Page 2: Dr. [Name] maintained all prior opinions without amendment. Dr. [Name] reviewed medical records and medical scans.
Page 3: He reviewed the transcript of prior testimony and thousands of pages of medical records. He reviewed remote follow-up notes from the patient.
Page 4-5: the patient completed remote visits with Dr. [Name] in January and March 2026. Dr. [Name] reviewed the complete medical file.
Page 6: He reviewed MRI scans of the brain, cervical spine, and lumbar spine from May 2023 and October 2024.
Page 7: He compared the recent scans to a 2021 brain MRI scan. The evidence supported a diagnosis of relapsing remitting multiple sclerosis present before the subject accident. The subject accident caused an exacerbation of the pre-existent multiple sclerosis.
Page 8: Medical records from other physicians supported an active multiple sclerosis diagnosis. The May 2021 brain MRI scan showed an old multiple sclerosis diagnosis, redemonstrated on the October 2024 brain MRI scan. The condition potentially transformed into a secondarily progressive form of multiple sclerosis.
Page 9: Medical providers first diagnosed the patient with multiple sclerosis in May 2021, after the December 2020 subject accident. the patient had no clinically significant multiple sclerosis symptoms before the subject accident. The traumatic injuries from the subject accident precipitated the multiple sclerosis symptoms and reactivated the condition.
Page 10: Dr. [Name] reviewed medical records from Regional Medical Center's from 2014. the patient received treatment for glucose intolerance, chronic lower back pain, and high blood pressure at that time.
Page 11: The 2014 Regional Medical Center's records supported the conclusion that the patient suffered no clinically significant multiple sclerosis symptoms before the subject accident.
Page 12: the patient experienced a significant decline in neurological status after the subject accident. Dr. [Name] diagnosed a secondarily progressive form of multiple sclerosis.
Page 13: Dr. [Name] agreed with Dr. [Name]. The secondarily progressive form is more severe, does not become quiescent, and responds poorly to medications.
Page 14: the patient had no symptom progression before December 2020. The subject accident reactivated the multiple sclerosis. the patient currently suffers from secondarily progressive multiple sclerosis.
Page 15: the patient reached a possible plateau in symptom severity. He started Ocrevus to treat the multiple sclerosis. the patient suffered a dramatic decline in neurologic function after December 2020. Multiple sclerosis and chronic pain both contributed to this neurological decline.
Page 16: the patient experienced no dramatic neurological decline over the past several months. The brain MRI scans from May 2021 and October 2024 contained lesions of varying ages.
Page 17: Old lesions, or "black holes" representative of brain tissue loss, appeared on both the 2021 and 2024 brain MRI scans. These indicated past multiple sclerosis episodes. Recent

hyperintense lesions appeared in the cervical and thoracic spine. An altered area in the cervical spine resulted from a different disease process, consistent with prior cervical spinal compression and subsequent cervical spinal fusion.
Page 18: He evaluated the spinal cord and the interaction between orthopedic injuries and the multiple sclerosis diagnosis. Pain from multiple sclerosis differs from the radiculopathy the patient presented with in December 2020. Radiculopathy involves nerve root compression and follows a strict dermatomal pattern, unlike multiple sclerosis lesions.
Page 19: He evaluated the scans for cervical spine compression from a prior disc protrusion.
Page 20: the patient developed new progressive weakness in the lower extremities after the subject accident. A causal relationship existed between the subject accident and the progressive neurological impairment from multiple sclerosis. This conclusion relied on the temporal relationship and the established pathophysiology between traumatic injuries and multiple sclerosis exacerbation.
Page 21: Medical literature supported the relationship between traumatic injury and multiple sclerosis reactivation.
Page 22: Neurologists recognize physical trauma, psychological stress, and chronic pain as significant risk factors for multiple sclerosis exacerbation.
Page 23: Traumatic injury and pain exacerbate pre-existent multiple sclerosis. The subject accident exacerbated the patient's latent multiple sclerosis, which was asymptomatic before the subject accident.
Page 24-25: Medical evidence linked traumatic brain injury to a new onset of multiple sclerosis. The most accurate explanation for the patient was trauma-induced exacerbation of pre-existent multiple sclerosis.
Page 26: Additional medical literature confirmed the relationship between physical trauma and multiple sclerosis deterioration.
Page 27: Traumatic injury damages the blood-brain barrier and activates the immune system. This explains the pathophysiology of multiple sclerosis exacerbation.
Page 28: Literature supported the distinction between multiple sclerosis symptoms and radicular symptoms.
Page 29-30: The Lhermitte's sign literature supported the distinction between multiple sclerosis and radicular pain. The 2021 and 2024 brain MRI scans showed dissemination in time, indicative of distinct multiple sclerosis flare events.
