Do Bus Accidents Cause Nonepileptic Seizures?: Complex Issues of Medicolegal Causation

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The question of causation is approached through a case description and analysis. An alternative perspective is discussed for addressing neuropsychiatric cases in the medicolegal context. Viewing medical litigation from the perspective of risk factors, timeline, and baseline may add clarity to the difficulty of understanding causation.

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The question of causation has confounded medicolegal investigations for centuries. Forensic evaluators are often asked to give an opinion to the court in medical cases as to the cause of a litigant's presentation. The case described herein illustrates the difficulties in establishing causation when viewing the insult-injury from an immediate proximate-cause perspective. The authors provide an alternative perspective for addressing neuropsychiatric cases in the medicolegal context.

The following case history has been modified for this presentation. C.J. sued the school bus company, the defendant in the case. Although the parties settled shortly before trial, the litigation continued for approximately three years. In the course of the litigation, the information referenced in this article is on public record in the motions and briefs filed in the court. The plaintiff also gave authorization for the attorneys to obtain her medical records, and she signed a release acknowledging that de-identified data may be used for research, teaching, and publication purposes.

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Case Description

The Accidents

C.J. was a passenger in two minor school bus accidents that occurred roughly one year apart. The same full-sized yellow bus was involved in each accident, and on both occasions she sat on the right side of the bus in the second or third row from the front.

The first accident happened before school on a November morning. C.J. was a 15-year-old junior in high school. The bus driver was traveling less than 35 miles per hour when a full-grown buck bolted a few feet ahead and to the right of the bus. The deer was clearly headed into the street in front of the bus. The driver braked hard; books and book bags flew forward. Opposite C.J., a girl who was sitting with her feet up and her back to the window slid forward onto the floor between her seat and the seat in front of her. Almost immediately after the bus driver brought the bus to a complete stop, the car traveling behind the bus rear-ended it. The damage to the car was the more extensive. The only damage to the bus was a slightly dented rear bumper, minor enough that the bus company never repaired it.

C.J. testified that she was thrown forward then backward by the impact. While her body most likely did go forward when the bus driver braked, a biomechanical expert retained by the school bus company concluded that the change in velocity from the rear-end collision was less than five miles per hour. The expert did not believe the forces involved in the accident were sufficient to cause physical injury. Be-

cause the accident involved a straight-on hit and C.J. confirmed in her deposition that she was facing forward, no rotation was involved. Findings in a neurological work-up were unremarkable. Despite this, C.J.'s parents took her to a chiropractor who treated her in 68 sessions over the next eight months for complaints of neck and back muscle strain.

Unfortunately, one morning the following September—exactly one week after C.J.'s chiropractor released her from treatment—the school bus was involved in a second traffic accident on the way to school. It was never determined definitively who was at fault, but one of the two drivers involved in the accident ran a red light near the school. The bus was traveling 25 to 30 miles per hour as it climbed a hill, and the bus driver slowed from that speed to go through the intersection. When she was in the middle of the intersection, the bus driver saw a car to her left, on a collision course with the bus. She did not have time to honk her horn, steer out of the way, or brake before impact. The car almost avoided the accident, but it hit the left side of the bus, so far to the rear that the only impact was to the portion of the rear bumper that folds around to the side. Again, the damage to the car was the more extensive, and the bus company saw no reason to repair the slightly dented bumper.

The biomechanical expert determined that this second collision had greater force than the first one, but it was still biomechanically insignificant. The bus had a change in velocity of five to seven miles an hour, with a rotational change in direction from approximately 11 to 10 o'clock at the left front end. These changes would have caused a slight forward and leftward motion of the students in the bus. A few of the students on the bus did not even realize that there had been a collision. Others described the impact as anywhere between light and medium. One student claimed a whiplash injury, and a few reported headaches (but some admitted later that they had reported a headache to get out of taking a test). Immediately after the impact, most of the students crowded the seats looking out to the left rear of the bus and regarded the incident as exciting.

