CORRESPONDENCE

(Letters to the Editor are welcomed. They may report new clinical or laboratory observations and new developments in medical care or may contain comments on recent contents of the *Journal.* They will be published, if found suitable, as space permits. Like other material submitted for publication, letters must be typewritten, double-spaced, and submitted in duplicate. They must not exceed **two** typewritten pages in length. No more than five references and one figure or table may be used. See "Information for Authors" for format of references, tables, and figures. Editing, possible abridgment, and acceptance remain the prerogative of the Editors.)

Biomechanics of Minor Automobile Accidents

To the Editor: Dr. Laborde's paper[1] on the biomechanics of minor automobile accidents presented an interesting theory on the genesis of chronic pain after whiplash, based on a highly selective review of the literature. Dr. Laborde chose to cite only the literature or sections of papers that he believed supported his position that whiplash injuries are nonpathologic and to ignore the vast majority of the existing literature that contradicted his position. This allowed him to draw his final conclusions, which were based on the tenet that chronic symptoms after whiplash injury are not a result of real injury or pathology. A careful evaluation of the use of the literature in this paper reveals some significant problems with the interpretation of that literature.

Dr. Laborde stated "Disk bulges, protrusions, and herniations are found on lumbar MRI in 80% of asymptomatic patients," referring to the results of a study of a series of 98 subjects (not the entire population, as implied by the statement). A careful review of the cited paper,[2] however, reveals a misinterpretation of the authors' conclusions; 36% of the authors' 98 subjects had normal disks, and thus it is not clear from whence Dr. Laborde derives his 80% figure. The authors of the paper reported that only 52% of their subjects had a bulge in at least one level; 27% were found to have a protrusion at one level (some of the subjects with protrusion also had bulges); and only 1% had an extrusion.

The relevance of the citation is not clear, since the subject of the paper is the *symptomatic* population with whiplash injury. It is reasonable to state that some asymptomatic people have disk disease, but this does not any inference regarding whether disk disease causes pain in symptomatic subjects. Pettersson et al[3] reported on a cohort of 39 subjects who had prospective follow-up for 2 years after a whiplash injury. Thirteen of the subjects were found to have disk herniation on MRI at the end of the study period, and only one was asymptomatic. Such a study is a more relevant source of information on whiplash injury and symptomatic disk herniation.

Dr. Laborde further stated "Disk herniations are uncommon in front and side impact automobile accidents severe enough to cause other serious spinal injuries." He referred to four studies of injuries resulting from moderate- and high-speed motor vehicle crashes. While this is an accurate representation of the findings of the authors of these papers, it is irrelevant to the subject of Dr. Laborde's paper, since low-speed motor vehicle crashes, particularly rear-end collisions, produce a different mechanism of injury than do high-speed crashes. The widely differing injury mechanisms for low-speed crashes versus high-speed crashes make comparisons uninterpretable. It is important to note that none of the subjects in the four papers cited by Dr. Laborde were considered to have whiplash injuries.

Dr. Laborde stated "In laboratory spinal testing, pure compression, torsion, and flexion do not result in disk herniation. Only a combination of lateral bending, hyperflexion, and severe compression can sometimes cause disk herniation." He then contradicted this statement by stating "Herniation of a degenerated disk frequently occurs without injury." This is a *non sequiter,* in that it cannot be true that only extreme trauma comprising all of the elements of lateral bending, hyperflexion, and severe compression can cause a disk herniation, and that such injuries can also occur without injury. Dr. Laborde is mixing experimental cadaveric study with the experience of clinical practice, which clearly illustrates that disk herniation can occur from a variety of causes.

Dr. Laborde stated "Otherwise, disk abnormality should be attributed to an automobile accident only if it is of a type not normally seen in the aging process, or if there is neural compression on MRI and appropriate localized new symptoms or objective findings." This statement further contradicts the earlier statement regarding the prerequisites for disk herniation. In clinical practice, attributing a disk herniation to a motor vehicle crash is done by history and chronology of symptom onset, as well as MRI and other objective findings. No other validated assumptions allow for a determination of causation. There is no established threshold for disk injury in the general population; depending on the state of degradation of a disk in a particular individual, symptomatic herniation may occur after any degree or trauma ranging from a sneeze to a life-threatening mechanical load.

Dr. Laborde cited several human volunteer crash test studies, including studies by West et al,[4] Szabo et al,[5] and Castro et al[6] as illustrations that there is a level of velocity change (or delta V of the struck vehicle) below which injury is unlikely. In drawing inferences from these studies regarding the real-world population of whiplash injuries, Dr. Laborde is misapplying human volunteer crash test studies as epidemiologic studies.

