has idiopathic Parkinson's disease stage 2 with symptoms starting in 2008. The patient demonstrates progressive asymmetric bradykinesia, muscular rigidity, rest tremor, postural instability that meets UK PD brain bank criteria for parkinsonism." He went on to note that "no other features clinically or radiographicaly to suggest an atypical parkinsonian syndrome or secondary cause of parkinsonism at this time." Of note, Mr. Sparks told Dr. Hawkins that "he was last seen about 2 years ago at the VA," and "[h]e has been experimenting with his own dosing and not following instructions." Dr. Hawkins reviewed with Mr. Sparks considerations of pharmacologic treatment of Parkinson's disease:

> including common side effects and medication related motor complications. Unfortunately he was under the impression that he could overdose on levodopa and also it would lose its effectiveness the more he took. He states that when he was stationed in Portugal his physician told him not to start levodopa until his symptoms were severe. Today we spent time discussing how there is no over dosing of levodopa and that we monitor clinically for side effects. We also discussed that there is no potentiation or tolerance buildup to levodopa and that it will continue to work decades into his disease course. Therefore there is no reason to hold off or limit dosing especially given his very clear and consistent benefit.

(00682_SPARKS_0000006708-14).

On 7/5/2024 Mr. Sparks was seen in follow up at Rocky Mountain Regional VAMC Neurology by Dr. Penhos and Dr. Feuerstein to establish care. It was noted at that time that Mr. Sparks had been experiencing worsening stiffness, wearing off of medication 3-4 hours after dosing with worsening RUE and RLE tremor, several falls, disrupted sleep; hyperkinetic jaw movements. At that time Mr. Sparks denied cognitive concerns or dysphagia. Exam was notable for full strength, increased tone and asymmetric resting tremor, head dyskinesia, hyperkinetic head movement, normal rapid alternating movements. It was noted that he "is responding well to Sinemet, and this response was observed during his appointment today." Plan was made to change last dose of Sinemet of day to ER to improve sleep quality and provide coverage during the night. (00682_SPARKS_VHA_0000001408). Attending addendum dated 7/25/24 summarized:

> I discussed and evaluated the patient with Dr. Penhos and agree with the assessment and plan as written. Briefly, 70yo M h/o IPD [idiopathic Parkinson's disease] (10+ yrs) who presents for follow up. Overall is doing well, but with interrupted sleep which he attributes to difficulty to turning over in bed and motor symptoms – as such will trial a CR dose at night in place of the IR dose to see if it covers sx. Additionally, patient with dyskinesia that he finds somewhat irritating, not interested in decreasing levodopa or trying amantadine at this time.

(00682_SPARKS_VHA_0000001413).

With respect to Mr. Sparks' mental health over the past several years, on 5/7/2024 VA psychiatrist Dr Mardis diagnosed depression secondary to Parkinson's disease; mood had improved with the new levodopa regimen. Mirtazapine and sertraline were continued (00682_SPARKS_VHA_0000001306-12), and psychotherapy was pursued (0682_SPARKS_VHA_0000001253). On 5/25/2024 Psychologist Dr. Ryan Kimball documented increased physical activity, improved outlook ("I refuse to let it get to me") and better medication adherence, supported by family co-residence (00682_SPARKS_VHA_0000001216). On 7/18/2024 in follow up with Dr. Mardis of VA psychiatry, Sertraline was escalated to 100 mg after PHQ-9 rose from 2 to 10; the potential impact of this medication regimen change on jaw movements was also discussed (0682_SPARKS_VHA_0000001203).