The Injuries and Medical Treatment

C.J. reported more serious injuries. She testified that she saw the accident unfolding and put her hands in front of her to brace herself. When the actual impact occurred, she was thrown forward, then backward. By the time the police came, she complained of back pain and tingling in her legs and was taken to the hospital by ambulance. Her parents took her from the emergency room to a chiropractor that same day. The next day, she also had pain in her neck. Three days later, she experienced numbness and twitching in her lower extremities. Within eight days of the accident, she had numbness on her entire right side and was demonstrating jerking of her trunk and extremities, with the movements on her right greater than those on her left. Over the course of a year, examination by various doctors resulted in a diagnosis of psychogenic nonepileptic seizures (NES) and a psychogenic movement disorder. A video electroencephalogram (EEG) confirmed the absence of epileptiform activity during her seizure-like episodes. A magnetic resonance image (MRI) of the brain was normal, and brain single-photon emission computed tomography (SPECT), which was initially read as abnormal, with right global hypoperfusion, was later read as normal after correction of the head rotation on the image slices. Initial outpatient neuropsychological testing was not completed because of the patient's movements. She scored in the very low range on verbal indices. Inpatient neuropsychological testing revealed a full scale IQ of 88, low average scores across measures, and a 1–3 scale elevation (hypochondriasis-hysteria) on the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) scale.[1]

C.J. was treated, without resolution of her symptoms, with several analgesic and antiepileptic drugs and medications for movement disorder, including levetiracetam, gabapentin, haloperidol, quetiapine, pimozide, tizanidine, and naproxen sodium. Alternative treatments included melatonin, tyrosine B, fish oil, calcium-magnesium-zinc, calcium-magnesium, ginkgo biloba, and quercetin supplements. She developed a pruritic body rash. One chiropractor described C.J. as having "left sided basal ganglionic demise with associated left neocortical compromise," and treated her with "cognitive stimulation," including motor exercises and high-dose oxygen therapy, with the goal of "increased protein replication to drive the mesencephalic areas away from threshold and decreased spontaneity of movement on the right." The chiropractor also referred C.J. to a naturopath for heavy-metal chelation therapy. She developed a metallic taste and polydipsia during treatment

with dimercaptosuccinic acid (DMSA), glutathione, magnesium, and taurine supplements.

The Neuropsychiatric Review

An independent neuropsychiatric examination and chart review revealed that C.J. was the full-term product of an uncomplicated gestation until the time of delivery, when her mother underwent emergency cesarean section under general anesthesia for vaginal bleeding. Fetal distress with bradycardia and no heart rate at birth were noted with an Apgar score of zero at both one and five minutes. C.J. was resuscitated and intubated, and she had a partial-exchange transfusion with packed red blood cells. She also had diffuse tonic clonic seizures that were treated with phenobarbital, phenytoin, and diazepam. A head computed tomographic (CT) scan on day seven was normal. She was discharged with the diagnosis of perinatal asphyxia and hypoxic ischemic encephalopathy, and she was followed up in the pediatric neurology clinic.

Over the course of 2.5 years, her EEG was normal. At 3.5 months of age, C.J's phenobarbital treatment was tapered off. She was discharged to early intervention. Her mother recalled her developmental milestones as being "a couple of months behind her brother, but not drastic." She never lost verbal or motor skills. Educationally, she required assistance with reading in grade school, and she performed below state and national norms on standardized language tests throughout her schooling. There was no family history of psychiatric diagnoses, seizures, movement disorder, or immunologic disorder.

Socially, C.J. recalled having many friends in school and getting along with teachers well. She played on three soccer teams, and she had a boyfriend in middle school. Although she denied a history of verbal, physical, emotional, or sexual abuse, she witnessed a physical altercation between her mother and brother approximately two years before the first accident.

