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STATE OF WASHINGTON
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No. 94559-4

SUPREME COURT OF THE STATE OF WASHINGTON

MICHAEL GILMORE, a single man,

Plaintiff-Petitioner,

vs.

JEFFERSON COUNTY PUBLIC TRANSPORTATION BENEFIT AREA, d/b/a Jefferson Transit Authority, a municipal corporation,

Defendant-Respondent.

PLAINTIFF-PETITIONER GILMORE'S SUPPLEMENTAL BRIEF

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I. INTRODUCTION

Michael Gilmore was rear-ended by defendant's bus. The impact caused injury to the disks in Mr. Gilmore's neck, as documented by MRI; the disks worsened over time, again as documented by MRI; and eventually his neck needed surgery. RP 657-58.

There is no dispute that the standard of review of the trial court's decisions herein is "abuse of discretion". This Court's most recent ruling on "abuse of discretion", *State v. Salgado-Mendoza (Ascencion)*, 93293-0 (October 12, 2017), is that an appellate court can find abuse of discretion only if "'***no reasonable person*** would take the view adopted by the trial court.'" Slip Op. at 6 (emphasis original). Defendant's position therefore is that: (1) no reasonable person would have found plaintiff's closing argument (which was never objected to and in fact was incorporated by the defense into its own closing) to be proper, or at least harmless; (2) no reasonable person would have excluded the testimony of Allan Tencer; and (3) no reasonable person would have obeyed RCW 51.24.100 when defendant provided no authority allowing its violation.[1]

[1]It is these three decisions by the Court of Appeals to which Mr. Gilmore assigns error. He asks that the Court of Appeals be reversed and the jury's verdict and Superior Court judgment be affirmed.

On appeal, defendant has repeatedly tried to reframe the issues away from the case it presented in Superior Court, and toward the case appellate counsel wishes defense trial counsel had presented. This is clear from defendant's emphasis on three irrelevant but potentially inflammatory themes, in oral argument at the Court of Appeals, and in its Answer to Petition herein, at 1-2: (1) there wasn't much damage done to Mr. Gilmore's vehicle, (2) the jury's verdict was $1.2 million, and (3) defendant is a taxpayer-supported government entity. This Court should not be fooled. The trial court record shows what truly occurred at trial.

Here is the case defendant now **wishes** it had tried:

> Michael Gilmore's truck was hit by our bus at low speed. The impact was small and he was only slightly injured. His injuries healed within weeks. Much later, he developed a serious, unrelated neck condition which eventually needed surgery. We are sorry our bus hit Mr. Gilmore, we are sorry he was hurt, and we are sorry when anyone needs surgery. But we did him no significant harm, and we are not responsible for his neck surgery. The jury should award him a modest amount for the harm we did, but far less than the amount he is asking for, and nothing for the surgery.

Here is the case the defendant **did** try:

> Michael Gilmore is a liar and a fraud! He scammed the VA out of a pension he didn't earn, and now he's trying to scam us out of money he doesn't deserve. He looks fine on surveillance video, and there's nothing wrong with him except he smokes and he's getting old. He didn't need medical treatment. We bet he cheated on his wife, and we think he's a drug addict or a drug dealer or both. Any verdict for him would be an outrage!

When we look to the record of what actually happened at trial, it is clear that the jury validly rejected defendant's case, that the trial court and jury did justice, that Mr. Gilmore's lawyer appropriately addressed the case the defendant presented, that Tencer's testimony was both unimportant and properly excluded, that collateral source evidence was properly excluded, and that the only injustice here was the decision by the Court of Appeals. Most of the facts established at trial are in Respondent's Brief to the Court of Appeals ("Respondent's Brief") at 5-19. Some additional events during the trial bear upon the issues before this Court, particularly plaintiff's closing argument, so they will briefly be summarized *infra*.[2]

II. STATEMENT OF THE CASE - THE DEFENSE THEORY AT TRIAL WAS THAT MR. GILMORE WAS A LIAR, A CHEAT, AND A FRAUD.[3]

Even before voir dire, defense counsel's theory was clear to all. "He was trying to commit a fraud. And I can prove it." RP 50. The trial judge restated the defense theory that "Mr. Gilmore is either a fraud or a

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[2]Plaintiff's closing and rebuttal took about 42 pages of transcript. Unfortunately defendant and Division II focused on tiny excerpts in reaching the wrong conclusion.

[3]In Respondent's Brief 15-18, and in the Petition for Review, p. 3 fn. 1, we listed numerous instances where the defense tried to convince the judge and jury that Mr. Gilmore was a liar and a fraud. We will try not to repeat all those instances here – instead we will provide many additional ones.

malingerer...", RP 56, and defense counsel made no attempt to contradict or correct the court.

Bowing to the inevitable, plaintiff's counsel told the jury in her opening that the defense would attempt to prove that Mr. Gilmore was a fraud, that the jury would need to decide whether he was, and that if they so found, they should award him nothing. RP 273. Defense counsel did not object to this characterization of the defense then or later, because that was exactly how defendant intended to – and did – present its case.

During plaintiff's case in chief, the defense questioned a lay witness about Mr. Gilmore's reputation for honesty at work. RP 466. Defense counsel repeatedly asked this witness whether he "knew" that Mr. Gilmore had allegedly made false statements to his doctors. RP 468-71, 473.[4] The defense asked another lay witness whether he knew Mr. Gilmore received disability from the VA for his low back. RP 309.[5]

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[4]Obviously, Mr. Gilmore's co-worker couldn't know what Mr. Gilmore told his doctors. These questions were intended to convey to the jury defense counsel's belief that Mr. Gilmore was a liar.

[5]Mr. Gilmore was not claiming low back injury from this crash, so the purpose of these questions was not to show that Mr. Gilmore had a preexisting bad back, but to bolster the defense claim (an attempt at improper "propensity evidence") that Mr. Gilmore was already committing fraud before this crash. Defense counsel emphasized this very point in his closing. RP 1023.

Another of Mr. Gilmore's lay witnesses was a female neighbor for whom Mr. Gilmore had done some plumbing projects before the crash. RP 561. Defense counsel sought to impugn Mr. Gilmore's character by asking this witness whether Mr. Gilmore's then-wife "had any issues" or "seemed to mind" when Mr. Gilmore helped his neighbor out. RP 572.

The defense even called one of Mr. Gilmore's other neighbors as its first witness, to testify that Mr. Gilmore had a bad reputation for truth and honesty. RP 827-34. As disclosed on cross exam, this witness had a grudge – he had built a fence encroaching on Mr. Gilmore's property, and Mr. Gilmore had sued this neighbor to have the fence removed. RP 833.

The defense's CR 35 examiner, Dr. Barbara Jessen, does not treat patients but instead does "defense evaluations". RP 852. She admitted that Mr. Gilmore was hurt in the crash, RP 873, but claimed his crash-related injuries healed within 6 to 8 weeks, RP 887. She testified that people who have neck injuries do better if they don't get treatment, RP 887-8, and she attributed Mr. Gilmore's abnormal MRI results to smoking, not to the crash. RP 892. She falsely reported that Mr. Gilmore had extensive pre-crash medical history regarding his neck, admitting only on cross that he did not. RP 904-06.

When shown on cross that a treating doctor, Dr. Suffis, and Dr. Niacon, and Dr. Marinkovich, and surgeon Dr. Cain all attributed Mr. Gilmore's ongoing neck problems to the crash, Dr. Jessen would not change her opinion – she just "disagreed" with them all. RP 923-29.

Dr. Jessen also speculated during her testimony that Mr. Gilmore is a drug addict, RP 946, and that if he isn't a drug addict, perhaps he is a drug dealer. RP 947. Plaintiff objected and moved to strike this speculation, but was overruled. *Id.*

III. ARGUMENT - PLAINTIFF'S CLOSING ARGUMENT WAS PROPER, AND THEREFORE DREW NO OBJECTION TO THE TRIAL COURT.

Defendant's appellate counsel claims, and Division II agreed, that plaintiff's closing was improper, but defendant's trial counsel clearly did not believe this at the time of trial. "[T]he lack of a clear and prompt objection is strong evidence that counsel perceived no error." *In re Black*, 187 Wn.2d 148, 154, 385 P.2d 765 (2016). Defense counsel never objected during closing argument or afterward.

This is **not** a case of counsel failing to object because he was inexperienced or incompetent.[6] Defense trial counsel was an experienced courtroom lawyer, a former felony prosecutor, and a member of the Bar

[6]There is no 6th Amendment right to effective assistance of counsel in a civil case in any event.

since 1980. Rather, in the context of the trial, neither the lawyer nor the trial judge perceived any impropriety in plaintiff's closing argument.

After the jurors were excused to begin deliberations, the trial judge asked, "Is there anything we need to put on the record or do?" Moments later, he asked, "Anything else?" Defense counsel twice said "no". RP 1036-37. After the jury announced its verdict, the trial judge asked yet again, "Is there anything the attorneys needed to put on the record...about anything?" and defense counsel declined yet again. RP 1044. It was only after appellate counsel was hired that plaintiff's closing argument allegedly became "inflammatory," "flagrant" and "ill intentioned." Op. at 23.[7]

Arguments are proper when based on the evidence or reasonable inferences from it, and "counsel are permitted latitude to argue the facts in evidence and reasonable inferences" in their closing arguments. *State v. Dhaliwal*, 150 Wn.2d 559, 577, 79 P.3d 432, 442 (2003). Faced with a defense that repeatedly attacked Mr. Gilmore's integrity using innuendo and biased, non-credible witnesses, plaintiff's counsel drew reasonable inferences: that defendant was trying to use its wealth and power to avoid ac-

[7]It was defense appellate counsel – not trial counsel – who prepared and argued defendant's CR 59 motion for a new trial or remittitur. CP 402.

countability for the harm it had done to Mr. Gilmore, and that it was the defense that was trying to commit fraud, to avoid paying what it owed.

Plaintiff's counsel pointed out that defense counsel had exaggerated the nature and seriousness of Mr. Gilmore's preexisting conditions, RP 980-82. She pointed out that Dr. Jessen disagreed with every other doctor, RP 986-8, and noted that Dr. Jessen gave inaccurate testimony about Mr. Gilmore's medical history, RP 991. She contrasted the testimony of Dr. Marinkovich, who explained the anatomy and medicine of Mr. Gilmore's neck injuries and pain, with Dr. Jessen's failure to explain anything. RP 995.

The defense had surreptitiously followed and videoed Mr. Gilmore and his family for 20 hours over 2 days, but showed the jury only 1 hour of video. Plaintiff's counsel asked rhetorically where the rest was; certainly the jury was entitled to draw a reasonable inference that the defense had failed to show any video favorable to Mr. Gilmore. RP 983.

Plaintiff's counsel reminded the jurors how the defense had cross examined Mr. Gilmore's female neighbor about "how his wife felt", RP 982, had presented a witness with an axe to grind because of a boundary dispute as a "bad character" witness, RP 984, and had called Dr. Jessen to

say that Mr. Gilmore wasn't really hurt and might be a drug addict or drug dealer. RP 979. Plaintiff's counsel argued that the defense had impugned Mr. Gilmore's character in an effort to avoid paying the debt defendant owed him. RP 982. She reminded the jury that it was their job to decide whether the evidence supported the defense theory. RP 977.

