A Systematic Approach to Clinical Determinations of Causation in Symptomatic Spinal Disk Injury Following Motor Vehicle Crash Trauma

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Clinical determinations of causation in cases of intervertebral disk (IVD) injury after a motor vehicle crash (MVC) are often disputed in medicolegal settings. No published systematic guidelines exist for making such determinations, which has resulted in infringement by nonclinical personnel into injury causation evaluations, a traditionally clinical activity. The result is causal determinations that are potentially disconnected from clinical observations of injury. The purpose of this review was to evaluate the current literature on causation, causal determinations after trauma and IVD injury after MVC, and to develop a practicable, logical, and literature-based approach to causation determinations of symptomatic IVD injury after MVC. The results of the review indicate IVD injury can result from any MVC regardless of magnitude, thus meeting the first criteria of causation, biologic plausibility. Individual determinations of causation depend entirely on the temporal association between the collision and the symptom onset (the second criterion) and a lack of a more probable explanation for the symptoms (the third). When these causal elements are met, clinicians can assert causation on a "more probable than not" or "reasonable probability" basis. Because of a lack of an established or reliable relationship between collision force and the probability of IVD injury the investigation of collision parameters is not a useful adjunct to causal determinations.

INTRODUCTION

The origin of injury to the intervertebral disks (IVD) of the spine is a common source of dispute in medicolegal circumstances. Whereas treating clinicians typically make determinations of causal relationships between a trauma and an injury primarily based on the patient-related history of the traumatic event and the onset of symptoms, opposing experts frequently rely on population-based assumptions as a basis for disputing causal relationships. This scenario is observed most often when disk injuries and symptoms are attributed to motor vehicle crashes (MVC), because many crash-related injuries result from the negligence of another driver; thus, the nature and extent of an occupant's injuries may be contested. There is typically a strong third-party (insurer) interest in minimizing compensatory payments on behalf of the individuals who are at fault for the crash. As a result, an adversarial system has developed in the United States and many other industrialized nations, with the injured patient and his or her treating physicians on one side and the party responsible for the crash, his or her insurer, and the insurer's medical and scientific consultants on the other side [1,2]. Although some clinicians prefer to avoid causal determinations altogether, it can be argued that such determinations are part and parcel of regular medical care. To avoid such a determination solely because it may be later disputed by a party with interests opposing those of the patient is to avoid a duty to the patient; a duty that can have a significant impact on the patient's well-being. This is not to say that dissemblance and fraud do not exist in the patient population; however, when there is no *evidence* of dishonesty, there is no reason to assume the presence of fraud simply because a patient is claiming injury after a MVC that was caused by another's negligence (the most common setting for a legal claim for monetary damages). Such events are, for the most part, randomly distributed in the population that uses motor vehicles and not selective for people

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Disclosure: 5B, expert witness

C.J.C. Spinal Injury Foundation, Westminster, CO
Disclosure: 5B, expert witness

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Disclosure: nothing to disclose

Disclosure Key can be found on the Table of Contents and at www.pmrjournal.org

Submitted for publication April 21, 2009; accepted July 22.

in whom fraudulent behavior is more common. When allegations of fraud are made in a medicolegal context, despite the absence of evidence of fraud, it is reasonable to question the motivation behind the assertion, because forensic consultants who provide partisan opinions are no more immune to secondary gain pressures than are litigants claiming injury [3].

There are some authors who maintain that for a determination of cause and effect to be made between a diagnosed injury and an MVC, a biomechanical analysis of the forces is necessary to determine whether or not a particular collision had the potential to cause the injuries [4] or that a detailed crash reconstruction is necessary before a causal association can be determined [5]. In contrast, most authors who have documented IVD injuries associated with an MVC make no mention of a biomechanical force analysis or a crash reconstruction [6,7]. The latter approach is most typical of the clinically pragmatic approach to causation. If it is known that an injury *can* be caused by a trauma, then there is no practical or logical purpose served in quantifying the degree of the trauma in order to assess the risk of injury *after* the event. Even injuries that are deemed highly unlikely or improbable are by definition still *slightly likely* or *possible*, and thus injury presence is typically evaluated after the fact clinically, and not called into question by a *post-hoc* suggestion of infrequency.

For the purposes of the present inquiry, a Medline search for the years 1980 through 2009 (as of July 2009) using the medical subject headings (MeSH) terms *intervertebral disk*, *intervertebral disk displacement*, and *disk*, *herniated* as well as the non-MeSH terms *cervical disk*, in conjunction with "*injury*" and/or "*trauma*," along with "*cause*," "*causal*," and "*causation*" in various combinations did not elicit any publications that addressed an organized approach to determinations of causation between disk injury and MVC exposure. It appears that, at the present time, there is no widely accepted scientific or clinical standard for such determinations.