C.J. displayed several medically unexplained symptoms after the altercation including arthralgia, myalgia, dizziness, light headedness, decreased energy, blurred vision, headaches, daytime somnolence, nocturnal leg movements, and chest pain. Extensive work-up for these symptoms was negative, and her consulting physician raised the question of anxiety or depression. The spring before the first accident, she pulled a groin muscle playing softball and that, together with the unexplained symptoms, led her to decrease her extracurricular activity on the three sports teams. In her deposition, she stated that her symptoms of dizziness, fatigue, and concentration problems in school "ended up going away that summer when I had no more homework and I stopped playing on a couple of the teams." Her explanation of her symptoms was, "I was exhausted, and it ended up catching up to me."

Neuropsychiatric Exam and Conclusions

A day-in-the-life video from a few months after the second accident showed erratic, continuous movements of her right arm and stamping movements of her right leg, with astasia-abasia on gait. By the time of her deposition, C.J. and her mother claimed that, although the worst of her symptoms abated approximately a year after the second accident, she was left with a complete inability to use her right arm and hand. A surveillance video filmed a few weeks before the deposition, however, showed her using her right hand to walk with a cane, drink from a mug, and hold a cell phone while eating a meal with her mother in a restaurant. Independent neuropsychological battery findings revealed scores consistent with a negative response bias (intentionally poor performance on testing or exaggeration of symptoms) on the Word Memory Test, the Test of Memory Malingering, and the Fake Bad Scale of the MMPI-2.

Her neuropsychiatric examination revealed non-neuroanatomic motor and sensory findings and the inability to use her right arm in daily activities, such as cutting, using a fork, or holding a cup. She had 5/5 motor strength throughout with distracting maneuvers. Mental status was notable for wearing sunglasses indoors, euthymia, and a 23/30 score on the Folstein Mini-Mental State Examination[2] in which she missed the date, one serial seven subtraction item, all three registration objects, and two of three recall objects after five minutes. When questioned, she and her mother both denied intentional production of her symptoms, and she did not meet criteria for a mood, anxiety, psychotic, or post-traumatic stress disorder.

A review of her symptomatic timeline showed that the NES and psychogenic movement disorder (PMD) occurred after the second accident; however, she had reported several medically unexplained symptoms beginning 16 months before the first accident, and the question of exaggeration of symp-

toms was raised. Her symptoms appeared to have changed from an underlying undifferentiated somatoform disorder before either of the accidents, through conversion disorder after the accident, into a possible factitious disorder with the litigation.

Discussion

Causation was a particularly thorny issue in the case of C.J., one that the attorneys could not resolve without the medical analysis supplied from a combined neuropsychiatric perspective.

The case confounded the attorneys who were defending the school bus company because it did not follow the usual rule of a linear relationship between the physical force involved in the collision and the severity of the injury: the bigger the hit, the worse the injury. Injuries that do not follow this model raise red flags. Certainly, by any reasonable standard, neither accident was particularly traumatic, physically or psychologically. Even factoring Blinder's observation that "[t]here is probably never a physical injury without some measure of psychic trauma or functional overlay" (Ref. 3, p 84) into the calculation, the NES diagnosis seemed far out of proportion to the actual events.

The plaintiff was asking for millions of dollars, which raised both the stakes and the attorneys' suspicions. In this case, how the symptoms were or were not connected to the accident(s) had to be established. The challenge was one that is found in many neuropsychiatric cases: "The determination [of] whether the defendant's misconduct has caused the plaintiff's injury in a factual sense is an element of every personal injury action. The difficulty in establishing factual causation is epistemological: the trier of fact never absolutely can determine the 'fact' of causation" (Ref. 4, p 276). In the absence of a neuroanatomic explanation, were her symptoms consciously or unconsciously produced, and given her denial that she could use her right hand in the neuropsychiatric evaluation, even after the video demonstrated her using the hand, was the plaintiff embellishing her condition or malingering?