Plaintiff's counsel then referred to the defendant as "the government"[8] and talked about "fighting the government", as in the expression, "you can't fight city hall."[9] She began discussing the damage instruction and described the nature and extent of Mr. Gilmore's injury, disability, physical pain, and mental pain. Counsel spoke simply and clearly from the evidence about the effects Mr. Gilmore's injuries had had upon him and his life. RP 996-1003. She made a valid type of "per diem" argument that is commonly made by plaintiff lawyers, comparing a life of pain and injury to having a very unpleasant job, and estimating the number of hours or days of pain someone would have to experience such

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[8]Defendant Jefferson County Public Transportation Benefit Area is in fact a government entity and throughout this case has been very happy to be one when that means paying only the special low government post-judgment interest rate, RCW 4.56.110(3)(a), CP 727, and being permitted to appeal the judgment without posting a supersedeas bond, RCW 4.96.050. *See* Appendix A.

[9]This expression may date back to the Tammany Hall/Boss Tweed era of 1850's New York City. Defense counsel made no objection, and in fact when it was his turn, he dismissed this and other arguments as "theater". RP 1007.

unpleasantness. RP 1002-04. She discussed a range of values for an hour or a day of pain, including Dr. Jessen's daily charge of $5,000, though she acknowledged that led to too high a figure. She made it clear to the jury that it was their decision, and she suggested that for all of Mr. Gilmore's past and future pain, disability, and lost ability to enjoy life, a total of $1.8 million was reasonable. RP 1005.

Far from being outraged, defense counsel made a tactical decision to leverage plaintiff's argument. He analogized both sides' arguments to theater: "A lot of this is theater. It's putting on a show." RP 1007. He dismissed the phrase "the government" by calling it a "lawyer technique", RP 1007-08.[10] He even told the jurors this is normal, expected lawyer behavior: "that's how you present your case", RP 1008 (emphasis added).

Defense counsel then went on the attack. He said that Mr. Gilmore had lied and intentionally withheld information, has "unclean hands", and should get nothing. RP 1022. He accused Mr. Gilmore of "hiding the facts", RP 1024-25, and of receiving a VA pension he did not deserve. RP 1023. He repeatedly called Dr. Marinkovich and Dr. Niacon

[10]Had the argument, in context, truly been inflammatory and prejudicial, and had defense counsel objected, and had he been sustained, then plaintiff's counsel would have used different words, such as "defendant" or "the bus company" or "the transit authority". The defense claim that no objection or instruction could "cure" the claimed prejudice is simply wrong.

"courtroom doctors", RP 1010-11, 1019, 1020,[11] and he twice accused Dr. Marinkovich of "making stuff up". RP 1015-16. He attacked Mr. Gilmore's lay witnesses, saying of one that he "doesn't know the difference between the truth and a lie." RP 1026.

And even though the defense had agreed and the trial court had ordered in limine that the defense not argue that money for plaintiff would be "a windfall", RP 18, defense counsel argued to the jury that the court system was not a "lottery", and not an "opportunity to retire." RP 1023. He told the jurors they should award $1,000 or nothing. RP 1028. Defense counsel concluded the substance of his closing thus: "It all boils down to credibility of the Plaintiff. How can you believe anything he says? How can you believe anything he says?" RP 1027-28.

In rebuttal, plaintiff's counsel correctly pointed out that defense counsel had said, "I won't call him a liar, a cheat, and a fraud" and then had proceeded to do just that. RP 1029. She responded to the defense accusation of VA fraud by pointing out Mr. Gilmore's honesty: the documents showed he had told the VA he could perform all his activities and

[11]Plaintiff's counsel also referred to Dr. Jessen as a "courtroom doctor" in her closing, RP 978. Sauce for the goose, sauce for the gander.

functions, and they nevertheless gave him a partial disability rating for his low back. RP 1030.

Defense counsel had started his closing in a strange way, talking about instances where the police had killed someone and then tried to escape accountability for their actions, only to be stymied by video of their misdeeds. His point appeared to be that the jurors should heavily weigh the surveillance video of Mr. Gilmore, RP 1005-06, but he certainly thereby accentuated the issue of "lack of government accountability".

As defense counsel had used plaintiff's themes in his own closing, so did plaintiff's counsel use defendant's themes in her rebuttal. She told the jury that Mr. Gilmore was trying to hold the government defendant accountable for the injury done to him, but that the defendant would not be held accountable unless the jurors did so. She argued that Mr. Gilmore had presented abundant evidence of his injuries, and that it was for the jury to determine the fair amount of compensation Mr. Gilmore was owed and thereby hold the defendant accountable. RP 1031-32. She did not just single out "the government" – in her conclusion, she told the jurors that in our society, "**people** are going to be held accountable when they're careless and they hurt somebody." RP 1032 (emphasis added).

The jury returned a verdict in Mr. Gilmore's favor. CP 401. Defendant then hired appellate counsel, who moved the trial court for remittitur or a new trial. CP 402-25. The trial court properly denied it.

In its CR 59 motion, defendant cited *Aluminum Co. of America v. Aetna Cas. & Sur. Co.*, 140 Wn.2d 517, 538, 998 P.3d 856 (2000) for the proposition that criminal cases on prosecutorial misconduct provide criteria from which a court should decide whether to overturn a civil verdict on grounds of misconduct. CP 404. What the case actually held, however, was: "the circumstances of a civil case, where life and liberty are not at issue, militate in favor of a standard that more generally upholds trial court decisions." 140 Wn.2d at 539 (emphasis added). The Court also held, quoting MOORE'S FEDERAL PRACTICE (ellipses original, citation omitted, emphasis added):

> [T]he movant must establish that the conduct complained of constitutes misconduct (and **not mere aggressive advocacy**) and that the misconduct is prejudicial **in the context of the entire record**.... The movant must ordinarily **have properly objected to the misconduct at trial**, ... and the misconduct must not have been cured by court instructions.

Id. The trial judge witnessed the entire trial and is in the best position to know whether any particular arguments or comments were made in such a manner as to be inflammatory and prejudicial. The trial court followed

case law precisely, holding "This was a hard-fought case characterized by aggressive advocacy, but the Court does not find, in the context of the entire record, that there was any event, misconduct, or discovery violation sufficient to justify a new trial or a remittitur (emphasis added)." CP 724.

IV. ARGUMENT - THE DECISION TO EXCLUDE TENCER WAS PROPER, AND TENCER WAS UNIMPORTANT TO DEFENDANT.

The defense and Division II focused solely on ER 703, which does permit expert testimony to be based on data not itself admissible. However, ER 703 also requires a showing that the data underlying the opinion is reasonably relied upon, not just by the proffered witness, but by other experts in the witness's field, and not just for purposes of litigation. *State v. Nation*, 110 Wn. App. 651, 663, 41 P.3d 1294 (2002). That foundation is missing from this record. Indeed, the record implies the opposite: there is no general acceptance in the engineering community that one can determine injury or lack thereof by considering vehicle damage. CP 213-17.

Even if a proper foundation had been provided, Tencer's testimony was properly excluded because the trial judge found it violated ER 403. The trial court specifically stated at RP 39 that Tencer's proffered testimony had an "aura of authority that I don't think is reasonable or

justified", *see*, *Stedman v. Cooper*, 172 Wn. App. 9, 16, 292 P.3d 764 (2012), and was "confusing ... [and] ... misleading to the jury".

Even if all the biomechanical calculations were correct, which they are not, the numbers are worse than useless. As the Court held in *Schulz v. Wells*, 13 P.3d 846, 852 (Colo. App. 2000), cited with approval in *Stedman* at 19, "Because of the lack of similarity between horizontal G-forces sustained during daily living activities and the numerous forces sustained during an unexpected rear-end automobile collision, evidence of the former would have been misleading." The trial court here correctly held that Tencer's testimony would be both confusing and misleading to the jury.

This type of biomechanical testimony also is inadmissible under ER 702. While most evidentiary decisions are discretionary, this Court has excluded certain types of evidence as a matter of law.[12]

Defense biomechanical testimony about the forces generated during a crash should never be admissible, because it is not helpful to the jury. Washington has long adhered to the "eggshell plaintiff" rule. *Jordan*

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[12] *See*, *e.g.*, *Kirk v. WSU*, 746 P.2d 285, 109 Wn.2d 448 (1987) (evidence plaintiff had an abortion inadmissible); *Salas v. Hi-Tech Erectors*, 168 Wn.2d 664, 230 P.3d 583 (2010) (evidence of immigration status inadmissible); *State v. Greene*, 139 Wn.2d 64, 79, 984 P.2d 1024 (1999) (evidence of dissociative identity disorder inadmissible despite being generally accepted, because it is not helpful to the trier of fact).

v. Seattle, 30 Wash. 298, 302-03, 70 P. 743 (1902). An engineer's testimony that a crash was "no big deal", whether dressed up in numbers or not, is not helpful to jurors in answering the question they must answer: did this particular crash cause these particular injuries to this particular plaintiff? Even if for some reason Mr. Gilmore were exceptionally vulnerable to injury caused by a rear-end collision, that is no defense. *Id.* Therefore, knowing how much force was applied is not helpful unless the jury also knows how much force it would have taken to injure Mr. Gilmore specifically. There is no way to know that.

"Scientific evidence that does not help the trier of fact resolve any issue of fact is irrelevant and does not meet the requirements of ER 702." *State v. Greene*, 139 Wn.2d 64, 73, 984 P.2d 1024 (1999). Expert testimony that does not tell the jury about the effect of a particular event upon a particular person is speculative and irrelevant. *State v. Lewis*, 141 Wn. App. 367, 389, 166 P.3d 786 (2007) Biomechanical testimony fits this description and should be excluded.

Finally, a trial court should be affirmed if there is any basis in the record to do so. *Thomas v. French*, 99 Wn.2d 95, 104, 659 P.2d 1097 (1983). The defense and Division II disregarded the voluminous materials

supporting the motion in limine regarding Tencer,[13] and the extensive oral argument about his proffered testimony, RP 33-39, and instead focused on just one phrase[14] out of a lengthy oral decision. From this isolated portion of the trial judge's oral ruling, Division II incorrectly concluded that the trial court had somehow abused its discretion.

"Abuse of discretion" means no reasonable person would take that position. *Salgado-Mendoza, supra.* A single alleged slip of a judge's tongue should not be the basis for finding an "abuse of discretion", when the record herein shows that at least 9 Washington Superior Court judges have excluded defense biomechanical testimony in car crash cases. CP 54-56, 179-80, 210-11, 231-32, plus the trial courts in *Stedman v. Cooper*, 172 Wn. App. 9, 292 P.3d 764 (2012) and *Berryman v. Metcalf*, 177 Wn. App. 644, 312 P.3d 745 (2013), plus numerous courts in other states, some of which are cited in *Stedman* at 19-20.