The purpose of this article is to assess the evidence for the causal relationship between disk injury and MVC trauma based on the published literature on causal standards and the science of both disk injury and MVC trauma. Further, the authors propose a systematic and scientifically based approach to individual determinations of causation.

PRIOR PUBLICATIONS ON CAUSATION

Determinations of causal association between a noxious exposure (microorganism, chemical, trauma, or other) and a disease or injury outcome in populations is an area of considerable interest to epidemiologists and others who study such issues. Individual clinical observations of cause and effect can serve as a reasonable basis for a case study and suggestion of a relationship. It is, however, difficult to draw generalizable conclusions from such reports. Case series provide stronger evidence of causation; however, without a control group of unexposed subjects, there is no way to determine which effects can be isolated to a particular exposure. Even when case-control studies, in which diseased or injured subjects are compared with healthy subjects for level of exposure to a suspected noxious agent, show positive correlations between exposure and disease or injury outcome, the results cannot be interpreted as validated evidence of a causal relationship, as confounding factors may exist that can obscure true relationships. An example of confounding would be the relationship between coffee drinking and lung cancer. Coffee drinkers may have a higher rate of lung cancer than non-coffee drinkers, but only because smokers are more likely to drink coffee than nonsmokers, not because coffee is causally linked to lung cancer.

In an attempt to more clearly identify causal relationships between noxious exposures and outcomes in populations, Hill outlined a set of 9 criteria that needed to be considered to reasonably conclude that a particular noxious exposure could result in a particular disease [8]. Hill's criteria have served as a kind of seminal gold standard, in that they have been adopted and modified by many others, including the Food and Drug Administration, academic investigators, and pharmaceutical companies, as a means of approaching issues of causation systematically [9]. Based in part on Hill's work, Miller et al proposed a 4-stage process for identifying environmentally caused rheumatic disorders, an application that is more applicable to the goals of this article, because it allowed for assessments of causation in individuals rather than as a population-based construct [10]. McLean subsequently adapted Miller et al's work for causation in instances of fibromyalgia after MVC [11]. All causal criteria can be distilled to a minimum of 3 common and essential elements, which are as follows [12,13].

1. There must be a biologically plausible or possible link between the exposure and the outcome. For example, trauma and fracture are plausibly linked, but trauma and leukemia are not. Plausibility is a low threshold that is exceeded with relatively weak evidence, such as from small observational studies (case studies or case series with small numbers of subjects) or from the results of well-designed experiments with many subjects. Biologic plausibility only pertains to whether an outcome can possibly result from an exposure, and is unrelated to the rate or frequency of the outcome. Thus, evidence of low incidence is not evidence of biologic *implausibility*.
2. There must be a temporal relationship between the exposure and the outcome. The outcome cannot preexist the exposure; however, the outcome of interest may have preexisted the exposure in a less severe form that was worsened by the exposure. Such a determination requires an accurate documentation of the signs and symptoms of the condition of interest both before and after the exposure of interest. Additionally, the outcome cannot postdate the exposure by a period that is considered, from a clinical perspective, to be too long or too short to relate the two. This determination is highly dependent on the specifics of any case. For example, an injury to a nerve root may cause immediate pain, but electromyogram changes may lag behind by weeks or months. The deter-

mination of reasonable temporal association is typically made as a matter of clinical judgment, rather than from clearly delineated guidelines or principles.

3. There must not be a more likely or probable alternative explanation for the symptoms. The term "likely" is of greatest importance, as, for example, it is not sufficient to simply point out that a patient with back pain after trauma is obese, that obesity is related to back pain, and thus it is more likely that the obesity rather than the trauma caused the back pain. For an alternative etiologic explanation to be considered *more likely* than an alleged exposure it must be both biologically plausible *and* have a stronger temporal relationship to the onset of symptoms than the alleged exposure. If plausibility is present and temporality is relatively comparable, then 2 exposures can be compared by examining the dose-response (intensity) of each exposure. Such a comparison may involve an analysis of the forces of the exposure as well as an epidemiologic assessment of injury frequency associated with the forces, and is beyond the scope of a clinical assessment of causation.

