

Bertil Aldman Memorial Lecture

Soft Tissue Neck Injuries and Other Important Things

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I am honoured, and a little nervous, to be delivering the Bertil Aldman Memorial Lecture to open this year's IRCOB Conference. I am used to presenting technical work in forums like this conference, but from my perspective the Bertil Aldman Memorial Lecture is about more than just technical work: it is an opportunity to present technical work within a broader context shaped and colored by one's personal views, past experiences, and perhaps even one's passions. For me as a listener to prior lectures, it is the inclusion of these personal views, experiences and passions that makes them interesting. For me as a presenter, however, expressing personal views, highlighting personal experiences, and conveying passions are not things I'm used to doing at the podium—hence my nervousness.

My invitation from the IRCOB Council to deliver this lecture suggested that I focus on “soft-tissue neck injury”. I have not asked, but I suspect they chose those words carefully. Soft-tissue neck injury goes by many different names—railway spine, neck sprain/strain, acceleration-deceleration injury, cervical spine distortion injury, hyperextension injury, whiplash injury, and whiplash-associated disorders—but I will refer to it simply as “whiplash injury”. I recognize that the name “whiplash injury” has baggage, something I'll address shortly, but it has the simple virtue of being direct: everyone knows exactly what I am talking about when I say “whiplash injury”.

As I prepared this lecture, the first thing that occurred to me was that the world does not need another review paper about whiplash injury. There are 512 review articles about whiplash injury indexed on PubMed. In the first six months of this year, there are already 8 review articles about whiplash injury. I have co-authored 3 of these 512 review articles and read only a fraction of the other 509 review articles, and I did not see much point in adding another one to the heap of potentially unread articles in the scientific literature.

So instead of writing a review about whiplash injury, I have instead compiled a few thoughts about whiplash injury. Some of these thoughts are based on data, some are based on experience, and some are pure opinion. Nevertheless, I will share them with you in the hope that they might inspire something in you when mingled with your own thoughts and ideas. Since the most important people in this room are the students and young researchers, I will also share a few insights I've gained over the years. They are not earth-shattering insights or even complete insights; but rather small things that I learned along the way that for me, at least, were important.

The Stigma of Whiplash Injury

But let's start where many conversations about whiplash injury begin (or end). To some, the words “whiplash injury” remain an oxymoron. Less so today than 25 years ago when I started studying whiplash injury, but I still regularly encounter prejudice against whiplash injury. The prejudice exists in many places: in the auto industry, in the insurance industry, in the medical industry and in society in general. I encountered it during the questions asked following my first conference presentation of whiplash injury biomechanics in 1997. And I still saw it during another researcher's presentation at the same conference a couple of years ago. The underlying premise

of this prejudice, like many prejudices, slips easily into the brain. It goes something like this: “How can someone be hurt with so little vehicle damage?” Or: “I’ve been in a more severe crash and I wasn’t injured.” Or more bluntly: “They’re just malingering to get insurance money.” Or most cynically: “Whiplash is an injury fabricated by plaintiff lawyers.”

The real problem, of course, is ignorance. We do not understand whiplash injury nearly as well as we should. Biomechanically, we partially understand a mechanism involving the facet capsular ligaments that explains why some patients have chronic pain. Other mechanisms have been hypothesized and some have been tested, but with no comprehensive conclusions within reach (Siegmund et al., 2009). Even without understanding the mechanisms of whiplash injury completely, we have devised seats that mitigate whiplash injury (Sekizuka, 1998; Wiklund et al., 1998; Jakobsson et al., 2000), although they are not nearly as effective as we would like them to be (Farmer et al., 2008; Trempel et al., 2016). Clinically, we have no objective diagnostic tests or effective treatments except for one: invasive anaesthetic blocks and ablation of nerves innervating the facet capsule (Lord et al., 1996; Barnsley et al., 2005), which from my understanding is unpleasant and is used in only a fraction of chronic whiplash patients. Our ignorance makes it difficult to think objectively about whiplash injury, and thus our subjective thoughts fill the gaps. It would be naïve to think that no one exploits this ignorance about whiplash injury—I think it’s in the nature of some human beings to exploit ignorance wherever it exists—but I also don’t believe that a worldwide conspiracy exists wherein regular people fabricate the same constellation of symptoms for personal gain.