Division II incorrectly stated at Op. 18 that excluding Tencer prevented defendant from presenting its theory of the case. But Tencer's

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[13]There were 10 pages of briefing and almost 200 pages of exhibits showing that Tencer's opinions and proffered testimony were scientifically and legally flawed, CP 47-232 (about 15 pages of one of the transcripts pertains to another witness in another trial).

[14]"based on facts that are not going to be in evidence", RP 39.

exclusion was harmless to defendant. As established above, defendant's theory of the case was that Mr. Gilmore was trying to commit fraud.[15]

To the extent that defendant's theory was that this crash was a minor impact, defendant had other, better evidence available to it regarding the nature of the impact: photos of vehicle damage which were admitted into evidence, RP 750-53; the ER record in which Mr. Gilmore himself described the crash as "low velocity", RP 989-90; and testimony about the impact from bus passenger David Sachi, RP 834-36.[16] The unimportance of Tencer is further highlighted by the fact that defendant made no motion for reconsideration, never argued that Mr. Gilmore's trial testimony about the crash had "opened the door" to Tencer, and did not argue his exclusion in its CR 59 motion. The claim that Tencer's exclusion was reversible error was first made in defendant's opening brief to Division II, after the trial court had rejected defendant's CR 59 claims of excessive verdict and attorney misconduct.

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[15] *See also* Op. 4 ("Jefferson Transit argued that because Gilmore was being untruthful to his treating doctors regarding past symptoms, the evidence could prove he tried to commit fraud.")

[16] For some reason, defendant chose not to call its own employee, the bus driver, as a witness regarding the impact.

Division II incorrectly stated that there was "contradictory evidence that the collision was not significant enough to cause injury", Op. 18, presumably suggesting that Tencer could somehow have cleared up this confusion. But there was in fact no evidence that this collision couldn't cause injury. Even low speed collisions do cause injury, CP 219-20, 222-30. Defendant's witness Dr. Jessen conceded that Mr. Gilmore was injured by this crash, RP 873, albeit slightly, RP 887. She admitted that a person can be uninjured in a very severe crash and can be injured in a non-severe crash, depending upon his or her vulnerability. RP 875. And even Tencer does not claim he can rule out injury for a given crash. CP 66-67.

V. ARGUMENT - THE COLLATERAL SOURCE RULE APPLIES TO THIS CASE AND COLLATERAL SOURCE EVIDENCE WAS PROPERLY EXCLUDED.

RCW 51.24.100 prohibits introduction of evidence of L&I payments, as does the common law collateral source rule. Defendant argues in part that the statute and the collateral source rule do not apply because plaintiff's trial counsel made the decision not to introduce Mr. Gilmore's medical bills into evidence. Defendant cites no authority for this proposition, and we are not aware of any.

Defendant also claims, again without authority, that the statute and the collateral source rule do not apply because, according to defendant,

L&I cannot subrogate here pursuant to *Tobin v. Department of Labor & Industries*, 169 Wn.2d 396, 239 P.3d 544 (2010). Answer at 18.[17] The statute's clear language contains no such limitation. Nor does the common law collateral source rule, which predates subrogation and reimbursement clauses by decades.

The collateral source rule was adopted because it is "contrary to public policy and shocking to the sense of justice" to allow a tortfeasor to benefit from collateral sources. *Heath v. Seattle Taxicab Co.*, 73 Wash. 177, 186, 131 P. 843 (1913). Only in the late 1950's did insurers even begin inserting reimbursement/subrogation clauses into their medical benefit policies. *Mahler v. Szucs*, 135 Wn.2d 398, 414 fn. 6, 957 P.2d 632 (1998).[18] The issue of reimbursement or subrogation, and the inadmissibility of collateral source evidence, have nothing whatsoever to do with each other.

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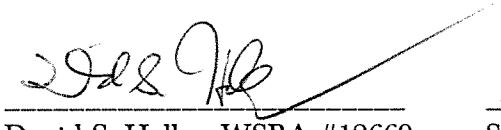
[17]This issue is not before the Court, nor is it ripe unless this Court reinstates the judgment and defendant satisfies it. If all that occurs, L&I has a process for determining its share of a recovery and for resolving disputes regarding that determination.

[18]In the early 1980's when plaintiff's lead appellate counsel first came to the Bar, there were still union benefit health insurance contracts which had no subrogation or reimbursement language. Some might argue that lack of subrogation results in a "windfall" to the injured person, but we believe the better view is that subrogation in fact provides a windfall to the insurer, which does not refund premiums it collected even when it is reimbursed for benefits it paid out. *See*, Roger M. Baron, *Subrogation: A Pandora's Box Awaiting Closure*, 41 S. D. L. Rev. 237, 243 (1996).

DATED this 3rd day of November, 2017.



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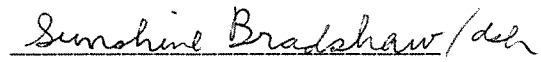

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NOTICE JUDGMENT IS SUPERSEDED WITHOUT BOND

RECEIVED
SEP 08 2015
FELLER LAW FIRM, P.L.L.C.

IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON
FOR THE COUNTY OF JEFFERSON

MICHAEL GILMORE, a single man, Plaintiff/Respondent, v. JEFFERSON COUNTY PUBLIC TRANSPORTATION BENEFIT AREA, dba Jefferson Transit Authority, a municipal corporation. Defendant/Appellant.	No. 10-2-00390-7 NOTICE THAT JUDGMENT IS SUPERSEDED WITHOUT BOND

Appellant Jefferson County Public Transportation Benefit Area supersedes the Judgment presently on review, without bond, pursuant to RAP 8.1(f) and RCW 4.96.050.

DATED this 1st day of September, 2015.

SMITH GOODFRIEND, P.S.

By: _____
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TRANSCRIPT EXCERPTS - POST-CLOSING COLLOQUY

1 phone call telling you that the case is over with.
2 And once that's done, you won't be here to hear my
3 final instruction, but once the case is completely done and
4 you're notified of that, you're free, then, to be able to
5 discuss the case or talk to anybody about it, uh, or do
6 anything you want. But as of right now, you're still part
7 of the jury pool. And, um -- and the -- the only
8 difference is you're not going in there and deliberating,
9 but you may be called on to do that.
10 Um, so, when, uh -- so, Mr. Erickson will take you
11 out, uh, to the jury room now to begin deliberating. And,
12 um, he'll handle all of the logistics of your work and your
13 schedule and lunch and all that stuff.
14 And Juror Number Twelve, make sure you have all your
15 personal belongings and, um, uh -- to take with you when
16 you go. And, uh, as I said, thank you for your service so
17 far.
18 And, um, the Bailiff will advise the Court, then, uh,
19 of anything else or when we may need to reconvene. Okay.
20 So, go ahead and go with Mr. Erickson. Thank you.
21 (JURY EXITS AT 11:22 A.M.)
22 THE COURT: Okay. Uh, two things. Uh, is there
23 anything we need to put on the record or do?
24 MS. BRADSHAW: No, Your Honor.
25 THE COURT: Okay.

1 MR. ROVANG: No.
2 THE COURT: Um, we -- we have what we've always
3 called, uh, the Ten Minute Rule. And if you could provide
4 your cell phone numbers to the Clerk, um, whenever we need
5 the jury to come back in and your presence is necessary,
6 then the Clerks will give you a call. And, um -- and the
7 idea is to try to be here within ten minutes of that call.
8 Um, and Mr. Gilmore, the same thing would apply to
9 you. You -- because you'd want to be present, unless your
10 attorney tells you differently, I guess. So, um, anything
11 -- anything else? Any questions?
12 MR. ROVANG: No.
13 MS. BRADSHAW: No, Your Honor.
14 THE COURT: Yeah. Okay. Thank you very much.
15 THE CLERK: Please rise. Court will be at recess.
16 (TAPE PAUSED)
17 (TAPE RESUMED)
18 THE CLERK: Please rise. This ends the recess.
19 Superior Court is again in session. The Honorable Keith
20 Harper presiding.
21 THE COURT: Thank you. Please be seated. Okay. Mr.
22 Erickson, if -- if you'd step up, I guess. Okay. I'm not
23 sure, uh -- we -- we were -- I was anticipating, um,
24 recessing at 4:15 today, but the Clerk told me the jury
25 wanted to come out here. Do they have a verdict?

1 MR. ROVANG: Your Honor, I'd like an opportunity if
2 any jurors are willing to, uh, stay behind, uh, and -- and
3 talk to me in the jury room, I'd appreciate it. Counsel,
4 too?
5 MS. BRADSHAW: Yeah, definitely.
6 THE COURT: Yeah. You bet. Um, the -- the building
7 will stay open until 4:30. I think somebody might shoo you
8 all out, um, at 4:30. But, um, yeah, anybody that wants to
9 take up the attorneys on that, you're welcome to stay and
10 talk. And, uh, like I say, it's totally up to you and it's
11 a very common practice. There's no restrictions
12 whatsoever, so.
13 Okay. Well, thank you again. And, uh, Mr. Erickson
14 will lead you out. And, um, go from there.
15 (JURY EXITS AT 4:10 P.M.)
16 THE COURT: Um, is there anything the attorneys needed
17 to put on the record or any --
18 MR. ROVANG: I don't think so.
19 THE COURT: -- about anything?
20 MS. BRADSHAW: No, Your Honor.
21 THE COURT: Okay. I, um -- I just wanted to take the
22 opportunity to thank each of you. Um, whenever I do a
23 trial, um, I always learn a lot. And I appreciate that.
24 And, uh, I appreciate the hard work that you both obviously
25 put into this case and everything. So, um, anyway. And I

CLERK'S PAPERS EXCERPT: "REAR-END IMPACTS: VEHICLE AND OCCUPANT RESPONSE"

PubMed

Display Settings: Abstract

J Manipulative Physiol Ther. 1998 Nov-Dec;21(9):629-39.

Rear-end impacts: vehicle and occupant response.

Davis CG.

Abstract

BACKGROUND: There is a controversy regarding the likelihood of injuries sustained when one car strikes another at a relatively low speed with little or no vehicle damage. Plaintiffs often claim injuries whereas defendants counterclaim that injuries could not have occurred with such a relatively minor impact.

OBJECTIVE: To review the dynamics of low-speed rear-end collisions resulting in little or no visible damage and to decide whether occupant injury can occur; also, to discuss diagnostic examination and treatment that may be helpful to the clinical practitioner.

DATA SELECTION: A Medline search for articles discussing low-speed rear-end collisions was conducted. Other articles and studies were reviewed that discussed low-speed rear-end collisions and factors impacting the neuromusculoskeletal system relevant to clinical practitioners. Articles included were human low-speed rear-end tests, lab tests on cadavers, automotive engineering articles, and peer-reviewed journal articles on whiplash. A few live animal and simulation studies were considered for the background of possible injury mechanism and vehicular deformation. Excluded were non-rear-end collison and single case reports.