8. Prior Medical and Surgical History

Review of Mr. Sparks' medical history reveals past medical and surgical issues that include but not necessarily limited to the following:

- Bilateral corneal scars due to history of RK in Brazil
- Bilateral retinal tears s/p repair
- Cataract of R eye
- Refractive error
- Basal cell carcinoma
- Pre-diabetes (A1c 5.9% 5/16/2024 -00682_SPARKS_VHA_0000001330)
- Allergic rhinitis
- GERD
- Melanoma
- Actinic Keratosis
- Ventral hernia
- Sensorineural hearing loss
- Tubular adenoma
- Throat surgery (2014 & 2016)
- Anxiety
- Spine surgery for "pinched nerve" (00682_SPARKS_UCH_0000002372)
- Mood disorder
- Left knee pain
- Benign prostatic hypertrophy
- Cervicalgia
- Hyperlipidemia
- Low B12
- Granulomatous disease
- Granulomatous disease (00682_SPARKS_VHA_0000001205)
- Melanoma
- Obstructive sleep apnea

Of these conditions, the following have been described in the medical literature as potential PD risk factors: head injury,[44,45] impaired fasting glucose / prediabetes,[84] GERD,[57] low B12.[85,86]

9. Family History

Mother – asthma
Paternal grandfather – blindness
His older brother, 3 years older than him, passed away in 2021 or 2022 with dementia.
COPD, lung cancer, brain tumor – father
No known family history of Parkinson's disease

10. Medications

Medications prescribed have included:

Ropinirole (Requip)
Carbidopa-levodopa (Sinemet; Rytary [extended-release])
Sertraline (Zoloft)
Trazodone (Desyrel)
Alprazolam (Xanax)
Amantadine
Vitamin D (Cholecalciferol)
Melatonin
Entacapone (Comtan)
Aspirin
Acetaminophen
Lidocaine patch
Folic acid (Folate)
Rasagiline (Azilect)
Sildenafil (Viagra)
Trihexyphenidyl (Artane)
Omeprazole
Vitamin B12 (Cyanocobalamin)
Mirtazapine (Remeron)
Polyethylene glycol (MiraLAX)
Fluticasone nasal spray (Flonase)
Bisacodyl

11. Social History

Review of Mr. Sparks' medical history reveals the following social history:

Married with 2 children and 6 grandchildren.
Former cigarette smoker.
Associate's degree from Donnelly College in Kansas City Kansas in 1976.
Attended Park College (Parkville, Mo.), St. Mary's College (Leavenworth, Ks.), Laredo Community College (Laredo, Tx.), and Texas A&M International University (Laredo, Tx) taking courses centered in Criminal Justice and Psychology.
State Department trained in Diplomatic protocol (1973/1974).

12. Allergies

No known medication allergies.
Amantadine – side effect of rash and edema
Ropinorole 8mg XR – hypersexuality, gambling

13. Assessment

Mr. Richard Sparks is a 71-year-old, right-handed male, born in Kansas City, Missouri (fourth of eight children), who served on active duty in the Marines from 03/71-06/75, rising to Sergeant (E-5), completing Embassy Marine Security Guard School (1972) and postings in Rio de Janeiro and Camp Lejeune (honorable discharge). Afterward he worked in fuel-service operations, Greyhound terminal management, policing/dispatch, at U.S. Customs & Border Protection, and as Homeland Security attaché in Lisbon (2008-2013), before retiring in 2017. His medical history is notable for bilateral retinal tears (repaired), right-eye cataract, sensorineural hearing loss, hyperlipidemia, impaired fasting glucose (prediabetes), GERD, ventral hernia, obstructive sleep apnea (OSA), granulomatous lung disease, basal-cell carcinoma, melanoma, actinic keratoses, benign prostatic hypertrophy, cervicalgia, left-knee pain, tubular adenoma, mood and sleep disorders, vitamin-B12 deficiency. In his mid-late 50's Mr. Sparks sought treatment for a constellation of neuromotor symptoms that ultimately led to a diagnosis of PD.

As discussed in prior sections, PD is a neurodegenerative disorder characterized by dopamine-producing neuronal loss and signaling disruption, resulting in motor features (e.g., tremor, rigidity, bradykinesia, postural instability) and a range of non-motor manifestations (e.g., autonomic dysfunction, sleep disturbances, anosmia, mood changes).