I have not experienced a rear-end collision outside the laboratory, but two of my family members have. My father, a stoic man if ever there was one, experienced a rear-end impact in his pickup when I was an adolescent. He suffered a whiplash injury that took months to resolve. He did not complain much following later heart and esophageal surgeries or during the cancer that ultimately took him, but I saw him suffer and complain about his whiplash injury. Similarly, my in-laws experienced a rear-end crash about 20 years ago. My mother-in-law, who was the front passenger, experienced a whiplash injury whereas my father-in-law, who was the driver, did not. Although she does not complain about it, her neck injury has still not resolved. Both my father and my mother-in-law received small insurance settlements for their time off work. Neither one of them malingered and neither one of them thought the money was worth the experience.

Their experiences, one well before I was researching whiplash injury and the other early in my research career, helped shape my way of thinking about whiplash injury. While obviously not a scientific sample, they are for me clear evidence that whiplash injury is real. It is not, in my view, an injury fabricated by plaintiff lawyers. And given no injury in my father-in-law and 15+ years of chronic pain in my mother-in-law, their crash also showed me that whiplash injury does not affect everyone equally. Aside from differences in anthropometry and posture, we all have different injury thresholds. Based on the tolerance data we as a community have generated for almost all other injuries, we can also expect wide variability in the tolerance threshold for whiplash injury across the population.

In the course of my research into whiplash injury, I have experienced hundreds of low-speed rear-end impacts—mostly of very low severity—and I have not experienced anything more than mild transient symptoms. My lack of chronic symptoms does not surprise me. For one, I’ve been seated upright with good head restraint geometry for all of my exposures at or above a speed change of 4 km/h. But more importantly, I understand that I am one individual and that I am not a surrogate for the general population, especially not for the more vulnerable end of the general population. My whiplash exposures and symptoms—or lack of symptoms—cannot be mapped onto others who are not me.

A Forensic Perspective

I did not become an engineer to fix the whiplash problem. I’m not even sure why I became an engineer, other than I enjoyed math and physics, and at the time engineering seemed to me marginally more useful than either pure math or pure physics. I graduated into the summer of Expo 86 in Vancouver and successfully used it to

distract myself from finding a job until I ran out of money and my roommate had to pay my rent. One of two opportunities posted at the university job office on the day I attended was for a consulting firm that reconstructed car crashes. I did not know engineers did such work. I liked puzzles and so I applied for and got the job. My first years learning forensic engineering were wonderful: someone was paying me to solve puzzles using math and physics. I would have done it for free, though I was never brave enough to share that thought with my boss. I've now been doing forensic work, mostly injury biomechanics, for 32 years and I still enjoy it. I find it brings a measure of reality to my research work that I do not think one can glean from, say, studying the NASS database or without examining crashed cars and autopsy/medical records.

Early in my career, there were some types of cases that appeared on my desk more often than others. The most common cases we saw at the time involved low-speed rear-end impacts and claims of whiplash injury. Our clients, typically an insurance company or a lawyer, wanted to know whether the collision caused the injuries being claimed by the claimant or plaintiff. This is a deceptively simple question that even after studying whiplash injury for 25 years, I often cannot answer. Nevertheless, our clients wanted an answer and so we set off doing our best to find an answer.

The first place we turned to was the scientific literature, but there was very little useful information in the literature to help us analyze or understand these cases. Based on the work of some very bright engineers—many of whom have graced this stage—we knew that the analysis process should go something like this: first quantify the vehicle collision severity, then use that information to quantify the occupant exposure, and then finally compare the occupant exposure to an injury tolerance value. Again, a deceptively simple-sounding process that still challenges us today, especially for whiplash injury.