DATA SYNTHESIS: The data were studied to find a relationship between the resultant vehicle dynamics and occupant movement, biological mechanisms of injury and the neurological mechanisms causing complaints. Data were also studied to investigate objective findings supporting subjective complaints.

CONCLUSION: [redacted] There does not seem to be an absolute speed or amount of damage a vehicle sustains for a person to experience injury. Crash tests indicate that a change of vehicle velocity of 4 km/hr (2.5 mph) may produce occupant symptoms. Vehicle damage may not occur until 14-15 km/hr (8.7 mph). Occupant soft tissue and joint injuries resulting from low-speed vehicle collisions respond positively to afferent stimulation of mechanoreceptors. The diagnosis of the occupant injuries relies on standard orthopedic neurological testing, autonomic concomitant signs and qualitative and quantitative testing.

Comment in

Rear-end impacts: vehicle and occupant response. [J Manipulative Physiol Ther. 2000]

PMID: 9868635 [PubMed - indexed for MEDLINE]

Publication Types, MeSH Terms

LinkOut - more resources

CLERK'S PAPERS EXCERPT: "LACK OF RELATIONSHIP BETWEEN VEHICLE DAMAGE & OCCUPANT INJURY"

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970494

Lack of Relationship Between Vehicle Damage and Occupant Injury

Malcolm C. Robbins
Robbins & Assocs.

Reprinted from: Motor Vehicle Safety Design Innovations (SP-1226)

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Lack of Relationship Between Vehicle Damage and Occupant Injury

Malcolm C. Robbins
Robbins & Assocs.

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Abstract

A common misconception formulated is that the amount of vehicle crash damage due to a collision, offers a direct correlation to the degree of occupant injury. This paper explores this concept and explains why it is false reasoning. Explanations with supporting data are set forth to show how minor vehicle damage can relate or even be the major contributing factor to occupant injury. Mathematical equations and models also support these findings.

Introduction

A common concept formulated is that the amount of motor vehicle crash damage offers a direct correlation to the degree of occupant injury. This paper explores this concept and explains why it is false reasoning. This false reasoning is often applied by insurance adjusters, attorneys and physicians and frequently results in costly unjustified litigation. Due to this litigation process, the injured parties often are not compensated, resulting in unjustified hardship to the party who has already been injured.

The object of this paper is to present a clear understanding of vehicle body performance when it is subjected to crash dynamics and the relationship to occupant dynamic responses and resulting injury.

Theory

One of the major factors relating to occupant injury due to a collision is the G force to which the occupant is subjected. [1][2] Even with seat belts air bags and other measures, severe injury and fatality occurs when a vehicle is subject to a collision. [3][4][5][6] This is a rather complex subject to answer in a single paper, but fundamentally even when seat belts are used, the G force sustained by the vehicle beyond the crush zone or arresting distance is transferred to the occupant.

Galileo Galilei formulated an equation that can be used to demonstrate the G force an occupant will receive, assuming a "fixed" seated position. If an object starts from rest, Galilei's equation states: [1]

$$V = \sqrt{2as} \qquad (1)$$

where V = Velocity of object
a = acceleration rate
s = distance moved by object.

Rearranging Equation 1 to get deceleration, we have:

$$a = \frac{V^2}{2s} \qquad (2)$$

where s = arresting or crush distance
V = Velocity at time of impact
a = deceleration

Applying this formula (2) to the scenario of a pole vaulter. If a pole vaulter jumps 6.5 meters (20 feet), his speed when reaching a 1.5-meter (5-foot) safety mat can be calculated thus, using Equation 1:

$$V = \sqrt{2as} \qquad (1)$$

where $s = 6.5 - 1.5 = 5$ meters
$a = 9.81$ m/sec^2

hence: V = 11.29 m/sec or 40 km/hr (25 mph)

The resulting G force to which the pole vaulter is exposed can be calculated to be as follows, using Equation 2:

$$a = \frac{V^2}{2s} \qquad (2)$$

where V = 11.29 m/sec
$s = 1.5 - 0.5 = 1$ meter

hence: a = 63.7 m/ sec^2 (6.5 Gs)

If the vaulter impacted a concrete surface, the results would be clearly different. It is the amount of crush in the safety padding that prevents injury to the pole vaulter.

Applying the formula to vehicles which impact a solid brick wall:

First Scenario: Vehicle Green is traveling at a velocity of 12 meters/second (25 mph) and crushes 1 meter (3.1 feet) while impacting a solid brick wall. Using equation (2) above, then V = 8 m/sec (25 mph), s = 1 m (3.1 feet) and deceleration is:

$$a = \frac{12^2}{2} = 72m/\sec^2 = 7G$$

Second Scenario: Vehicle Red undertakes same velocity as Vehicle Green but crushes only 0.2 meters while impacting the solid brick wall. Deceleration is:

$$a = \frac{12^2}{0.2} = 360m/sec^2 = 37G$$

The results show that the greater the crush distance of the vehicle, the less the G force received by the occupant.

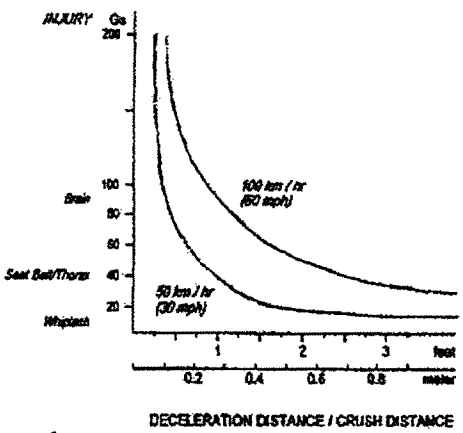

Figure 1

The graph shown in Figure 1 demonstrates the effect a vehicle's crushing distance has on the G force with a fixed collision speed.

Discussion

The average force an occupant of a motor vehicle experiences with normal driving is in the range of 0.2 to 0.5 G. Under these conditions motor vehicle occupants can readily change direction or speed with the vehicle. Some braking will impose a resulting force of 0.9 G, which may cause unrestrained occupants to be thrown forward in the occupant area. The limiting factor is the amount of coefficient of friction available between tire and road for braking and steering. Injury has been known to result due to severe braking, typically when occupants did not have time to brace themselves and were restrained.

If a motor vehicle impacts an object, loads on occupants can rise to very high values. When this takes place, the unrestrained occupant cannot keep pace with the vehicle's change in speed or direction. Hence the unrestrained occupant continues to move within the interior passenger compartment, colliding with the compartment surfaces such as steering wheel, windshield or dashboard. The introduction of seat belts is an attempt to keep the occupant restrained and moving along with vehicle speed and direction. Air bags and padded interior surfaces are provided to cushion the occupant's limbs and head, which contact with occupant interior surfaces.

While the amount of crush a vehicle sustains does not relate to occupant injury, provided no penetration occurs to the occupant compartment, the amount of crush does relate to the impact velocity or speed in the event of a collision with another vehicle or object. In fact, evaluation of occupant injury when related to vehicle damage can only be made when several factors are taken into account. Some of these factors are the following:

Dynamics of force applied to occupant.

- Velocity of vehicle or objects on impact.
- Crushing or arresting distance of vehicle or object. [7][8]
- Ability of vehicle or objects to dissipate the energy of the impact.
- Combination of above factors will establish the dynamics of force applied to occupant.
- Initial positioning of occupant in relation to safety devices such as seat belts or air bags.

Physical condition of occupant.

- Degree of muscular stimulation at the time of impact, i.e., was impact anticipated by occupant?
- Structural strength of occupant, i.e., sex, age, bone mineral content and joint strength. [9]
- Geometric dimensions, i.e., height, weight.

One main factor for determining the dynamics of occupant injury due to a motor vehicle collision is the amount of crush or arresting distance, known as value "s" and previously discussed. This value can vary a great deal from vehicle to vehicle and its location on the vehicle. If we examine a soft drink extruded aluminum can and likea it to a motor vehicle body, several observations can be made:

- Firstly, force applied on the top of the can downwards meets a greater resistance than a force applied to the sides. Clearly the type of structure of the can plays a major part in the deformation resistance.
- Secondly, if a force is applied to the top, a relative great deal of resistance is initially met, then slowly, as the can is crushed, the amount of resistance deteriorates and the can yields.

Likewise, on a vehicle with a chassis, no serious visual deformation may occur even though it is subjected to relatively high speeds of impact. Classically, we see this in the case of pickup trucks or all-terrain vehicles that are traditionally fitted with a solid bumper-to-bumper chassis. Many of these types of vehicles are subjected to relatively severe impacts with little or no resulting damage to their bodies and bumpers. The classic whiplash injury associated with a great deal of litigation is most likely founded on the reasoning that if there was little or no vehicle damage, no injury can result. Motor vehicle bodies or bumper-to-bumper chassis offer little or no crushing effect on arresting obstacles when impacted; thus, relatively high G forces can be experienced by occupants when rear-ended, resulting in whiplash injury. The use of stiff motor vehicle bodies and chassis will also produce a spiked G force loading to occupants, even if little damage occurs to vehicle body or chassis.

Spike loading is a result of a non-linear yielding of a vehicle body, as previously discussed in the scenario of the tin

can. In actual practice, deceleration rates during an automobile collision are rarely uniform, especially when chassis, drive trains and mounting panels are involved in the collision.

It is not uncommon to see a motor vehicle that has experienced mass destruction and damage, yet the occupants sustained little or no injuries. This is often a prime example of a situation in which the vehicle or vehicles have absorbed the dissipating kinetic energy of the collision. The occupants are thus not subjected to severe G forces. It is for this very reason that racing cars, when seen in a collision, appear to almost shed their body structure. Wheels are seen detaching and the body structure is seen to dissipate and crush almost in every direction. High-performance racing cars as seen on the Grand Prix circuit are designed with state-of-the-art crash engineering. The main outside structure of these racing cars is designed to allow for crushing and to dissipate energy in the event of a collision. The driver is protected by a rigid enclosure and is also very effectively restrained. These design factors in high-performance crash engineering account for the low driver-injury rates, even though the collisions involve very high speeds. So here we see heavy vehicle-body damage and relatively low occupant injury rates. i.e., the body of the racing car is sacrificed to prevent driver injury or death.

Summary

The amount of crush or damage received by a motor vehicle in a collision is an indication of velocities involved when the stiffness of the motor vehicle and object or objects is known. However, the crush damage does not relate to the expected occupant injury, i.e., the more vehicle damage, the more chance that the occupant is injured, is not a conclusion that can be made. In fact, it is more likely the reverse. If the occupant is decelerated over a greater time/distance due to a large crush/arresting distance, then the likelihood of injury is reduced.

This conclusion has been demonstrated by both mathematical expression and practical examples. The first example is that of the pole vaulter who survives his 5-meter (16-foot) drop by the crush of the padding or mat. It is this crush which breaks the vaulter's fall and hence allows for increased stopping distance and time. The second practical example is that of the high-performance racing car which makes use of a rigid driver compartment for protection. However, the compartment is surrounded by a body which is designed to allow for crush or deformation due to a collision. The result is a reduced number of injuries or fatalities.