Onset and Progression of PD

Mr. Sparks' Parkinsonian prodrome surfaced in his mid-50s, while stationed in Lisbon (circa 2008–2009), with a resting tremor of the right hand. By 2012 a similar tremor appeared in the right leg. After repatriating to the U.S., Mr. Sparks' neurologic evaluation documented hypophonia; right greater than left cogwheel rigidity, bradykinesia; progressive resting tremor; and reduced arm swing with gait, confirming PD. Subsequent reviews (Dr. Leehey; NP Christen Epstein; Dr. Armenia; Dr. Penhos) have reaffirmed the PD diagnosis. Pharmacotherapy has included escalating dopaminergic medication regimens. Despite these measures Mr. Sparks has advanced to bilateral tremor, dyskinesias, wearing-off every 3-4 hours, freezing episodes, and frequent falls. Non-motor symptoms have included orthostatic light-headedness, urinary urgency, constipation, anosmia, REM-sleep behavior disorder, visual "shadow-person" hallucinations, impulse-control symptoms (hypersexuality, gambling), mood changes, and cognitive changes, susceptible to fatigue and mood. The longitudinal evidence confirms a progressive PD course that limits ambulation, self-care, and vocational capacity.

Etiology and Risk Factors

PD is most frequently idiopathic, meaning no singular cause is definitively identified. Commonly cited risk factors include age, possible genetic predisposition, pesticide exposure, lifestyle factors, certain infections and autoimmune conditions, and history of head injury. In Mr. Sparks' case, potential risk factors include head injury,[44,45] occupational risks (e.g., working in gasoline service station[87-90]), male sex, low B12.[91,92] While each of these factors could, in theory, incrementally contribute to neurodegenerative risk, no single factor can be definitively pinpointed as causative

under the current state of medical knowledge. Even if Mr. Sparks had not been exposed to TCE, it is my opinion, within a reasonable degree of medical certainty, that Mr. Sparks could have still developed PD. Mr. Sparks' Parkinson's disease could therefore be reasonably regarded as idiopathic, potentially arising from multifactorial risk factors rather than from a single, clearly defined cause.

Functional Limitations and Disability

Mr. Sparks' current disability stems from the cumulative burden of idiopathic Parkinson's disease and multiple comorbidities. Tremor, bradykinesia, "off" periods every 2-3 hours, dyskinesias, and frequent falls, limit Mr. Sparks' mobility and motor function. Untreated obstructive sleep apnea, granulomatous lung disease and chronic low-back pain may compound his fatigue and limit endurance. Sensorineural hearing loss, urinary urgency, REM-sleep behaviors, visual "shadow-person" hallucinations, dopaminergic impulse-control symptoms, anosmia, and mood disorder add non-motor morbidity. He manages personal finances and basic self-care but needs assistance with some instrumental activities. Mr. Sparks' cumulative comorbidities diminish safety, independence and vocational capacity. Ongoing multidisciplinary management and collaboration including neurology, physical therapy, speech therapy, mental-health support, sleep medicine and home-safety interventions remain essential to mitigate further decline and preserve quality of life. Such collaboration, along with Mr. Sparks' commitment and adherence to recommended treatment plan, is crucial for addressing both his PD and the range of comorbid conditions impacting his overall prognosis and quality of life.

14. Conclusions

Based on my comprehensive review of the medical records, I conclude the following within a reasonable degree of medical certainty:

1. Mr. Sparks has a well-established diagnosis of PD, supported by comprehensive neurologic assessments, and characterized by progressively worsening motor features and non-motor features.

2. There is insufficient evidence to conclude to a reasonable degree of medical certainty that Mr. Sparks' Parkinson's disease was definitively caused by exposure to contaminated water at Camp Lejeune. My opinions regarding Mr. Sparks' exposure history relied on a review of toxicological evidence of general causation by Dr. Goodman and exposure calculations/risk assessment reports of Drs. LaKind and Bailey respectively. Other causes must be considered in this analysis.