We started by attempting to quantify collision severity. Most of the vehicles we encountered had bumpers equipped with isolators, which are small horizontal shock absorbers between the bumper beam and the vehicle frame/body. Based on an overly literal interpretation of FMVSS 215 (Exterior Protection), we used a model that assumed a 1-mph speed change (1.6 km/h) caused the isolators to begin compressing, a 5-mph speed change (8 km/h) caused the isolators to fully compress, and the system behaved linearly in between. Since we could measure the amount of compression an isolator experienced from the paint scrapes on the piston, we could use this model to calculate the severity of a particular crash. I don't know who created this model, but it had the alluring attribute of distilling the problem to a simple equation. And what engineer doesn't love an equation?

Having found a potential solution for estimating collision severity, we then consulted the scientific literature for a risk function that linked collision exposure to the risk of whiplash injury. Not surprisingly, there was no such risk function for whiplash injury—this was about a decade before Maria Krafft and Anders Kullgren began publishing their datasets beginning in 2000 (Krafft et al., 2000, 2002, 2005; Kullgren et al., 2003). Nevertheless, there were a number of volunteer exposures to low-speed crashes in various publications and they reported that their exposures did not generate symptoms. So based on a simple collision severity model and limited human subject data, we would offer clients a conclusion, something like, "The speed change of the vehicle was below 8 km/h and this level of exposure is not normally associated with a neck injury." Looking back, I'm now struck by the naivety needed to offer such a conclusion, but I'm also struck by the fact that the conclusion was largely correct—the isolator model wasn't too far wrong for many vehicles and most people are not injured in low-speed rear-end crashes.

I suppose this brings me to my first insight: When confronted with a seemingly intractable problem, make some assumptions—even sweeping assumptions—provided that: i) they move you forward, and ii) you later test these assumptions. (And by later, I really mean sooner.) I initially thought that science was a linear thing whereby we incrementally built knowledge upon the solid foundations of prior knowledge. In reality I've found that knowledge is often built rapidly on shaky foundations and then shored up (or torn down) by later studies that test assumptions implicit in the rapid, shaky experiment. It's not a pretty process, and it can make reading the scientific literature challenging; but, it can work relatively well provided that everyone understands and respects the assumptions and that the knowledge gained from the shaky experiment does not mutate

prematurely into fact. Now I'm not advocating that you run out and conduct a series of large experiments based on wild assumptions. That may get you fired. What I am advocating, though, is that periodically you shoot for the moon and see what you find. If done well, I guarantee it will either be right or provide you with years' worth of ideas that would not have occurred to you without the moonshot.

Despite being more or less correct, our assumed isolator model and our conclusion about the average person's injury risk at an 8-km/h speed change were singularly unhelpful answers for most clients from a forensic perspective. We had no way of determining whether the plaintiff's injury tolerance was below, near or above the average injury tolerance. And in a courtroom, it is the plaintiff's injury tolerance that matters, not the average injury tolerance of the general population.

Muscling In

In 1995, we turned our attention to the human subject responses to rear-end crashes and how these responses may modulate the risk of injury. By now we had staged over 1000 low-speed crash tests to document how car bumpers behaved, and we thought the best approach to understanding occupant responses was to conduct instrumented human subject tests. There were already some human subject tests published in the literature, but each one had issues that we thought limited their generalizability. The kinematics and kinetics had been well described as early as 1967 by Mertz and Patrick (1967), but their data were confined to a single male subject in a rigid seat. Moreover, there were only six female subjects tested in all of the whiplash literature (Matsushita et al., 1994; Szabo et al., 1994), which seemed odd given that we knew whiplash injury affected females more than males (States et al., 1972; O'Neill et al., 1972). However, I don't want to minimize the importance of these prior human subject studies: their data remain relevant and without their data, we would have made many more mistakes.