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About the Author

Malcolm C. Robbin's training and education began with a formal engineering apprenticeship in the UK. His qualifications include City and Guilds in Manufacturing and Mechanical Engineering and a B.Sc. degree in Production Engineering. He is a member of the American Society of Safety Engineers and the Institution of Production Engineers and Industrial Managers in the UK.

Mr. Robbins has worked as an engineer in the electronics, electro-mechanical, aerospace and automobile industries and has been a full-time faculty member of the Mechanical Engineering Department at San Diego State University.

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CLERK'S PAPERS EXCERPT: "CLINICAL RESPONSE OF HUMAN SUBJECTS TO REAR-END AUTOMOBILE COLLISIONS"

Clinical Response of Human Subjects to Rear-End Automobile Collisions

John R. Brault, MS, Jeffrey B. Wheeler, MS, Gunter P. Siegmund, BASc, Elaine J. Brault, MS, PT

ABSTRACT. Brault JR, Wheeler JB, Siegmund GP, Brault EJ. Clinical response of human subjects to rear-end automobile collisions. Arch Phys Med Rehabil 1998;79:72-80.

Objective: Forty-two persons were exposed to controlled low-speed rear-end automobile collisions to assess the relation between both gender and impact severity and the presence, severity, and duration of whiplash-associated disorders (WAD). Individual measures were also assessed for their potential to predict the onset of WAD.

Design: Experimental study subjecting individuals to a speed change of 4km/h and 8km/h and utilizing pretest and posttest physical examinations (immediately after and 24 hours after impact) to quantify subjects' clinical response.

Results: Approximately 29% and 38% of the subjects exposed to the 4km/h and 8km/h speed changes, respectively, experienced WAD symptoms, with cervical symptoms and headaches predominating. Objective clinical deficits consistent with WAD were measured in both men and women subjects at both 4km/h and 8km/h. At 4km/h, the duration of symptoms experienced by women was significantly longer when compared with that in men ($p < .05$). There were no significant differences in the presence and severity of WAD between men and women at 4km/h and 8km/h or in the duration of WAD at 8km/h. There was also no significant difference in the presence, severity, and duration of WAD between 4km/h and 8km/h. No preimpact measures were predictive of WAD.

Conclusion: The empirical findings in this study contribute to establishing a causal relationship between rear-end collisions and clinical signs and symptoms.

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APPROXIMATELY 6.5 million motor vehicle accidents occurred in the United States in 1994, resulting in approximately 3.2 million injuries. Eighteen percent of the accidents involving passenger cars were rear-end impacts that caused injury to 500,000 persons.[1] Rear-end impacts result in a higher frequency of "whiplash" injuries in comparison with other crash configurations.[2-4] According to the Insurance Research Council,[5] the incidence of bodily injury liability claims associated with motor vehicle accidents has been increasing, with sprain/strain injuries accounting for the greatest share of injuries. Frequently, scientific literature uses the term *whiplash* to describe not only a mechanism of injury but also the injury or syndrome associated with the mechanism. According to the recommendations proposed by the Quebec Task Force in 1995, this study uses the term *whiplash-associated disorders* (WAD) to refer to the various clinical manifestations associated with the whiplash injury mechanism, including cervical soft-tissue strain, headache, dizziness, tinnitus, memory loss, temporomandibular joint (TMJ) pain, and others.[6]

Because of the prevalence of this condition in low-speed rear-end automobile collisions, much research has attempted to identify who is at risk for the disorder. Epidemiologic studies[2,4,6-10] have concluded that women sustain WAD more frequently than do men. States and coworkers[2] showed that women have a greater ratio of head mass (represented by head circumference^3) to neck cross-sectional area (represented by neck circumference^2) in comparison with men and proposed that this head-to-neck (H^3/N^2) ratio may explain the increased incidence of WAD. Using mathematical modeling based on anthropometric data, Snyder and colleagues[11] concluded that decreased cervical strength may be responsible for the increased incidence of cervical injuries in women exposed to rear-end collisions.

In the search for predictive factors, researchers have investigated the association between vehicle or crash-related factors and WAD injury severity following rear-end collisions.[12-14] In a descriptive case series study, Radanov and colleagues[14] found no correlation between the patient's perception of the severity of the automobile collision and the duration of pain. Likewise, Olsson and coworkers[12] found no correlation between the duration of neck symptoms and impact severity in rear-end collisions; however, the duration of neck symptoms increased if the estimated horizontal distance between the occupant's head and the vehicle's head restraint exceeded 10cm at the time of the collision. Using five measures of neck strain severity and two measures of impact severity, Ryan and coworkers[13] concluded that initial severity of neck strain is positively correlated with impact severity.

It is presumed within the medical community that WAD involve soft tissues, and that clinical examination often detects only subjective findings. Moreover, because the disorder is nonfatal and usually does not require surgical treatment, no pathologic studies have identified the site or nature of the offending lesions.[15] Current physical models (anthropometric devices and cadavers[16]) and mathematical models[17] typically employed by automotive safety laboratories can be useful to study general impact response. They have not, however, been fully validated against human subject response during low-speed rear-end collisions. Currently, these models are not suitable for human surrogates to study injury response during low-speed, rear-end collisions. For this reason, researchers have turned to human subjects to study the injury mechanism in staged low-speed, rear-end impacts.[18-21] In these studies, human subjects were exposed to rear-end impacts with speed changes ranging from 3.2 to 10.9km/h, with WAD produced at changes in velocity of 6 to 7km/h and above. Speed change is a common measure of collision severity in vehicle occupant studies and is equal to the difference between the preimpact and postimpact velocity of the vehicle. In the situation of a stationary vehicle being rear-ended, the speed change is equal to the postimpact speed of the rear-ended vehicle.

From Biomechanics Research & Consulting, Inc. (Mr. Brault, Mr. Wheeler, Ms. Brault), El Segundo, CA; and MacInnis Engineering Associates, Ltd. (Mr. Siegmund), Richmond, BC, Canada.

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Given the greater frequency of WAD reported in women, a specific goal of this study was to expose an equal number of male and female volunteer human subjects of a specific age group to two controlled low-speed rear-end automotive collisions (change in velocity of 4km/h and 8km/h) to assess the relation of gender and impact severity with the presence, severity, and duration of WAD. A second purpose of this study was to assess the potential for individual parameters to predict the onset of WAD in rear-end automobile collisions. The study was also designed to assess the subjects' eventual clinical outcome after the impacts. It is hoped that our research findings will contribute to establishing the causal relationship between the whiplash injury mechanism and the objective clinical response in rear-end collisions.

METHODS

Subjects

This study was part of a larger project that analyzed occupant kinematics and kinetics in low-speed rear-end automobile collisions through the use of high-speed video, accelerometry, anthropometry, and electromyography (EMG). Forty-two volunteers recruited by local newspaper and university job-line advertisements participated in the study. Men and women were equally represented. Because injury statistics suggest a high incidence in this age group,[6] and in an attempt to select a homogenous population, subjects ranging from 20 to 40 years old were targeted (men, 26.4yrs [SD ± 4.5], women, 27.1yrs [SD ± 4.8]). The mean height of the men was 175cm (SD ± 5); the mean height of the women was 164cm (SD ± 5). Mean body weight was 75kg (SD ± 10) for the men and 62kg (SD ± 9) for the women.

Subjects had no medical conditions or history of soft-tissue disease or related syndrome. Additionally, subjects with a history of neck or back injury or having treatment within the previous 3 years and those with a current insurance claim for neck or back injuries were excluded. Only subjects in the 10th to 90th percentile heights for gender[22] (men, 165 to 185cm; women, 152 to 173cm) and 10th to 90th percentile weights for their height[22] were eligible for the study. In addition, a maximum erect seated height of 95.5cm was permissible to ensure head but no neck contact with the head restraint. This latter criterion was required to allow kinetic analysis of data. Pretest magnetic resonance imaging (MRI) of the head and cervical spine, interpreted by a radiologist, ensured that no subject had a fracture, disk herniation, disk protrusion greater than 2mm, spinal canal stenosis, tumor, or any degenerative findings rated as moderate or greater. These criteria were developed to exclude subjects with MRI findings that may have predisposed them to acute injury. Subjects were asked to refrain from drastically changing their activity level in the week before testing and to not drink alcohol 24 hours before and after testing.

Test Apparatus

Subjects were seated in the right front seat of a stationary 1990 Honda Accord, which was struck on the rear bumper by a 1976 Volvo 240D station wagon. The Volvo was accelerated down a ramp from a predetermined height to impact speeds of 4.86 ± .12km/h and 10.02 ± .06km/h to produce a speed change of 3.95 ± .11km/h and 8.10 ± .11km/h, respectively, for the Honda. Both vehicles were in neutral gear with the engine off. After impact, the Honda rolled forward into gravel located about 3m ahead of the vehicle and decelerated to a stop. Speed change of the target vehicle was measured with an MEA 5th wheel[a] (commercial device for low-speed impact testing) and verified with bumper-mounted load cells.[b]

The choice of 4 and 8km/h speed changes was based on previous testing of human subjects in rear-end collisions which reported no symptoms at a speed change of 4km/h and only minimal WAD of 2 days' duration with a speed change of 8km/h.[18,19]

Human Subject Protection

Protection policies and procedures for the human subjects were in accordance with and approved by the Western Institutional Review Board. Preliminary subject eligibility was determined by an initial telephone interview, after which subjects visited the test facility where the project's principal investigator (GPS) explained the test protocol and procedures to each subject and informed consent was obtained. Subjects also completed a questionnaire assessing their availability, occupation, medical history, and physical activity. The head and neck of each subject was examined using MRI to determine eligibility based on the previously defined radiologic criteria.

Anthropometry

Anthropometric measures were obtained from each subject by a physical therapist (EJB). Head circumference was recorded via metal tape measure at the level of the glabella (most anterior protrusion of forehead) and opisthocranion (most posterior protrusion of back of head) perpendicular to the midsagittal plane. Neck circumference, also recorded via metal tape measure, was obtained at the midpoint of the neck perpendicular to the long axis. These two anthropometric measurements were used to calculate the ratio of head volume to neck cross-sectional area (computed using H^3/N^2).

Pretest Clinical Examination

At least 48 hours before the collision testing, maximum isometric cervical flexion and extension force was measured by a physical therapist with a MicroFET[c] hand-held dynamometer. Maximum isometric cervical flexion was measured with the subject supine on an examination table with the head and neck in a neutral position, C7 placed at the edge of the table so that the head and neck were extended over the edge, arms folded across the chest, and the torso stabilized. The dynamometer was centered on the glabella, and the subject was instructed to maintain maximally the head-neutral position while a downward force was applied to the forehead. Maximum isometric cervical extension was measured with the subject prone, positioned with the sternoclavicular joints at the edge of the table. The head and neck were over the edge of the table in a head-neutral position, hands behind the back, and torso stabilized. The subject was instructed to maintain maximally that head position while resistance was applied to the opisthocranion in a downward direction. A minimum 48-hour delay between isometric cervical testing and impact testing was chosen because the maximum isometric contractions of the cervical muscles potentially could have induced symptoms in these cervical spine muscles that mimic WAD, thus confounding our assessment of symptoms caused by the vehicle impacts. Measurement of maximum isometric cervical flexion and extension was performed to investigate whether these measures predispose individuals to WAD. In addition, the cervical flexion and extension maximal isometric forces were measured 24 hours after both impacts and compared with preimpact forces as an objective clinical measurement of cervical muscle function.