A Causation Algorithm for Disk Injury after MVC

In this section, the authors present a practical guideline for clinical determinations of causation in symptomatic disk injuries after an MVC, based on the application of the aforementioned 3 causal elements to the facts and findings in an individual case. The following definitions and assumptions are made. An IVD injury is defined by all of the following characteristics [14,15] (for this definition, "injury" is used only to define a disk that has become symptomatic after a discrete loading event of any magnitude, and the cause of the injury is not addressed).

1. It is symptomatic. This may include classic symptoms of segmentally appropriate radiculopathy, regional axial pain, or diskogenic referral of pain in a nondermatomal distribution.
2. There is clinical imaging evidence (magnetic resonance imaging [MRI], computed tomography, or diskography) that the symptomatic disk fits the generally accepted definition of a degenerative/traumatic disk [14]. This includes anular tears, herniations, and degeneration. Although there are findings specific to disk trauma, such as vertebral endplate fracture and edema presence in the marrow adjacent to the endplate, many traumatically initiated disk injuries are indistinguishable from degenerative changes in the disk [16]. Because of mediocre sensitivity and specificity (ie, true- and false-positive rates), imaging findings alone are insufficient evidence for either a causal or noncausal determination in a case of suspected or disputed traumatic disk injury [17,18].

MVC trauma is defined as follows.

1. The patient was an occupant of a motor vehicle at the time of a collision or event.
2. The collision or event transmitted a force through the patient's spine over a very short time, typically 70-120 ms [19]. Note that the magnitude of the force is not addressed in the definition, because any degree of forceful loading could potentially produce a symptomatic disk injury in a sufficiently susceptible individual.

There are 2 most likely scenarios in which a forcefully loaded IVD may become symptomatic.

1. A healthy disk with little or no changes associated with degeneration is subjected to a significant load resulting in disruption of the annulus and possibly instantaneous migration of nuclear material posteriorly toward the disk periphery and into the epidural space or the lateral recesses. This is the scenario most likely to produce injury to tissue surrounding the disk, including vertebral endplate fracture and ligamentous disruption, and is most likely to be associated with higher energy crashes with significant vehicle damage [20].
2. A degenerated disk (defined as a disk in which one or more of the following are present to some degree: desiccation, fibrosis, narrowing of the disk space, diffuse bulging of the anulus beyond the disk space, anular tears, endplate sclerosis, and osteophytes at the vertebral apophyses [14]) is loaded in a manner that initiates a symptomatic response. A significant proportion of the asymptomatic population has some degree of disk degeneration as seen on MRI; Matsumoto et al have performed the largest study to date on the topic, with MRI scans of the cervical spines of 497 asymptomatic subjects [21]. These authors describe a progressive direct relationship between the presence and severity of degenerative changes and age, with the disk degeneration noted in a range of 17% men and 12% of women in their 20s to 86% and 89% of men and women older than 60 years. Other authors have described similar findings with broad ranges of values depending on the age of the subjects; from virtually no disk degeneration in subjects <30 years of age, to 14% in those 30-40, and 62% in subjects >40 years of age [22,23]. Similar observations have been published with regard to lumbar spine degeneration among asymptomatic patients. Jensen et al described a cohort of 98 subjects who underwent lumbar MRI, noting that only 36% of subjects had no abnormal disks. Fifty-two percent of the scans were interpreted as having a bulge at a minimum of one level, 38% had abnormality at more than one level, 27% had a protrusion, and 1% had an extrusion [24]. These findings are consistent with those reported by other authors [25].

Causal Elements

Biologic Plausibility. Can an MVC of any severity cause a disk injury? Crash testing of intact cadavers at accelerations recorded for no-damage rear-impact collisions has demonstrated IVD injury that cannot be detected by conventional

imaging such as CT and MRI [26]. Although the biomechanical literature on experimental loading of disks in cadaveric sled testing has demonstrated disk injuries even in relatively low accelerations (3.3 and 4.5 times the force of gravity or "g" [26]), such ex vivo testing of healthy disks does little to demonstrate minimal thresholds for injury in live human populations exposed to real-world crashes. No cadaveric testing can duplicate the significant variation in the manner in which loads are transmitted to the human spine in a MVC, given the variation in occupant position, restraint systems, vehicle interiors, and muscle loads. Additionally, the potential for variation in the condition of an individual IVD, including the ability of the disk to resist forceful loading from an external source, is too large to ever define in an experimental setting.

A relevant literature search revealed a complete absence of any publications suggesting that symptomatic IVD derangement *cannot* result from MVC exposure of any magnitude. To the contrary, injury to the disks of the cervical and lumbar spine has been documented in the relatively low level accelerations associated with little to no-damage collisions, roller coaster rides, and even sneezing [27-30]. It is reasonable to conclude, as a general precept, that the forceful loading of the spine that can occur in any MVC is a biologically plausible (possible) cause of symptomatic disk injury.