At the time, there were a number of prior studies that advanced the idea that neck muscles did not matter to whiplash injury because the neck muscles activated too late and because the delay between muscle activation and full force production was too long (Foust et al., 1973; Snyder et al., 1975; Reid et al., 1981). These studies were conducted by tugging the head of human subjects with a cable and measuring muscle activation times. There were two assumptions made in these studies that we thought might be incorrect. First, tugging on the head does not mimic the neurosensory stimulus of a whiplash exposure. During a rear-end crash, the pelvis and torso are accelerated forward while the head initially remains stationary. During a head tug, the opposite is true. Given the different mechanoreceptors and related neural pathways that exist in the head versus the pelvis and torso, we thought these differences might be important. And second, full force production may not be needed to alter the whiplash kinematics, and the onset of force production occurs very soon (~15 ms) after the onset of measurable electrical muscle activity (Corcos et al., 1992).

Just before we ran our study, Szabo and Welcher (1996) published muscle activity data in low-speed rear-end crashes showing that neck and lumbar muscle activation was transient, variable, and at levels below maximal activation. As it turns out, Ono et al. (1997) was running a contemporaneous study that showed that fully braced muscles prior to impact could alter the kinematics, but we did not know about their data until he presented it right before me at a Stapp Conference.

While all of these studies contributed to our understanding of whiplash injury, the pieces were disconnected and difficult to integrate. So we set out to generate a connected set of kinematics, kinetic, neuromuscular and clinical data from a relatively large group of subjects that included equal numbers of males and females. We collaborated with forensic consultants from Los Angeles: Jeff Wheeler and John Brault, and later with Terry Smith. When I reflect on this experiment, it was naively ambitious: aside from Terry, who joined partway through the study, none of us had a PhD, we collectively owned two accelerometers, and no one had a university affiliation and therefore we had no access to an ethical review board. Undaunted, we did what any engineer would do: we deconstructed the problems, learned the things we didn't know, and assembled what we thought was a solution. We ended up with a 3-2-2-2 array on the head, tri-axial linear accelerometers and

angular rate sensors on the chest, surface EMG electrodes over the sternocleidomastoid and cervical paraspinal muscles, 6DOF accelerometers in the vehicle, high-speed video, a head-restraint mount with 40 strain gauges to create a custom load cell (we couldn't afford actual load cells because we'd spent our resources on other equipment), and additional strain gauges at the seat hinges and seatbelt D-ring to measure the timing and amplitude of these loads as well (Figure 1). Subjects underwent clinical exams before, immediately after and 7-days after testing with daily self-evaluations. In the last month before we tested our first human subject, five of us worked until after dawn each morning to get the experiment up and running. I don't recommend this approach, but over the years I have noticed that complicated experiments seem to have considerable inertia during working hours, but tend to yield after midnight. Nevertheless, 42 subjects and 3 months later, we had acquired 21 seconds of data. I had no idea at the time, but this experiment led to 12 separate publications with very little overlapping data, except in some of the later publications that looked at dummy development and validation. Sometimes a little naivety can go a long way.



Figure 1. Experimental set up for human subject tests showing the pre-impact posture of an exemplar female subject in the right front seat.

This leads me to my second insight: creativity and hard work are more important than money and resources. That's not to say that you can conjure expensive instrumentation from nothing, but rather that good work does not depend on lavish resources and that hard constraints force you to be creative and focus your questions. We were—and remain—a small private company and we could not afford to compete with research being done at automotive companies or in better-funded university labs. Instead we focused our limited resources on a problem we could afford to investigate and where our work could stand out. This approach might be more important to others now that funding for impact biomechanics research has universally declined.