Before each impact, the physical therapist examined the cervical spine of each subject. The purpose of the clinical examinations was to use objective clinical measures to assess the subjects' neuromuscular system, to establish baseline values for

the measurements, to identify objective clinical changes in these measurements attributable to our intervention, and to identify any abnormal neuromuscular condition present either preimpact or postimpact that might require medical attention, which helped to ensure human subject protection. Components of a basic orthopedic examination of the cervical spine were performed, including measurement of cervical and TMJ active range of motion (ROM), assessment of the C4-T1 myotomes and dermatomes, deep tendon reflexes, and point tenderness.[23]

Cervical flexion, extension, right and left lateral flexion, right and left rotation, protraction, and retraction active ROM were measured with the cervical range of motion device (CROM),[d] a plastic instrument that rests on the bridge of the nose and ears and that is secured at the back of the head with Velcro. The CROM had three orthogonal goniometers to measure sagittal, frontal, and coronal plane motions and a bubble level and ruler for measuring protraction/retraction. Previous studies have demonstrated its intratester reliability.[24,25] Subjects were seated in a straight-back chair with the feet flat and hands on the lap, facing and focusing forward, with a lumbar support to maintain the lumbar lordosis. The CROM was applied to the head and the subjects given verbal and visual instructions on the performance of each cervical movement. Subjects were allowed one to two practice trials for each motion followed by the experimental trial, which was documented.

Range of motion of the TMJ was measured with the subject in a seated position. The examiner instructed the subject in maximum jaw opening, left excursion, and right excursion, and measurements were made in millimeters with a ruler.

Assessment of the C4-T1 dermatomes was done to evaluate sensation of cervical nerve roots emanating to the upper extremities. This was accomplished by performing a sharp/dull test with a pinwheel. With the subject seated, eyes closed, and the hands supinated on the lap, the right and left C4-T1 dermatomes were given either a sharp or dull sensation via the alternate ends of a pinwheel, and the subject was asked to answer appropriately.

The C4-T1 myotomal strength was assessed bilaterally using manual muscle testing (MMT) with the MicroFET dynamometer following published guidelines.[26] The C4 myotome, represented by the upper trapezius muscle, was tested by measuring the subjects' maximum isometric force output at 90° of arm elevation in the scapular plane. Arm elevation has been shown to recruit maximally the upper trapezius.[27] The C5/6 myotomes, represented by the elbow flexors, were tested by measuring maximum isometric elbow flexion. The C7 myotome, represented by the triceps muscle, was assessed by measuring subjects' maximum isometric elbow extension. The C8/T1 myotomes, represented by the first dorsal interosseous muscle, were assessed by measuring maximum isometric abduction of the index finger. All of the MMT with the hand-held dynamometer was performed with the subject seated on an examination table, except the test for the C7 myotome, which was performed with the subject supine on a mat to give the examiner a mechanical advantage.

Deep tendon reflexes of the bilateral brachioradialis and triceps were measured with a reflex hammer and graded on the established 1+ to 4+ scale.[28] Point tenderness of specific regions of the body was assessed by the examiner through palpation of the soft tissue overlying the occiput and suboccipital area, as well as the bilateral sternocleidomastoid (SCM), scalene, cervical paraspinal, upper trapezius, and middle trapezius muscles and TMJ. Subjects were seated in a chair and instructed by the examiner to relax as the specific regions were palpated. The examiner asked the subjects to report any tenderness elicited by the palpation, which was graded on a 1+ to 4+ scale (1+ = slight, 2+ = minimal, 3+ = moderate, 4+ = severe).

Electromyography

Surface EMG electrodes were applied to the subject's bilateral SCM and cervical paraspinal muscles by an investigator experienced in the use of surface EMG (JRB). The primary purpose of the EMG sampling was to investigate cervical muscle response during the impact (EMG results will be presented in another report). A secondary purpose of the EMG was to ensure that preimpact cervical muscle response, which could alter the occupant kinematics during impact, did not increase above seated resting levels. Four-millimeter IVM Ag-AgCl reusable disk electrodes[e] were taped to the skin overlying the clavicular head of the bilateral SCM muscle in a bipolar configuration using the technique of Zipp.[29] Eight-millimeter IVM Ag-AgCl reusable disk electrodes[e] were taped to the skin overlying the posterior cervical spine in a bipolar configuration 1cm lateral to the C4 and C6 spinous processes bilaterally with a 4cm interelectrode distance. To reduce impedance at the electrode sites, the skin was shaved, lightly abraded with sandpaper, wiped with 50/50 alcohol/distilled water, and coated with electrode gel.[30] The electrode leads were connected to a transmitter.[f] The EMG signals were telemetered to a receiver, converted from analog to digital by an A/D board,[g] and then analyzed by LABVIEW[g] software. Proper placement of the electrodes was confirmed by visual inspection of the muscle interference pattern during manual muscle testing. EMG data were sampled at a frequency of 1,000Hz and band-pass–filtered with a second-order Butterworth filter with high- and low-pass filters of 40 to 500Hz, respectively.

Collision 1

Following the initial examination, subjects were exposed to their first test collision. To control for series effect, the order of impact severity was randomized by flipping a coin. Subjects were not told which speed change they would experience.

In the test vehicle (Honda), the fore/aft seat position, seat back angle, and head restraint height were kept constant for all subjects. Subjects were instructed to sit with their backs against the seat back, face forward with the head horizontal, feet on the floor, hands in the lap, and to otherwise assume a normal seated position. The lap/shoulder belt was worn by all subjects, and the subjects were instructed not to rest their heads on the head restraint. The horizontal distance between the opisthocranion and head restraint was measured with a FaroArm 3D digitizer[h] and confirmed from the digitized video frame corresponding to impact.

Ryan and coworkers[31] found that automobile occupants who were unaware of the impending collision were 15 times more likely to have persisting WAD 6 months postimpact; therefore, we attempted to control for this variable by minimizing subject expectation of the impending collision. First, subjects did not see the striking vehicle (Volvo) on the ramp prior to their test collisions and therefore derived no preconception of impact severity from its height on the ramp. Second, a curtain was placed between the Honda and Volvo to eliminate visual cues of the impending collision, and no test personnel were visible to the subjects in the minutes immediately preceding the test collision. Third, subjects wore foam ear plugs, and loud music was played before and during the test collisions to eliminate aural cues. Fourth, the subjects' seated position was monitored before impact via closed-circuit video from a camera mounted to the Honda's driver's side A-pillar to ensure no change of position prior to impact. Lastly, the subjects' EMG output from the bilateral SCM and cervical paraspinal muscles was visually monitored for a minimum of 1 minute before impact to ensure

no increase in muscular contraction above that present in the normal seated position.

After the first test collision, subjects were told the severity of the rear-end collision to minimize apprehension in those subjects first exposed to the 8km/h speed change and who might elect to withdraw if they presumed the first collision was a 4km/h speed change.

Posttest Clinical Examination

A second clinical examination was performed within 30 minutes after the first test collision. All of the tests performed in the preimpact clinical examination were repeated by the same examiner, and subjects were asked whether they were experiencing any symptoms not present before the impact. Because the primary symptom of WAD is often the subjective expression of pain,[6] the objective of the examination was to quantify the subjects' symptoms to treat the data statistically. Therefore, if subjects reported symptoms following a test collision, the postimpact examination included the McGill Pain Questionnaire, which was designed to provide quantitative measures of clinical pain that can be treated statistically.[32] Through the use of word descriptors chosen by subjects to best represent their pain, the quality of pain was documented. The intensity of the pain was assessed with the questionnaire's Present Pain Intensity scale (PPI), which is a combination numerical and descriptive measure of pain. The PPI was recorded as a number from 1 to 5, with each number associated with the following words: 1 = mild, 2 = discomforting, 3 = distressing, 4 = horrible, and 5 = excruciating. Subjects recorded the location of pain on the questionnaire's body diagram, which portrayed an anterior and posterior outline of a body of appropriate gender. Subjects were instructed to shade the area on the body diagram corresponding to the location of their pain. Because the subjects could not be observed continuously after the test collisions, the assessment of the subjects' pain experience included the McGill Home Recording Card.[32] After each test collision, the subjects were given a card on which they recorded their symptoms and level of pain (PPI) at four different times throughout the day (morning, noon, dinner, bedtime) between the time of their first postimpact examination and the 24-hour follow-up examination. The Home Card provided a method of documentation away from the test site and allowed continuous tracking of symptoms and injury severity.

24-Hour Posttest Examination

Subjects returned to the test facility the day after the test collision (approximately 24 hours posttest) for a third clinical examination during which all of the previous cervical spine testing was repeated. If symptoms were reported, the McGill Pain Questionnaire was completed. In addition, subjects returned the completed McGill Home Recording Card and were observed until their symptoms and clinical deficits subsided.

Collision 2

Subjects returned for the second test collision a minimum of 7 days after the first test collision or 7 days after resolution of their symptoms attributed to the first test collision. The protocol for the second test collision was identical to that in the first, except that subjects were aware of the severity of the second test collision beforehand.

Statistical Analysis

The null hypothesis was that there would be no difference in the presence, severity, and duration of WAD between genders and impact severity. Comparisons of the presence, severity, and duration of WAD between genders were performed using Fisher's exact test,[33] Wilcoxon rank sums test,[34] and log-rank test,[35] respectively. The tests were performed on the 4km/h and 8km/h data separately. Comparison of the presence of WAD between impact speeds was carried out using McNemar's chi-squared test.[36] Comparisons of the severity and duration of WAD between speed changes were carried out using the Wilcoxon sign-rank test.[34] Comparisons of severity and duration were performed using only the data of subjects with WAD.

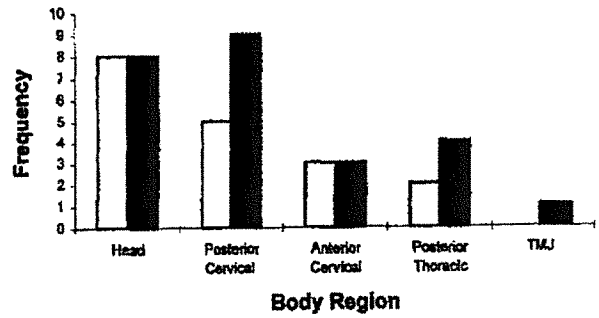

Fig 1. Symptom distribution for the men and women subjects combined: □, 4km/h; ■, 8km/h.

Logistic regression[37] (modeled using a log linear model) was used to determine whether the measured variables of H^3/N^2, pre-impact cervical flexion and extension isometric force, preimpact cervical ROM, and horizontal distance from head to head restraint could be used to predict the onset of WAD. The likelihood ratio chi-squared was used to test for significant effects of the various measurements. A separate regression analysis was carried out for the 4km/h and 8km/h trials.