Temporal Association. Temporality is the strongest evidence of causation in evaluating the patient with post-MVC disk injury. It must be first established that the MVC preceded the onset of symptoms attributed to the disk injury. The exception to this rule is when a previously symptomatic disk is exacerbated by an MVC to the point that the course of care is significantly altered (ie, a previously diagnosed nonsurgical disk condition becomes surgical directly after an MVC). In such cases, the determination must be made clinically as to whether the disk symptoms were likely to have worsened to the point that the surgical intervention was inevitable absent the MVC. The mere fact that the disk was previously symptomatic is not sufficient to draw the conclusion that it would have required surgery, given the good outcomes for patients with diskogenic symptoms who use a variety of surgical and nonsurgical treatments [31-33]. The symptom onset must be in reasonable proximity to the time of the MVC; however, disk injury symptoms may initially present as identical to symptoms of spinal strain or sprain, and it may be weeks or months before an MRI is performed and the disk injury diagnosis is first seen [34]. Additionally, disk injury symptoms may be progressive, as an injury to the annulus may progressively allow nuclear migration toward neurologic structures over a period of time while the patient is in weight bearing and active. Determinations as to whether delayed symptoms fulfill the temporal association criteria must be made by clinicians on a case-by-case basis.

An obvious weakness of the temporality criterion is that it can only be established from the history given by the patient, and patient histories can be erroneous or falsified. The former is unlikely in the case of injury, however, because one of the hallmarks of injury is that there is a close temporal relationship between cause and effect (this lack of clear temporal proximity in repetitive traumatic exposures is the reason that injuries such as carpal tunnel syndrome are classified as diseases) [35]. This close temporal relationship means that it is unlikely that a patient will misattribute symptoms to a traumatic cause. As a practical matter, the most reliable indicator of when a patient began to have symptoms after a trauma is the patient. This is not to say that patients do not get details concerning an injury event wrong; patient perception or recollection of vehicle speeds, crash sequence, and other details of a collision may be inconsistent with the facts of a case for reasons other than untruthfulness. For example, an occupant who is injured in a minimal damage rear end collision may believe that the impact must have been at high speed despite a lack of physical evidence that this was the case.

With regard to the truthfulness of the patient, it is not the role of the clinician to investigate the veracity of the history given by the patient. Absent some ancillary indication of deception, clinicians are generally safe to believe their patients. The rate of fraud of any kind in auto insurance claims for treatment benefits (personal injury protection coverage) was estimated to be 6% for 2007 [36]. Even if every instance of fraud in auto insurance cases consisted of a patient giving a false history, clinicians would still be justified in believing their patients 94% of the time.

Intuitively, it makes sense that the closer the onset of symptoms is to the time of the collision the stronger the causal relationship between the MVC and the disk injury, and the strength of this relationship can be quantified using an indirect approach. For example, in a case of a 40-year-old male with neck pain and cervical radiculopathy that arose within 12 hours of a rear impact collision (later attributed to MRI findings of disk herniation), assuming no intervening trauma, it can be postulated, as a tautology, that either the crash caused the disk injury or that it was coincidental to the collision, regardless of cause. Thus, the probability that the crash caused the disk injuries [$P(disk_{MVC})$] plus the probability the disk symptoms are coincidental to the crash [$P(disk_{COINC})$] when added together account for all of the possible causes (100% or 1) of the disk injury and associated symptoms. If [$P(disk_{MVC}) + P(disk_{COINC}) = 1$] then rearranging the terms gives [$1 - P(disk_{COINC}) = P(disk_{MVC})$], meaning that if the probability that a disk injury occurred coincidentally on the same day of the MVC can be determined then the probability the MVC caused the disk injury can also be determined indirectly.

The probability of 2 unrelated events occurring in close temporal proximity can be calculated by multiplying the probability or odds of one times the other (odds are the ratio of 2 probabilities). For example, the odds of rolling 2 sixes in a row with a fair die are [$(1:6) \times (1:6) = 1:36$]. This is because there are 36 possible combinations for any 2 rolls, including 1 and 1, 1 and 2, 1 and 3, etc, and only one of the combinations is a 6 and 6.