Although NES is infinitely more complex than whiplash, both diagnoses raised the same question in the legal case. Did the minor traffic accidents cause C.J.'s condition? If so, she deserved to be compensated fully. If not, or if the accidents were responsible for only a portion of her symptoms, the bus company owed her for only the portion of her condition that the accidents caused. Given the known influence of litigation on the potentiation of a claimant's symptoms, another question that arose in this case was how much of an influence on her symptoms was the first accident in the readiness of the patient and her family to seek remuneration for the second accident? Attempts to distinguish somatoform from factitious from malingered symptoms, to discern volitional versus unconscious behavior, and to disentangle psychological versus external motivation can be extremely difficult. Weissman wrote, "Protracted litigation creates conditions that promote mnemonic and attitudinal distortions, as well as conscious and unconscious motivations for secondary gain" (Ref. 5, p 67).

Causation Standards in Law, Science, and Medicine

Collaboration on the C.J. case brought together two professions with disparate standards of causation. As Trimble wrote, "In general, the law seeks to assess the straw that breaks the camel's back; medicine and psychiatry recognize the polyphonic nature of pathogenesis" (Ref. 6, p 221).

A cause is "something that brings about an effect or result" or "an agent that brings something about."[7] The legal standard for determining causation presented a real problem to the attorneys defending the bus company because it truncated the examination to the fairly narrow question of the trigger of C.J.'s condition. *Scroggins v. Wal-Mart Stores, Inc.*,[8] a case decided by the Iowa Supreme Court, analyzed the causation standard typically applied by the American tort system. As the court discussed, causation has two aspects: factual causation and legal causation. The trial court must first determine whether the defendant's conduct did in fact cause the plaintiff's injury. Generally, courts require plaintiffs to meet a "but-for" test of causation in fact (i.e., but for the defendant's action or inaction, the plaintiff would not have been injured). In other words, the defendant's conduct must have been a cause of the injury. However, the second component of the causation element, legal causation, requires plaintiffs to prove that the action or inaction in question also proximately caused the harm. Conceivably, many factors can contribute to an injury, but the law does not necessarily extend legal responsibility to all of them. The general rule is that the defendant's conduct is a proximate cause of harm to another if it is a

substantial factor in bringing about the harm and if there is no other rule of law that relieves the defendant of liability. In determining whether the conduct meets the substantial-factor test, the proximity of the defendant's misconduct and the foreseeability of the plaintiff's harm are the two key considerations.

The legal standard of causation works well in a straightforward situation—for example, an injury claim by a passenger who broke her hip when she fell on a city bus that was involved in a traffic accident. If the collision occurred when the driver veered into oncoming traffic through a lapse of attention, the driver's conduct would be a cause in fact of the passenger's injury. But for his looking down to answer his cell phone, the passenger would not have broken her hip. The law even applies this same rule to the so-called eggshell plaintiff who sustains an injury much more dramatically than would normally be expected (e.g., a passenger with osteoporosis). The evaluation of cause in fact is equally clearcut in this example. Even if the call the bus driver answered was from his wife reporting that their teenage son had gotten a ticket for running a red light, their son's actions—although a cause—would not be a substantial factor in the passenger's injury. The links in the chain closest to the injury are given the greatest weight.

However, C.J.'s case presented a situation that is far from straightforward. Even tempering the but-for test of causation with the substantial-factor test of proximate cause failed to factor a variety of significant events into the analysis of what caused C.J.'s NES and PMD. The attorneys defending the bus company believed that the before-and-after rule of legal causation would lead to a harsh and unjust result in a complex claim of neuropsychiatric injury.