A repeated measures analysis of variance (ANOVA)[38] was used to test the effect of the presence of symptoms and the time of measurement (preimpact, postimpact, 24-hours postimpact) on ROM and cervical MMT values. This analysis was conducted on the cervical ROM values of flexion and extension, right and left lateral flexion, right and left rotation, and protraction and retraction to test for statistical significance. Again, the analyses were performed separately for the 4km/h and 8km/h trials. A p value of .05 level of significance was used for all statistical comparisons.

Power was calculated for the duration of symptoms between the 4km/h and 8km/h collisions, assuming the observed values in this study are exponentially distributed. Under this assumption, the power of the study to detect a significant difference in duration of symptoms is 90%.

RESULTS

Because no women at the 4km/h and only one woman at the 8km/h impact severity experienced symptoms at the time of either of the postimpact clinical examinations, the data from the component of the McGill Pain Questionnaire using word descriptors of pain were not utilized for statistical purposes. Given that nearly all of the symptoms experienced by the subjects occurred while the subjects were away from the test site, data obtained from the PPI index on the McGill Home Card and Pain Questionnaire were treated statistically.

Figure 1 shows the combined male and female distribution of WAD by body region for the 4km/h and 8km/h impacts. Headaches and posterior neck symptoms were the predominating complaints. After the 4km/h impact, the examiner identified three subjects with 1+ to 3+ tenderness with a duration of up to 48 hours. The 8km/h impact produced eight subjects with

Table 1: Comparison Between Genders at 4km/h

	Men	Women	p Value
Presence*	5 (23.8)	7 (33.3)	.73†
Severity‡	1.2 (1.1)	1.3 (1.4)	1.00§
Duration‖	2 (1-8)	12 (1-48)	.03¶

* Count (percent).
† Fisher's exact test.
‡ Mean (SD) PPI of subjects with symptoms.
§ Wilcoxon rank sums test.
‖ Median duration (range) in hours of subjects with symptoms.
¶ Log-rank test.

1+ to 2+ tenderness lasting up to 48 hours. In both impacts, the areas of tenderness included the bilateral cervical paraspinal, SCM, scalene, upper trapezius, middle trapezius, and occiput areas. No significant differences were noted postimpact for the reflex, sensory, or upper extremity MMT.

At 4km/h, there were no significant differences in either the presence of WAD between men and women or in the mean severity (PPI) of symptoms between men and women (table 1). A significant difference, however, did exist in the median duration of symptoms between men (2 hours) and women (12 hours). An unpaired *t* test between male and female H^3/N^2 ratio revealed a statistically significant higher ratio in women in our sample population. At 8km/h, no significant differences existed in the presence, severity, or duration of WAD between men and women (table 2). There were no differences in the presence, severity, or duration of WAD between the 4km/h and 8km/h collisions when both men and women were combined (table 3).

The logistic regression analyses failed to identify a pre-impact measurement variable predictive of the presence of WAD at either 4 or 8km/h (tables 4 and 5, respectively).

Analysis of the effect of the 4km/h impact severity on ROM measurements over time revealed that, at both postimpact examinations, subjects with and without symptoms had a significant decrease in cervical flexion, extension, retraction, and right lateral flexion, with left lateral flexion ROM approaching statistical significance (table 6). In addition, an analysis of subjects with and without symptoms revealed that the subjects with symptoms had significantly reduced left and right lateral flexion ROM values preimpact and at both postimpact examinations (table 6). At the 8km/h impact severity, ROM values were significantly reduced immediately postimpact for cervical flexion, right and left lateral flexion, right rotation, and retraction for both groups with and without symptoms (table 7). An analysis of the effect of the interaction between the presence of symptoms and the time of examination on ROM values at both the 4km/h and 8km/h impact severity revealed no significant differences (tables 6 and 7).

Significantly higher cervical flexion MMT values were measured postimpact in subjects with and without symptoms at both 4 and 8km/h, but there was no difference in cervical MMT values when the groups were analyzed by the presence of symptoms.

Three subjects (one male and two female) having experienced only the 4km/h trial were lost to observation for the 8km/h trial. No increase in cervical EMG activity was observed immediately preimpact in comparison with the resting levels, suggesting not only adequate control of the potential confounding variable of a preimpact increase in cervical muscle resistance but also that the subjects were unprepared for the collisions.

Table 2: Comparison Between Genders at 8km/h

	Men	Women	p Value
Presence*	8 (38.1)	7 (36.8)	1.00†
Severity‡	0.8 (0.8)	0.9 (0.4)	1.00§
Duration‖	8.8 (0.3-111)	24 (0.5-24)	0.91¶

* Count (percent).
† Fisher's exact test.
‡ Mean (SD) PPI of subjects with symptoms.
§ Wilcoxon rank sums test.
‖ Median duration (range) in hours of subjects with symptoms.
¶ Log-rank test.

Table 3: Comparison of 4km/h With 8km/h

	4km/h	8km/h	p Value
Presence*	12 (28.6)	15 (38.5)	.17†
Severity‡	1.3 (1.2)	0.8 (0.5)	.99§
Duration‖	5.8 (1-48)	24 (0.3-111)	.14§

* Count (percent).
† McNemar χ^2 test.
‡ Mean (SD) PPI of subjects with symptoms.
§ Wilcoxon sign-rank test.
‖ Median duration (range) in hours of subjects with symptoms.

DISCUSSION

The results of this study reveal objective clinical deficits consistent with WAD at both 4km/h and 8km/h speed changes in both men and women. The distribution of symptoms by body region agrees with clinical reviews and descriptive case series of WAD,[6,14,15] with cervical symptoms and headaches predominating. A potential confounding variable which may have influenced the number of complaints of headache was the tightness of the head gear worn to measure head acceleration. The WAD experienced by the subjects is consistent with the Quebec Task Force clinical classification of grade II, which is defined as neck complaints and musculoskeletal signs such as decreased ROM and point tenderness.[6]

The lack of a significant difference in the presence of WAD between male and female subjects is inconsistent with many previous reports documenting an increased incidence of WAD in females.[2,4,6-10] These previous epidemiologic studies, utilizing clinical reviews of patients, emergency room medical records, insurance claims, and traffic collision reports, have incorporated a wide range of impact severity. Perhaps the speed changes in our study were too low, narrow, or both to delineate a difference between genders. In a recent questionnaire-based study of 202 victims of rear-end collisions,[39] no significant difference was evident in the prevalence of chronic neck pain or headache between males and females. That study, however, investigated

Table 4: Logistic Regression of Factors Used to Predict for the Presence of WAD Following a 4km/h Collision

	Subject Without Symptoms	Subject With Symptoms	
	Mean (SD)	Mean (SD)	p Value*
Head to head rest (mm)	44.0 (20.1)	49.1 (15.4)	.44
H^3/N^2 (cm)	152.7 (22.70)	156.5 (24.27)	.63
Cervical MMT (Newtons)†			
Flexion	104.2 (46.6)	81.6 (43.2)	.14
Extension	217.9 (62.8)	193.4 (67.3)	.26
Cervical ROM (°)‡			
Flexion	61.0 (6.7)	58.0 (8.4)	.23
Extension	69.9 (7.6)	70.5 (8.8)	.81
R lateral flexion	45.3 (6.6)	40.7 (7.4)	.06
L lateral flexion	46.5 (6.2)	43.3 (5.3)	.13
R rotation	68.9 (7.6)	67.7 (8.4)	.63
L rotation	68.7 (6.7)	66.0 (4.7)	.21
Retraction	3.3 (1.1)	3.1 (0.9)	.68
Protraction	3.7 (0.9)	3.8 (1.4)	.78

* Likelihood ratio χ^2 from logistic regression.
† Pretest cervical manual muscle test.
‡ Pretest cervical range of motion.

Table 5: Logistic Regression of Factors Used to Predict for the Presence of WAD Following a 8km/h Collision

	Subject Without Symptoms	Subject With Symptoms	
	Mean (SD)	Mean (SD)	p Value*
Distance to head rest (mm)	41.7 (18.3)	40.9 (18.5)	.90
H^3/N^2 (cm)	153.3 (21.4)	154.7 (24.9)	.84
Cervical MMT (Newtons)†			
Flexion	100.3 (47.2)	97.6 (44.1)	.88
Extension	217.5 (65.1)	211.2 (56.6)	.98
Cervical ROM (°)‡			
Flexion	61.5 (7.4)	59.3 (6.4)	.35
Extension	69.8 (8.6)	71.9 (8.2)	.44
R lateral flexion	45.2 (7.4)	41.7 (5.7)	.14
L lateral flexion	46.6 (7.3)	44.5 (6.7)	.38
R rotation	70.8 (8.0)	67.7 (7.3)	.23
L rotation	68.1 (6.4)	66.1 (5.9)	.34
Retraction	3.0 (1.0)	3.2 (0.9)	.24
Protraction	3.8 (1.3)	4.0 (1.2)	.70

* Likelihood ratio χ^2 from logistic regression.
† Pretest cervical manual muscle test.
‡ Pretest cervical range of motion.

chronic symptoms and not initial symptoms, precluding any direct comparison to our results.

The presence of WAD at the 4km/h speed change conflicts with all previously published accounts of low-speed rear-end automobile testing involving human subjects.[18-21] The different results found in our study may be explained by the small sample size, predominately male population, variable age range, variable speed changes,[18,19,21] multiple impact exposures, and failure to perform a complete pre-impact and postimpact objective clinical examination in the previous studies. In addition, in all cases, the subjects were either the investigators or professionally associated with the investigators, which could potentially introduce researcher bias. Although these studies provide the groundwork for understanding occupant response to low-speed rear-end impacts, the results are difficult to apply to any subset of the general population given these limitations.

Although the median duration of symptoms at 4km/h was significantly longer for women in comparison with men (12 hours versus 2 hours), no clinical significance can be deduced from this information given the fact that few persons would be expected to seek medical treatment beyond an initial consultation for symptoms of such short duration.

The failure to demonstrate a difference between the 4km/h and 8km/h trials with respect to the presence, intensity, and duration of WAD is noteworthy, because one might expect greater injury as speed change increases. Perhaps the range of speed change was too low and narrow to elicit such a biologic effect in our subjects. Increasing the range of impacts is not a viable option, however, given the potential harm associated with exposing human subjects to greater impact severity. Previous studies have failed to reveal a dose-response for duration[12] and severity[14] of WAD following rear-end collisions. However, the studies included higher speed collisions than used in our study, and the measure of accident severity used by Radanov and coworkers,[14] the patient's perception, could be considered unreliable.