As we mentioned in our 1999 article in *Spine*,[7] referenced by Dr. Laborde in his paper, human volunteer crash tests are conducted in a manner that is least likely to result in injury in prepared, healthy, and mostly male subjects who have participated in such tests. Dr. Laborde further states "The studies of Szabo et al and Castro et al indicate that thresholds of injury hold in the presence of arthritis. Patients with soft tissue injury and arthritis had rapid healing of soft tissue injury, as did those without evidence of arthritis. Short-term symptoms did not result in chronic pain in any of the studies." It is important to note that Dr. Laborde has lost sight of the fact that these studies were not of real-world injuries, but rather crash tests using healthy subjects. They illustrate the fact that it is *possible* to undergo human volunteer crash testing with-

out sustaining significant injury, not that it is *impossible* to be injured in crashes with similar force levels and different subjects. Delta V explains only a small proportion of crash injuries; much of the variance observed in real-world crashes is explained by factors relating to individual susceptibility to injury, such as sex, age, history of previous trauma, preexisting degenerative joint and disk disease, and other factors.

Dr. Laborde has lost sight of the primary tenets of scientific method; observations are made, hypotheses are developed to explain the observations, and experimental studies are devised to relate the hypotheses to the observations. He is citing small experimental studies to invalidate the real-world observations that low-speed motor vehicle crashes can and do cause injury. This is an inversion of scientific method and leads to faulty and unsupported conclusions.

Dr. Laborde stated "The threshold for spinal injury of an occupant is usually higher than the threshold for vehicle damage. Two thousand volunteer collisions have never produced chronic neck symptoms. Collisions with no vehicle damage would usually not cause neck pain and would be even less likely to cause chronic symptoms." The cited basis for this statement is an opinion piece published in the *Journal of Rheumatology* in 1997.[8] In this editorial, Ferrari and Russell[8] provide no citation to support their claim of 2,000 crash test studies. My colleagues and I have searched the literature of human volunteer crash tests and found no more than 140 human volunteers who have participated in vehicle-to-vehicle crash tests. (Although some of the volunteers were involved in multiple crash tests, these tests are not independent of each other and cannot be considered separately.)

Regardless of the number of human volunteer crash tests that have been conducted without injury, it is a fact that some people are injured in low-speed motor vehicle crashes with minimal or no property damage. The largest study ever conducted of insurance claims for injury after motor vehicle crashes showed that 18% and 23% of women involved in a motor vehicle crash with $500 or less in vehicle damage were injured.[9] Other authors have reported cervical disk injuries with radiculopathy in ≤7 mph motor vehicle crashes (minimal to no damage) in a cohort of 237 real-world crash-injured subjects.[10]

While it is understandable that Dr. Laborde is interested in furthering his theories that psychosocial factors are at work in whiplash injury, these theories should not be promulgated at the expense of an even-handed evaluation of the literature regarding whiplash injuries. Science is best served when all facets of a problem are exposed, not just those that best suit one's present needs. Dr. Laborde's conclusion that chronic spinal pain after a no-damage rear-impact collision must be psychogenic is not supported by any valid research or hypothesis and is in fact contradicted by much of the existing literature on the subject.

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Reply

Dr. Freeman's first paragraph seems to criticize the length of my article's bibliography and interpretation of the literature. After this article was published, I found a book by Robert Ferrari entitled *The Whiplash Encyclopedia. The Facts and Myths of Whiplash*.[1] Ferrari attempted a complete review of "all of the English language scientific literature about whiplash." Since he arrived basically at the same conclusions I did, a more extensive literature review would not change my conclusions.

In his book, Ferrari[1] noted the following:

(1) Researchers have failed to identify the physical source of chronic pain after whiplash (pp 9, 20, 23, 34, 470).

(2) Psychosocial factors generate the clinical picture of chronic pain (pp 87-132, 472, 473, 485-497).

(3) Chronic pain of whiplash does not exist in every country with cars and collisions (pp xix, 82, 94) and is culturally constructed behavior resulting in hypervigilance and symptom amplification of life's daily aches and pains (pp 5, 38, 52, 66, 67, 83, 87-89, 99-103, 147).

(4) Disk degeneration is a natural consequence of aging and does not indicate injury (pp 29, 30).

(5) Human volunteer collision experiments that cause acute pain do not result in chronic pain (pp 22, 40, 53).

(6) Change in velocity (delta V) is proportional to severity of collision and probability of injury (p 42). The thresh-

old for minor symptoms (lasting hours to 1 day) is a delta V of at least 5 mph for rear-end and 10 mph for front and lateral collisions (pp 40, 51, 55, 469). Sex, head position, headrest, and seat belt were irrelevant (pp 57, 475-483). Acute injury is unlikely if damage to struck vehicle is under $500 (p 151).[1]

Dr. Ferrari and I are both medical doctors, and I also have training in biomedical engineering. Dr. Freeman's training appears to be in public health, chiropracty, and epidemiology. Different persons in different specialties reading different literature and having different experiences may be part of the explanation for our apparently different conclusions. *The Whiplash Encyclopedia*[1] supports both the extent of my literature review and my conclusions well.