Science and medicine, on the other hand, view causation on a much larger plane. Koch's postulates served as the basis of science's understanding of disease causation based on germ theory. The postulates are summarized as: (1) the microorganism will occur in every case of the disease and can explain the pathology and clinical changes associated with the disease; (2) the microorganism must be shown to be distinct from any others that might be found with the disease; and (3) if the microorganism is isolated, and repeatedly grown in culture, it will induce a new case of disease in the susceptible animal.[9]

Medicine, similarly, allows for a broadened timeline. Sir A. B. Hill[10] proposed guidelines for assessing causation in medicine. He suggested a set of nine guidelines to help determine if associations between the environment and disease are causal. These included strength of the association, consistency, specificity, temporality, biological gradient, plausibility, coherence, experiment, and analogy. Susser[11] described three essential attributes of true causes in epidemiology: association, time order, and direction. A web-of-causation model was proposed in the 1960s as a paradigm for noninfectious diseases, such as lead poisoning, incorporating both the host and environmental determinants.[12] Rothman proposed the sufficient-component theory of causation to bridge the gap between theoretical ideas of causation and epidemiology.[13] He defined a sufficient cause as a "complete causal mechanism" that "inevitably produces disease" (Ref. 14, p 8). Fundamental to this model, a sufficient cause is not a single factor, but rather a minimal set of factors that unavoidably produce disease. These include a minimal set of factors, or component causes, whereby "blocking the action of a single component cause stops the completion of the sufficient cause and prevents the disease from occurring by that mechanism" (Ref. 15, p 392).

Application to the Medicolegal Problem

This intricate and complicated case illustrates the utility of the expanded definition of risk factors in the medicolegal context. The causation argument varies, depending on the parameter of time. In the context of a timeline, if a confined period of time is selected, attributable causation is limited. When the timeline is more lengthy, causation is seen as the result of exposure to a set of factors, or "hits," incurred during a period of life that culminate in the observed disease or disorder.[16]

The expanded perspective has to be taken with neuropsychiatric causality, because the contributing events may often be interspersed over an extended period. The association of a defendant's bat striking the plaintiff's head and causing an epidural hematoma is easily made. Risk factors, however, are dependent on exposure, time period, and environmental/personal baseline. An extremely hazardous risk factor is not a threat or a cause of disease if an individual is never exposed. Conversely, a low-level risk can cause mortality if exposure occurs over a long period in a person susceptible to a disease.

Bus accidents do not cause NES, any more than they cause cancer. Most people who drink tap water

do not get cancer. Causation is invoked, however, in the child who develops leukemia and has a family history that makes him or her genetically susceptible, and who drank for five years from a well containing probable carcinogens.[17,18] The point is not that the well caused cancer; the point is that the right person, with multiple predisposing factors, may be more prone to the adverse effects of exposure to a particular risk factor.

None of the other children or adults involved in the accidents developed NES. Soon after the second bus accident, C.J. presented new symptoms, but it would be false to conclude that the bus accident was the sole contributor to her symptoms. While we do not have information on the other children's birth, developmental, academic, familial, social, medical, and psychiatric histories, it is improbable that anyone else on the bus had exactly the same risks, and so it is fair to say that C.J. was unique in the precursors to her symptoms. The bus accident was one link in a chain of causation along a continuum of hits in an individual with multiple risks and various prior exposures that occurred during the developmental period of life.

Neuropsychiatric research increasingly illustrates the importance of the link in the chain in symptom development. In this case, the combination of infantile stress, early childhood developmental difficulties, psychosocial stressors, intrapersonal conflicts, and motivation all contributed to the plaintiff's presentation. Several etiological models for the development of NES have been described.[19] Two medicopsychological models are pertinent to this case: allostatic load and social cognitive theory.

Selye[20] showed the protective and damaging effects of stress on our bodies as we adapt to stressors. Further studies of allostasis (the ability to achieve homeostatic stability through change) have built our scientific understanding of a neurophysiological model for the accumulated burden of stress on the brain and body.[21] Given multiple developmental, environmental, and genetic factors, certain individuals are less prone to regulation of their allostatic systems, increasing their allostatic loads (that is, the wear and tear that results from chronic over- or underactivity of allostatic systems). If the inactivation is inefficient, as McEwen explains, this results in "overexposure to stress hormones. Over weeks, months, or years, exposure to increased secretion of stress hormones can result in allostatic load and its pathophysiologic consequences" (Ref. 22, p 172). In this case, expanding the time frame to incorporate C.J.'s history and the prior and current exposures helps us to understand the diagnosis of somatoform disorders as contributing to the initial production of NES, in an ostensibly atraumatic event.