3. Mr. Sparks' PD should be regarded as idiopathic, potentially arising from multifactorial risk factors rather than from a single, clearly defined cause. Potential risk factors in Mr. Sparks' case include but are not necessarily limited to occupational exposures (e.g., in gasoline service station,[87-90] and dust exposure during spill as Customs officer[89,93]), male sex,[94,95] and head injury.[44,45] While each of these factors could, in theory, incrementally contribute to neurodegenerative risk, no single factor can be definitively pinpointed as causative under the current state of medical knowledge; while research has demonstrated increased risk in

various contexts, current scientific evidence and diagnostic science does not allow for precise weighting or ranking of these risk factors individually or in combination in any given person.

4. Mr. Sparks' functionally limiting fatigue is multifactorial. In addition to the contribution of PD, untreated obstructive sleep apnea (OSA) is a major, remediable driver. Although continuous positive-airway pressure (CPAP) remains first-line therapy for OSA, Dr Griffith noted that Mr. Sparks "cannot use CPAP despite OSA diagnosis due to PTSD from an event while living in Portugal" (00682_SPARKS_VHA_0000000569). Alternative OSA treatments such as mandibular-advancement devices, positional therapy, or other advanced options, could therefore be explored in consultation with a sleep-medicine specialist, as improving nocturnal ventilation is likely to enhance restorative sleep, mitigate daytime fatigue, and potentially aid in mood and cognition.[96,97] Importantly, OSA itself is a risk factor for PD and PD progression.[98-101]

5. Symptomatic management with pharmacotherapy, physical therapy, speech therapy, mental health support, and advanced interventions can optimize function and quality of life for Mr. Sparks. Mr. Sparks appears to be under competent, multidisciplinary clinical management, which is appropriate given the complexity of his medical and mental health needs. Re-engagement with physical therapy, occupational therapy and speech and language therapy along with sleep medicine may help to safeguard and optimize function and wellbeing. The late-life emergence of artistic activities that Mr. Sparks experienced (producing over 100 paintings until increasing fatigue led to a pause) is occasionally documented in the context of PD,[102,103] and may be purposefully nurtured through art-based therapeutic programs.[104,105]

6. Ongoing specialized neurologic care, coupled with targeted interventions for mental health, speech and swallow, pain, and autonomic regulation, as well as close monitoring and management of comorbid conditions will remain paramount to managing Mr. Sparks' complex medical presentation.

PD is a progressive and complex disorder that requires specialized, multidisciplinary care, which Mr. Sparks appears to be receiving by a highly competent and caring team. Advances in treatment options continue to evolve, offering opportunities to optimize function and quality of life.[106-109]

Comments on the Expert Report of Dr. Schwarz

I agree with Dr. Schwarz's conclusion that Mr. Sparks has Parkinson's disease. I also generally concur with Dr. Schwarz's assessment of expected PD prognosis, with the caveat that some individuals may defy prognostic expectations, and innovations in PD therapy may in the future alter anticipated prognosis, offering hope to patients afflicted by PD.[110-112]

With respect to etiology, the potential factors considered in Dr. Schwarz's report (page 10) report include head trauma; genetics (family history); drug exposure; environmental exposure; and TCE/PCE exposure. I agree that there is no evidence of a genetic predisposition or family history of PD. There is a history of dementia in Mr. Sparks older brother who passed away several years ago; family medical records were not available for review, however, and it is not clear what type of dementia this brother had. Regarding head trauma, Dr. Schwarz remarks while head injury may be a risk factor, "Mr. Sparks has no history of head trauma and denies head injury" (page 10). This assessment appears to overlook a significant head injury occurred while Mr. Sparks was in Brazil; available records indicate that during this incident Mr. Sparks *"became unaware of his surroundings, and in that state was involved in a multi-car accident, hitting two cars and a tree. After each of these episodes, the patient related that he had 'gone blank' and had no conscious reason for his behavior at those times."* (00682_SPARKS_NARA_0000000004-6).