In his second paragraph, Dr. Freeman is correct in his statement that the article by Jensen et al[2] states that 36% of the authors' 98 subjects had normal disks and they found disk bulges, protrusions, and herniations in 64%, not 80% as my article stated. This, however, is the only part of Dr. Freeman's letter with which I can agree. Jensen et al[2] concluded that bulges and protrusions on MRI in symptomatic patients may frequently be coincidental. Pettersson et al[3] agree and state "Correlating initial symptoms and signs with Magnetic Resonance Imaging is difficult because of a relatively high proportion of false-positive results." Boden's review[4] of the article by Pettersson et al[3] states "The results of this study are consistent with the notion that the greater determinants of chronic symptoms are related to psychological factors."

The results of mechanical laboratory testing of cadaver spines imply that front and side collisions are more likely than posterior collisions to cause disk herniations. Herniations, however, are uncommon, even in severe front and side collisions. Dr. Freeman states (fourth paragraph), "The widely differing injury mechanisms for low-speed crashes versus high-speed crashes make comparisons uninterpretable." I disagree, since all of the forces present in a low-speed crash would initially be present in a high-speed crash but at a higher level. Therefore, the likelihood of injury should be higher in a high-speed crash.

Dr. Freeman's next criticism (fifth paragraph) about mixing experimental and clinical information, I believe, misses the point yet again. The point is that degeneration precedes herniation and that disk herniation is frequently not traumatic in etiology.

Causation of neck problems is not necessarily important to the treating physician since causation does not determine treatment. It is well known that determination of causation by history alone can be inaccurate. Objective information is a much more reliable source regarding causation.

Dr. Freeman also questions (paragraph 7) the use of volunteer crash test studies. One can always say "not enough testing has been done." However, based on available information, some conclusions can be made.

Dr. Freeman states (paragraph 8) "delta V explains only a small portion of crash injuries." There is no scientific basis for this statement, and I disagree. Delta V is proportional to the energy of the crash and is a major determinant of whether someone is injured. The crash testing was done with variation in sex, age, and presence of degenerative arthritis. The data clearly indicate that low delta V indicates low probability of injury, whereas higher delta V causes acute pain to be more likely.

Dr. Freeman also says (paragraph 9) that I have cited small experimental studies to invalidate the real-world observations that low-speed motor vehicle crashes can and do cause injury. My review of the literature, including *The Whiplash Encyclopedia*,[1] reveals no scientific documentation that low-speed motor vehicle collisions cause objective evidence of physical injury.

Dr. Freeman (paragraph 10) curiously asks me to explain a statement by Ferrari and Russell.[5] They appear to be referring to 2,000 crashes that were studied and not 2,000 different volunteers, but the authors of that statement are in the best position to clarify this.

In paragraph 11, Dr. Freeman again claims, ". . . it is a fact that some people are injured in low-speed motor vehicle crashes with minimal or no property damage." I again know of no objective scientific evidence of physical injury to support this statement. The authors cited by Dr. Freeman (Farmer et al[6] and Tencer and Mirza[7]) reported symptoms, not objective evidence of physical injury. Dr. Freeman appears to be making the unproven and probably erroneous assumption that patients who complain of pain necessarily have a physical injury causing that pain. My article and *The Whiplash Encyclopedia*[1] both explain why this assumption is especially likely to be incorrect in chronic pain.

Ferrari[1] listed 200 references regarding the psychology of whiplash. There is also a much larger number of references on psychologic causes of chronic pain that Dr. Freeman either is unaware of or chooses to ignore. Ferrari's chapter 24 on tertiary gain explains better than I could why some caregivers sometimes rebel against established scientific facts.[1]

Dr. Freeman appears to have confused his beliefs and assumptions with facts. I believe anyone who reads *The Whiplash Encyclopedia*[1] with an open mind would agree that my conclusions are reasonable, based on the available information.

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Incorrect Posterior Axial Line

To the Editor: I read the article by Murphy et al[1] because of its important ramifications for those in primary care. I noted that Figure 4 is not correctly drawn; also, the legend is incorrect. In Figure 4, the legend states "Assessment of occipitoatlantal instability. Odontoid process and posterior axial line drawn along C2 should lie within 12 mm of basion. Greater distance signifies cranial distraction and anterior displacement." The posterior axial line as drawn in Figure 4 is not correct. It should be drawn along the line posterior to the odontoid process, as in line B in Figure 3. The posterior axial line as currently drawn follows the posterior articular masses of the atlas—C1, not C2. Readers of the *Journal* should be made aware of this serious error.

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