In developing social cognitive theory, Bandura[23] challenged the theory of stimulus-response behaviorism when he described the interaction and change of both the individual and the environment. Bandura addresses causation by further developing the concept of agency, or the individual's responsibility and part in the production of his or her own behavior. He writes:

> Human agency is characterized by a number of core features . . . [that] include the temporal extension of agency through intentionality and forethought, self-regulation . . . , self-reflectiveness about one's capabilities, quality of functioning, and the meaning and purpose of one's life pursuits. Personal agency operates within a broad network of sociostructural influences. In these agentic transactions, people are producers as well as products of social systems [Ref. 24, p 1].

In this case, psychosocial stressors and interactions between family and plaintiff may have generated further symptoms.

Through analysis of the factors inherent in C.J.'s condition, the neuropsychiatric evaluation contributed to the case on several levels. The case presented an enormously complex medical scenario. Throughout the process, the attorneys sought tutoring from the neuropsychiatric/neuroscience perspective while attempting to parse the neurological, psychiatric, developmental, and social contributors to her presentation and condition. The attorneys needed to acquire specific expertise that they could not gain on their own.

The knowledge gained from informal consultations guided the defense's theory, which in turn guided the cross-examination of the plaintiff, her family members, and the various medical professionals who treated her. Further, following the independent neuropsychiatric evaluation and chart review, a report was issued to both parties in the case. Because each side in any litigation engages in a constant process of weighing the risks and benefits of going forward with the case, the contribution of the neuropsychiatric perspective influenced the decision to settle the case and, indirectly, the monetary amount of the settlement. Therefore, viewing C.J.'s progression of symptoms not merely in the context of proximate

causation, but rather from an "exposure(s) over time" perspective contributed directly to the parties' reaching a settlement and avoiding protracted court time.

In assisting the parties to reach a result that reflected the contribution of multiple causal factors, the neuropsychiatric evaluation contributed to a societal benefit as well. The American tort system exists to achieve a fair allocation of the costs of civil wrongs. Someone bears the costs, whether it is the injured person, who may not have caused the injury, or, in cases in which the injured person's resources are expended, society as a whole. Ideally, however, the tort system requires that the person who causes the harm pays for it. Therefore, completely in line with the principles of the tort system, the addition of the neuropsychiatric perspective to the case also helped achieve a result that compensated C.J. in an amount that balanced the multiple factors in her condition, some of which were related to the two bus accidents, many of which were not.

As a model of clinical neuroscience informing the law, this case is representative of the landmark project developing on the national front.[24] The recently formed Law and Neuroscience Project is one of the first systematic efforts in the United States in which the practices of neuroscience and law are gathering experts to develop guidelines, primers, and research on ways that neuroscience can be integrated into the legal system.[25] Topics examined by the project's workgroups include addiction, brain abnormalities, and normal decision-making, as they relate to concepts in the law, such as criminal responsibility. Neuroscience is already being used in the legal system, and this effort has the potential to aid the justice system in making accurate judgments and more effective interventions with less bias.

In summary, for medicolegal analysis, incorporating a risk-exposure timeline in a sufficient-component cause context, informed by the biophysiological constructs of allostasis and the psychosocial theory of social learning, may help to elucidate complex, multifactorial scenarios beyond the legal perspective of immediate proximate cause. To be just is to be impartial and discerning in making objective, reasoned decisions, in the view of plaintiffs and defendants. Viewing medical litigation from the perspective of risk factors, timeline, and baseline added much needed clarity in the C.J. case, and it may add clarity to the understanding of difficult questions of causation in future cases as well.

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