Startle Response

Despite all the sensors and wiggly lines on graphs that this experiment generated, the most important thing I observed came from simply watching the subjects during the tests. All of the subjects blinked very early after impact, and many subjects appeared startled—some subjects even elevated their arms in surprise. We had intentionally blinded them from knowing when the impact would occur in an attempt to make the laboratory exposure more realistic. None of the sensors—except the high-speed video—captured the startle response, but even high-speed video has a way of diminishing responses while at the same time allowing us to see other details we would otherwise miss. It was only by playing the high-speed video at full speed that the startle responses were readily visible. For me, the surprise exhibited by our subjects was somewhat of a revelation and

I began to wonder whether this response was important. For instance, it might not have been present during the head tug tests that had been used to justify why muscles could be ignored in whiplash injury. Also, having sat through many low-speed crashes myself prior to these tests, I recalled that my first exposure often produced a sympathetic response that caused me to sweat, but that this response disappeared following subsequent exposures. This combination of ideas got me thinking about whether the first exposure could be different from subsequent exposures and about whether our laboratory experiments were testing what we thought they were testing. Our human subject tests—and everyone else's human subject tests—were based on the assumptions that our laboratory tests were externally valid. In reality, however, we did not know if the responses we were observing were representative of the real-world responses we were trying to study.

This brings me to another insight: It's often the assumptions we don't know we're making that bite back the hardest. Earlier I encouraged you to make assumptions—even sweeping assumptions—to move your research forward. My current point is different. It's okay to make assumptions you know you are making; it's not okay to make assumptions you do not realize you are making. As humans, we are good at making assumptions: the floor is solid, the chair is stable, the food is safe, etc. We are so used to making assumptions that even if we stop and think critically about our assumptions, we may miss some. Others who review and read our work may not miss the same assumptions, and this is why we pitch our ideas to colleagues, and why we have peer review. I encourage you to view both as opportunities to improve your work.

My fleeting observations of this startle response led me back to school to do my PhD in biomechanics and neurophysiology. Based on this and later works, we were able to show that the initial muscle responses of deceived subjects (those who were told to relax so that we could get baseline muscle activity levels and then received an unexpected perturbation) were delayed compared to subjects who knew approximately or exactly when their first perturbation would occur (Siegmund et al., 2001). Further we were able to show that multiple sequential exposures to the same stimulus led to attenuated muscle responses and altered kinematics (Blouin et al., 2003; Siegmund et al., 2003). This process, called habituation, had been observed in perturbations to standing balance (Nasher et al., 1976), but we did not know if and to what extent it was present in seated subjects exposed to a rear-end impact. The presence of habituation in our subjects indicated that they were only useful for a single exposure if we wanted to maximize external validity. The simultaneous changes in muscle activation and kinematics also showed that muscles played a role in modulating the head and neck kinematics during a rear-end collision. A few years later, we finally put together the right combination of experiments and data analysis techniques to show that the exaggerated response to a novel stimulus appeared to be the superposition of a startle response and a postural response, and that habituation was likely the extinguishing of the startle response (Siegmund et al., 2008). We still don't know whether habituation is present when the stimulus is very noxious or painful: a noxious/painful stimulus could sensitize the subject and potentially amplify subsequent responses. Nevertheless, we do know that for the kinds of whiplash exposures we use to study human subjects, habituation is present. We subsequently showed that adding a loud acoustic stimulus could restore the startle response (Blouin et al., 2007), and that a 20-minute gap between perturbations combined with a loud acoustic stimulus eliminated the significant habituation-related changes we had previously observed (Mang et al., 2015). It's important to note that the lack of a statistically significant change doesn't mean we eliminated habituation: it just means that we attenuated it below a threshold where we had the power to detect it.

Bucking the Trend

In contrast to a decline in the overall rate of auto-related injuries, the rate of whiplash injury has stayed the same or increased over the last few decades (Temming and Zobel, 1998; Styrke et al., 2012; NHTSA, 2018). I believe there may be structural reasons for this pattern. First, there has been too little research into whiplash injury, too few innovations to mitigate or prevent whiplash injury, and too little penetration of innovations into the vehicle fleet. Compared to the nearly universal introduction of airbags, seatbelt pre-tensioners, crumple zones, etc., the penetration of anti-whiplash seats into the fleet and the success of these anti-whiplash seats pale.