Page 31: Medical literature established a firm connection between traumatic brain injury and multiple sclerosis exacerbation.
Page 32: He reviewed recent medical articles from Dr. [Name].
Page 33: Neurologists consistently observe the relationship between trauma and multiple sclerosis exacerbation in patients.
Page 34: Medical literature contained evidence that supported the relationship between multiple sclerosis and traumatic events.
Page 35-36: Comprehensive meta-analyses demonstrated a significant correlation between general injuries and multiple sclerosis exacerbation. The Lunny 2014 study evaluated over a thousand individual studies and found a significant relationship.
Page 38: Neurologists routinely investigate recent physical trauma as a cause for multiple sclerosis flare events.

Page 39: The clinical neurology community agreed that trauma exacerbates multiple sclerosis symptoms.
Page 40: Trauma can induce the onset of autoimmune conditions, inclusive of multiple sclerosis.
Page 41: A traumatic event can trigger an autoimmune process in a previously healthy spinal cord.
Page 42: The comprehensive Lunny systematic review represented the accepted knowledge in the neurological community.
Page 43: Clinical experience demonstrated that physical trauma altered the clinical course of multiple sclerosis.
Page 44: Prior literature discussed the difference between multiple sclerosis lesion pain and radicular pain from nerve root compression.
Page 45: The physical examination portion of the report remained unchanged from the initial in-person evaluation in September 2024.
Page 46: the patient completed only one in-person clinic visit in September 2024.
Page 47: During the remote visit on 03/17/2026, the patient reported brain fog, fatigue, and chronic headaches. He reported side effects from a multiple sclerosis medication.
Page 48: Dr. [Name] offered behavioral recommendations for sleep habits and brain health.
Page 49: The clinic prescribed Modafinil. the patient reported general fatigue and low energy.
Page 50: Multiple sclerosis and traumatic brain injury both contributed to the fatigue. Modafinil treats excessive daytime sleepiness and attention deficit disorders.
Page 51: Physician Assistant [Name] conducted the remote follow-up visit. Dr. [Name] concurred with the treatment plan.
Page 52: the patient used Rizatriptan with significant success for chronic post-traumatic and cervicogenic headaches.
Page 53: The headaches originated in the occipital region and radiated to the posterior head. the patient experienced severe multiple sclerosis flare events after the subject accident. These resulted in progressive gait instability and ambulation difficulties.
Page 54-55: the patient reported new cognitive impairment and depressed mood. the patient reported low mood and depression.
Page 56: Medical records before the subject accident contained no reports of clinically significant cognitive impairments or mood issues.
Page 57: During the 01/21/2026 visit, the patient reported a cessation of Modafinil due to a rash. He reported significant relief of brain fog and low energy when on Modafinil.
Page 58: Dr. [Name] performed the initial physical examination on 09/16/2024.
Page 59-60: The clinic requested medical records to better define the multiple sclerosis diagnosis. Dr. [Name] knew of the multiple sclerosis diagnosis at the initial evaluation.
Page 61: The physical examination results in the report accurately reflected the exact clinical observations.
Page 62: The physical evaluation included an interview, cranial nerve assessment, gait and balance tests, motor and sensory evaluations, and cognitive tests.
Page 63-64: the patient presented to the clinic in a motorized wheelchair. He exhibited no acute distress. Palpation revealed significant tenderness in the paraspinal neck muscles and rhomboid muscles. The bilateral occipital notches lacked tenderness.

Page 65: The cranial nerve evaluation revealed nystagmus with lateral gaze. Nystagmus indicated abnormal midbrain and brainstem circuit function. The nystagmus resulted from both the multiple sclerosis and a traumatic brain injury.
Page 66: Dr. [Name] previously diagnosed the patient with a traumatic brain injury and post-concussive disorder.
Page 67-68: The subject accident caused the traumatic brain injury. The subject accident caused chronic post-traumatic headaches, post-concussive disorder, and post-traumatic cognitive impairment.
Page 69: Dr. [Name] concurred with Dr. [Name] on the traumatic brain injury diagnosis.
Page 70: Neurologists do not use brain scans to diagnose traumatic brain injuries.
Page 71: The traumatic brain injury resulted from a significant shockwave of mechanical force. This force traveled through the body and brain during the subject accident.
Page 72: Direct blunt force trauma to the head was not necessary for this brain injury diagnosis. Indirect force transfers from the body to the brain.
Page 73: The severe force of the subject accident caused serious spinal injuries. The brain is more fragile than joints.
Page 74: The significant pain presentation after the motor vehicle collision was consistent with concussive forces.