During the IME, Mr. Sparks recounted this motor-vehicle collision resulting in head injury when in Brazil: while riding in "an old-fashioned Suburban" on "wet cobblestones," the vehicle "sideswiped several parked cars," and recalled that his head struck the dashboard. He stated that he was "probably not wearing a seat belt," required emergency-department evaluation and sutures to the eyebrow and chin, after which he was sent stateside and received treatment at a hospital in Bethesda. This episode constitutes a significant head injury to be considered in the risk-factor analysis. It is notable here that even mild head injuries have been shown to increase PD risk[45,113-115] and may have thus contributed as a risk factor in Mr. Sparks' case.

In discussing environmental exposures, Dr. Schwarz remarks that *"A question was raised about his exposure to diesel fumes while working for a short time at the bus terminal. However, Mr. Sparks testified that he spent little time outside near the buses but was at the indoor ticket counter".* This analysis does not appear to account for potentially significant exposures during the time Mr. Sparks spent working refueling vehicles prior to enlisting (while living with his father after dropping out of high school), as well as after discharge from service when Mr. Sparks reported working for 6-7 months at a full-service gasoline station (00682_SPARKS_DHS_0000000200). Separately, Mr. Sparks' responsibilities at the bus terminal also were reported to span beyond ticketing, to include handling of baggage and freight (00682_SPARKS_DHS_0000000200). Additionally, during his time in the Customs and Border Patrol, Mr. Sparks would have been exposed to exhaust fumes; Mr. Sparks described Laredo, TX as *"one of the largest land border cargo facilities in the United States, so we had a lot of trucks and a lot of rail traffic"* (Sparks Deposition Transcript, 75:23-25). Continuous exposure to gasoline fumes in these occupational roles are likely to have contributed as a risk factor in Mr. Sparks' case and should be considered in the risk-factor analysis.[87-90]

While I agree with Dr. Schwarz that *"Mr. Sparks past medical history is notable for hyperlipidemia, vitamin B12 deficiency (on adequate oral replacement), fatty liver and pulmonary, liver and spleen granulomas likely due to a prior infection" (*page 9), the suggestion that *"[n]one of these diagnoses would predispose him to Parkinson's*

disease" warrants nuance, insofar as B12 deficiency[85,86,116] and prior infections have been variably associated with elevated PD risk.[47,117]

Although various factors may increase the likelihood of PD, no single factor categorically guarantees its onset; many people with known risk factors never develop PD, while some without any known risk factors nonetheless do. Most cases are idiopathic with no clear cause is identified and even when a specific cause is found, presentations vary widely, reflecting PD's multifactorial and heterogeneous nature. Moreover, risk factors do not necessarily constitute mechanisms of causation. They emerge from population-level associations and do not singularly drive disease processes, known to be multifactorial.[65,66] For a more extensive discussion of PD causation, I refer to Dr. Goodman's thorough report and to the discussion provided earlier in this report starting on page 4.

Based on the detailed analysis above, I diverge from Dr. Schwarz's conclusion in that the attribution of causation to TCE exposure does not sufficiently account for the broader range of potential risk factors present in Mr. Sparks' case, including but not necessarily limited to occupational exposures,[87-90] male sex,[94,95] and head injury,[44,45] each of which individually or in combination may serve as a contributor to PD risk. Given the current state of medical knowledge and the multifactorial nature of PD, I thus find that there is insufficient evidence to conclude within a reasonable degree of medical certainty that TCE exposure definitively caused Mr. Sparks' PD. I likewise cannot conclude within a reasonable degree of medical certainty that Mr. Sparks would not have developed PD if he were not exposed to TCE. While one cannot know the counterfactual, it is essential to consider the full spectrum of individualized risk factors rather than attributing causation to a single exposure.[6,118]

All of the above opinions are offered within a reasonable degree of medical certainty. I reserve the right to modify my opinions should additional relevant information become available in the future.

Dr. Michael Young, MD

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