In a similar fashion the odds of a disk injury occurring coincidentally on the day of a collision can be calculated for the example given earlier. The annualized odds of involvement in a MVC for a 40-year-old male are 1:14 (once every 14 years), and the daily odds of crash involvement are approximately 1:5100 (one crash every 5100 days) [37]. The odds of the spontaneous development of disk symptoms are a bit more difficult to estimate. For example, it can be said that the patient had a single-day episode of neck and arm pain 12 months before the MCV, with no intervening episodes until the time of the collision. Thus, the odds that the symptoms would recur on any single day were no greater than 1:365. Multiplied together these 2 odds yield an odds of a disk injury occurring coincidentally on the day of the collision of 1:1,861,500. Conversely, the odds in favor of a causal relationship between the collision and the disk injury, given the onset of symptoms on the day of the collision, are 1,861,500:1.

Lack of Likely Alternative Explanations

Alternative explanations for a disk injury include an intervening trauma that followed the MVC and preceded the symptoms indicative of disk injury. Another alternative explanation is the insidious onset of symptoms, possibly associated with a trivial perturbation such as sneezing. Although minor forces are possible causes of disk injury, they cannot be preferentially selected over the substantially greater forces of a collision when the two have occurred in close temporal proximity. If, for example, a patient sustains what is diagnosed as a lumbar strain in a collision and a week later sneezes and has a sudden onset of radicular pain associated with a disk extrusion, then it is most likely that the disk was injured in the MVC and the sneeze only prompted a progression of symptoms. It is not reasonable to point to minor forces associated with daily activities as a likely cause of a symptomatic disk in preference to the significantly greater trauma associated even with minimal and no-damage MVCs that is temporally relevant to the symptoms, particularly when the patient performed such minor activities with no difficulty prior to the MVC. A "likely" alternative explanation is one that is most probably causal and the best explanation to fit all of the facts at hand, not one that is only a *possible* cause with no historical or temporal evidence to link it to the symptomatic disk injury.

DISCUSSION

Individual determinations of causation for IVD injury after MVC by clinicians require only that temporal association and lack of likely alternative explanation be satisfactorily present. The biologic plausibility or possibility that symptomatic disk derangement can follow any degree of forceful loading of the spine is satisfactorily present and need not be revisited in individual cases. Thus, the concept of measuring crash forces in detail as a means of determining disk injury potential can be abandoned as serving no useful purpose in causation determinations.

There is no reliable index of the degree of force required to cause a particular degree of disk derangement. Walz and Muser theorized that unless a crash reconstruction and biomechanical loading assessment was performed for a particular crash and occupant that causation could not be determined, or, put another way, it must be determined that a given crash was sufficiently forceful to cause diagnosed injuries [4]. These authors suggested that parameters such as seat belt use, head restraint and seat properties, age, body size, and preexisting damage (sic) to the spine, *inter alia*, be taken into account when making causation determinations, effectively taking causal determinations out of the hands of clinicians. What these authors do not explain is how an evaluation of any or all of such factors could help determine whether or not any type or severity of injury is possible after a crash. Even if it could be determined that the risk of disk injury was exceedingly low for a particular occupant in a particular crash, this would not be evidence that contradicts any of the 3 elements of causation. Thus, this biomechanical risk model of injury presence has no utility for real-world determinations of causation.

After a determination of causation has been reached, the legal standard for expressing the opinion is as "more probable or likely than not" or as a "reasonable probability" or "reasonable medical probability" [38]. In some jurisdictions, the standard is that the clinician must be "more than 50% certain" that the opinion is correct. The purpose of such language is to describe the results of an internal process of weighing evidence, and to arrive at a conclusion that the clinician is more certain than not that his or her opinion is accurate or true. The methodology presented in this article is designed to provide a framework for clinicians to arrive at opinions of causation that can be expressed in terms of what is more likely than not. Thus, when using these guidelines in a narrative report setting, the clinician can write that the essential causal elements of biologic plausibility, temporality, and lack of likely alternative explanation have been met for a given case (if true), and that a particular symptomatic disk injury resulted from a particular motor vehicle crash, as a reasonable medical probability.

It must be noted that the model of causation presented herein suffers from the fact that there is no other alternative against which it can be evaluated for accuracy. Clinicians receive little or no formal education in medical school or residency regarding a systematic approach to causation. Thus, the authors recommend that guidelines presented herein be evaluated, modified if necessary, and considered for adoption by consensus by appropriate scientific and medical organizations.

CONCLUSIONS

Individual determinations of causation for disk injury after MVC are most appropriately conducted by clinicians, based primarily on an evaluation of the temporal association be-

tween the MVC and the symptom onset. The lack of a valid or meaningful collision force threshold below which it can be said that a disk injury will not occur means that investigation of collision parameters for the purposes of injury causation assessment is a pointless endeavor.

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