Page 75-76: The brain traveled through space and stopped suddenly against the skull, typical of a coup-contrecoup injury. The force transfer created a shockwave.
Page 77-78: Evidence of direct head impact was absent and unnecessary. The human body cannot withstand the severe forces of a motor vehicle collision.
Page 79-80: No other medical providers besides Dr. [Name] and Dr. [Name] focused on the traumatic brain injury diagnosis. Other neurologists focused on the multiple sclerosis diagnosis.
Page 81: Dr. [Name] possessed medical records from Community Hospital's and Regional Medical Center's.
Page 82: the patient reported chronic headaches to a pain management physician.
Page 83: A quantitative EEG test from 10/31/2024 further supported the traumatic brain injury diagnosis. The EEG revealed theta and delta frequency slowness in the frontal lobe.
Page 84-85: The EEG showed no epileptiform discharges, which ruled out post-traumatic seizure activity. Frontal lobe theta and delta frequency slowness in an awake patient is a pathological biomarker for post-traumatic brain fog and cognitive fatigue.
Page 86: The abnormal EEG possessed a pathological correlation.
Page 87: the patient had multiple subcortical white matter lesions. The abnormal EEG resulted from the theta and delta slowness.
Page 88: The frontal lobe slowness did not correlate with the multiple sclerosis lesion burden, as the multiple sclerosis lesions resided primarily in the posterior brain.
Page 89: Nonrestorative sleep could not explain the level of EEG abnormality.
Page 90: The medical history, symptom onset, physical injuries, abnormal cranial nerve test, and abnormal EEG all supported the traumatic brain injury diagnosis.
Page 91: The traumatic brain injury contributed to the cognitive and mood symptoms.
Page 92: The traumatic brain injury and multiple sclerosis had a synergistic effect on the patient's neurological decline. the patient suffered an uncomplicated traumatic brain injury.
Page 93: the patient reported severe difficulty with concentration, focus, and memory.

Page 94: the patient experienced severe difficulty with focus on complex tasks.
Page 95: the patient frequently lost his train of thought during complex conversations.
Page 96: Other neurologists documented the cognitive deficits before Dr. [Name] evaluated the patient.
Page 97: the patient previously reported memory, concentration, and focus issues to providers at Valley Medical Center. the patient reported tearfulness, irritability, and anxiety.
Page 98: The Montreal Cognitive Assessment (MoCA) contained a typographical error regarding a perfect score.
Page 99: the patient lost points on the delayed recall portion of the MoCA.
Page 100-101: Upper extremity reflexes were normal. Right lower extremity reflexes were diminished, and left lower extremity reflexes were normal. Sensation remained intact in all extremities. the patient's prior spinal surgery in February 2020 caused no progressive neurological impairments and did not exacerbate the multiple sclerosis.
Page 102: Dr. [Name] based opinions on physician reports and patient history.
Page 103: Multiple sclerosis causes central neuropathic pain, such as Lhermitte's sign. Lhermitte's sign produces an electric shock sensation down the spine and affects a widespread area of pain perception. Radicular pain stems from peripheral nerve root compression and follows a narrow dermatomal pattern.
Page 104: Multiple sclerosis pain affects the entire region distal to the lesion, rather than a single localized area. Radiculopathy involves irritation of a nerve root.
Page 105: Multiple sclerosis lesions reside in the central nervous system and do not compress peripheral nerve roots.
Page 106: A nerve conduction study performed by Dr. [Name] showed evidence of radiculopathy.
Page 107: Central neuropathic pain and radicular pain share similar qualities but affect different locations. Neuropathic pain originates from demyelinated lesions in the brain and spinal cord.
Page 108: Peripheral radiculopathy originates from peripheral nerve roots. Multiple sclerosis causes central neuropathic pain from the spinothalamic tracts or thalamus.
Page 109: Multiple sclerosis does not cause disc herniations or nerve root compression.
Page 110: Lhermitte's sign indicates an issue within the spinal cord that affects a large array of nerve endings.
Page 111: Spinal cord lesions alter pain perception but leave peripheral nerve endings intact, resulting in normal nerve conduction studies for those specific nerves.
Page 112: A spinal cord lesion affects the entire downstream circuit.
Page 113-114: Lhermitte's sign can occur in multiple sclerosis and other spinal cord injuries. Lhermitte's sign is specific to spinal cord pathology and does not occur from a cervical nerve root compression.
Page 115: Lhermitte's sign does not indicate radiculopathy.
Page 116-117: Dr. [Name]'s nerve conduction study confirmed a diagnosis of lumbar radiculopathy. Dr. [Name] agreed with this diagnosis based on the nerve conduction study results.
Page 118: Dr. [Name] did not evaluate the lumbar MRI scans for radiculopathy.