The inability of the H^3/N^2, cervical flexion and extension isometric force, and horizontal distance from head to head restraint to predict the presence of WAD failed to support several theories[2,11,12] which purport that preimpact parameters may influence an individual's susceptibility to WAD. Differing study

Table 6: ANOVA for Range of Motion Values at 4km/h

	Time Period	No Symptoms*	Symptoms*	Symptom Effect†	Time Effect†		Symptom × Time Effect†
Flexion (°)	Pretest	61.0 (6.7)	58.0 (8.4)	.21	.004‡	Posttest < pretest	.51
	Posttest	59.9 (7.0)	56.2 (9.0)			Posttest < post-24hrs	
	Post-24hrs	61.3 (6.6)	59.0 (8.8)				
Extension (°)	Pretest	69.9 (7.6)	70.5 (8.8)	.90	.02	Posttest < pretest	.88
	Posttest	68.6 (7.8)	68.7 (7.3)			Posttest < post-24hrs	
	Post-24hrs	70.0 (7.3)	70.3 (10.3)				
R lateral flexion (°)	Pretest	45.3 (6.6)	40.7 (7.6)	.04‡	.03‡	Posttest < pretest	.57
	Posttest	44.4 (6.4)	40.0 (6.8)			Posttest < post-24hrs	
	Post-24hrs	45.9 (6.6)	40.7 (8.5)				
L lateral flexion (°)	Pretest	46.5 (6.2)	43.3 (5.3)	.04‡	.06		.28
	Posttest	46.5 (5.7)	41.7 (6.8)				
	Post-24hrs	47.7 (6.3)	43.2 (7.6)				
R rotation (°)	Pretest	68.9 (7.6)	67.7 (8.4)	.38	.56		.16
	Posttest	68.9 (7.0)	67.3 (6.6)				
	Post-24hrs	70.5 (7.1)	67.0 (6.6)				
L rotation (°)	Pretest	68.7 (6.7)	66.0 (4.7)	.14	.16		.67
	Posttest	68.1 (6.6)	64.7 (3.5)				
	Post-24hrs	68.4 (6.6)	65.7 (4.3)				
Protraction (cm)	Pretest	3.7 (0.9)	3.8 (1.4)	.98	.28		.67
	Posttest	3.8 (0.9)	3.9 (1.4)				
	Post-24hrs	4.0 (0.9)	3.8 (1.6)				
Retraction (cm)	Pretest	3.3 (1.1)	3.1 (1.0)	.78	.0002‡	Posttest < pretest	.19
	Posttest	3.0 (1.0)	2.8 (0.8)			Posttest < post-24hrs	
	Post-24hrs	3.4 (0.8)	3.6 (0.8)				
TMJ opening (mm)	Pretest	47.5 (7.2)	45.3 (4.3)	.34	.17		.06
	Posttest	47.5 (7.5)	45.7 (4.5)				
	Post-24hrs	47.6 (7.2)	45.2 (3.9)				
TMJ L excursion (mm)	Pretest	3.5 (1.4)	4.4 (1.6)	.03‡	.28		.46
	Posttest	3.4 (1.3)	4.5 (1.6)				
	Post-24hrs	3.3 (1.3)	4.4 (1.6)				
TMJ R excursion (mm)	Pretest	3.6 (1.7)	4.1 (1.0)	.41	.53		.73
	Posttest	3.6 (1.8)	4.0 (1.0)				
	Post-24hrs	3.6 (1.7)	4.0 (1.0)				

* Mean (SD) ROM.
† p value.
‡ $p < .05$.

Table 7: ANOVA for Range of Motion Values at 8km/h

	Time Period	No Symptoms*	Symptoms*	Symptom Effect†	Time Effect†		Symptom × Time Effect†
Flexion (°)	Pretest	61.6 (7.4)	59.3 (6.4)	.23	.04‡	Posttest < pretest	.62
	Posttest	60.7 (7.4)	57.3 (6.0)			Posttest < post-24hrs	
	Post-24hrs	61.8 (7.3)	59.5 (6.2)				
Extension (°)	Pretest	69.6 (8.6)	71.9 (8.2)	.47	.03		.92
	Posttest	69.1 (9.0)	71.1 (7.3)				
	Post-24hrs	69.9 (7.8)	71.6 (8.7)				
R lateral flexion (°)	Pretest	45.2 (7.4)	41.7 (5.7)	.07	.01‡	Posttest < post-24hrs	.36
	Posttest	45.2 (5.8)	40.4 (6.2)				
	Post-24hrs	46.2 (6.3)	42.9 (7.4)				
L lateral flexion (°)	Pretest	46.6 (7.3)	44.5 (6.7)	.34	.0001‡	Pretest < post-24hrs	.39
	Posttest	45.6 (6.0)	42.8 (6.2)			Posttest < pretest	
	Post-24hrs	48.0 (5.7)	46.9 (7.5)			Posttest < post-24hrs	
R rotation (°)	Pretest	70.8 (8.1)	67.7 (7.3)	.23	.04‡	Posttest < post-24hrs	.68
	Posttest	69.6 (7.7)	67.1 (6.6)				
	Post-24hrs	71.4 (8.6)	68.0 (7.1)				
L rotation (°)	Pretest	68.1 (6.4)	66.1 (5.9)	.24	.65		.29
	Posttest	68.4 (6.1)	65.3 (5.1)				
	Post-24hrs	68.1 (5.9)	66.7 (4.5)				
Protraction (cm)	Pretest	3.8 (1.3)	4.0 (1.2)	.38	.6		.57
	Posttest	3.8 (1.2)	4.2 (1.1)				
	Post-24hrs	3.8 (0.9)	4.0 (1.0)				
Retraction (cm)	Pretest	2.9 (0.9)	3.2 (0.8)	.32	.0001‡	Pretest < post-24hrs	.71
	Posttest	2.9 (0.8)	3.0 (0.9)			Posttest < post-24hrs	
	Post-24hrs	3.4 (0.8)	3.7 (1.2)				
TMJ opening (mm)	Pretest	47.3 (5.4)	46.9 (7.8)	.85	.95		.48
	Posttest	47.2 (5.5)	46.9 (7.6)				
	Post-24hrs	47.4 (5.6)	46.8 (7.8)				
TMJ L excursion (mm)	Pretest	3.6 (1.4)	4.3 (1.8)	.16	.76		.49
	Posttest	3.6 (1.3)	4.2 (1.8)				
	Post-24hrs	3.5 (1.1)	4.3 (1.8)				
TMJ R excursion (mm)	Pretest	3.6 (1.9)	3.9 (1.7)	.69	.66		.07
	Posttest	3.7 (1.8)	3.7 (1.7)				
	Post-24hrs	3.6 (1.9)	3.9 (1.8)				

* Mean (SD) ROM.
† p value.
‡ $p < .05$.

designs may account for this discrepancy. In an epidemiologic survey of police accident reports, States and colleagues[2] hypothesized that a higher female H^3/N^2 ratio suggested a larger head relative to neck size and thus an increased susceptibility to WAD in rear-end automobile collisions; however, the investigators relied on military anthropometric data and did not directly measure the H^3/N^2 ratio of the subjects in their population. Our results could not be compared with those of Olsson and coworkers,[12] who reported an increased duration of neck symptoms in rear-end collisions when the head to head restraint distance exceeded 10cm, because our data did not include any head to head restraint distances greater than 8cm. Regarding the postulate by Snyder and coworkers[11] that decreased cervical strength is responsible for the greater incidence of WAD in women in rear-end collisions, no comparison can be made with our study as Snyder's group examined only neck characteristics (anthropometry, ROM, strength, and muscle reflex times) and did not expose the subjects to rear-end automobile collisions.

In our attempt to assess the outcome of clinical measurements in the subjects over time, we discovered an increase in cervical flexion isometric force postimpact, which we attributed to a learning effect resulting from the unusual nature of the activity.

Of most importance clinically was the statistically significant decrease in cervical ROM immediately following both 4km/h and 8km/h trials, which suggests that the rear-end collisions caused measurable clinical deficits. Surprisingly, these deficits occurred in subjects with and without symptoms. The presence of cervical ROM deficits after a rear-end automobile collision without concomitant symptoms may be a function of an individual's tolerance for, or perception of, pain. Although statistically significant, the magnitude of these ROM deficits was sufficiently small to be of limited clinical value. The reduced ROM measured immediately following the impacts fell within a normal range of values,[41] and, without specific knowledge of a patient's preimpact ROM values, a health care professional would not likely be capable of detecting these deficits. The CROM device was capable of measuring sagittal plane rotation, lateral flexion, and coronal plane rotation to the nearest 2°, whereas retraction and protraction could be measured to the nearest 0.5cm. Based on the standardized protocol for measurement performed by a single examiner for all subjects and the small standard deviations in ROM data, we are confident that the observed ROM deficits are real and not an artifact of the measuring device or examiner reading error.

Most interesting from a clinical perspective was the fact that the cervical ROM deficits were characteristically similar to the subjective reports of symptoms in that both were transient. In most cases, the ROM deficits were present immediately postimpact, but by the 24-hour postimpact physical examination, cervical ROM had returned to normal, consistent with most subjects' short duration of symptoms. These results are consistent with a study by Ryan and coworkers,[31] who found a strong correlation between objective measures of neck strain and the subject's own rating of neck symptoms.

The degree to which theories about causation of a disease are supported by empirical evidence is termed *construct validity*.[40] The clinical findings of this experimental study contribute to the construct validity of the whiplash injury mechanism and establish the causal relationship between the rear-end collisions and the clinical signs and symptoms. The highly variable clinical findings observed among the subjects in our study also attests to the nonspecific effect of the whiplash injury mechanism (low descriptive validity) previously reported in the literature.[40]

Our sample was one of convenience and consisted of volunteers who were selected to control for variables such as age, previous medical history, and pre-existing spine pathology. Therefore, our study population does not entirely reflect the total population exposed to rear-end automobile collisions. Furthermore, the controlled conditions under which these automobile collisions were conducted (normal seated posture, aligned impacts, head restraint available) are not representative of all rear-end collisions in a noninvestigative setting producing WAD. Additionally, we did nothing to control for other variables such as subjects' psychologic profile[42] or culture,[43] which previously have been reported to influence the outcome following the whiplash injury mechanism. Therefore, meaningful prognoses of WAD in all rear-end collisions cannot be derived from the data, because the accuracy of a given outcome is dependent on many variables.

CONCLUSIONS

Approximately 29% and 38% of individuals exposed to rear-end impacts at 4km/h and 8km/h speed changes, respectively, experienced WAD symptoms of minimal severity and short duration, with cervical symptoms and headaches predominating. The presence, severity, and duration of WAD were similar in men and women exposed to rear-end automobile collisions with speed changes of 4km/h and 8km/h. In addition, the study failed to identify useful predictive factors predisposing individuals to the development of WAD from rear-end automobile collisions. The empirical evidence gained from this experimental study contributes to the construct validity by establishing a causal relationship between rear-end collisions and clinical signs and symptoms.

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The undersigned declares under penalty of perjury under the laws of the State of Washington that on November 3, 2017, I arranged for filing and service of the Plaintiff-Petitioner Gilmore's Supplemental Brief by e-filing and e-service through the Court's web portal, upon all counsel of record herein.

Dated this 3rd day of November, at Burien, WA.

David S. Heller

HELLER LAW FIRM PLLC

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