Second, cars have become stiffer and more elastic over the same period of time. Where cars used to be equipped with bumper isolators that dissipated energy and would sustain energy-dissipating damage during low speed impacts, they now have rigid bumper mounts and foam liners that sustain little or no damage at much higher impact severities than before. Foam liners are also more elastic than isolators at higher impact severities and return more energy to the system during rebound. Both stiffness and elasticity have changed because of cost, government regulation and insurance industry pressure. The drive for fuel efficiency required lighter vehicles, and foam-lined rigid bumper systems are lighter than isolator-equipped bumper systems. For a problem like whiplash injury, which only affects a small proportion of the population, a push to minimize vehicle damage by making stiffer bumpers with higher damage thresholds also makes economic sense. If a minority of occupants are injured and a majority of cars are damaged, then the obvious solution for the insurance industry is to push for cars that damage less in these crashes. Of course, this solution assumes that the injury rate won't change as car designs change. But as engineers, we know that if we increase the vehicle stiffness and collision elasticity for a low speed impact, we increase the peak acceleration and speed change respectively of both vehicles for a given closing speeds. Given that we expect an increased number of injuries at higher collision severities, an unintended consequence of these bumper-system changes could be higher whiplash injury rates.

Third, there are a greater number of vehicles on the road, more traffic, and thus more low-speed collisions. These numbers are hard to track because many low-speed collisions go unreported; however, low-speed collisions occur more frequently in traffic than on the open road, and I think we can all agree that there is more stop-and-go traffic now than in the past. If we also accept that the distribution of closing speeds has not changed, then a greater number of low-speed crashes are occurring and these crashes are generating higher peak accelerations and higher speed changes for the occupants inside the vehicles. Without a parallel and offsetting improvement in seat technology and equally large penetration of these seats into the fleet, should we be surprised that the number of whiplash injuries has risen?

Thus in some respects, we can view whiplash injury as a manufactured problem. We essentially created the injury through mechanization (first trains, and then cars), and we've exacerbated it by optimizing aspects of the system (e.g., improving fuel economy and raising damage thresholds) unrelated to occupant safety. The irony is that in some jurisdictions, the solution to the increasing number of whiplash injuries has been to limit the insurance claims for these injuries. This strikes me as unfair. The injured parties appear to be bearing the burden of a problem created in part by the automotive and insurance industries.

A Small Rate Problem

It is discouragingly difficult to study an injury that not only lacks an objective diagnosis, but one where only a minority of the acutely injured population transitions to a chronic injury. When 25 to 40% of individuals experience acute symptoms following a rear-end crash (Zuby et al., 2010) and fewer than 10% go on to develop chronic symptoms (Suissa, 2000), the ways we approach our studies of whiplash injury change. For instance, epidemiology studies need large populations, but many study participants who could help identify causal factors are never enrolled because they don't even report being in a collision. These enrolment losses affect our calculations of injury risk. Experimental studies may also be misleading if they focus on evaluating the mean responses, which may mask the outliers that potentially capture the mechanics responsible for injury. Dummies and computational models are then built to mimic these mean responses and injury metrics are then developed; all of which may miss the factors that cause lasting injury in the subset of the population who develop chronic injury. Imagine these dummies and metrics then being adopted by an insurance rating system or into a government standard that ensures the average person doesn't get whiplash injury. Isn't this where we began?

While seemingly absurd, this series of events has in fact happened. One of the few things we know with some certainty about whiplash injury is that it affects females more than males. And yet we have a 50th percentile male BioRID dummy that is used to evaluate seats that are then designed to perform well with this dummy. A

partial solution to a small part of the problem. To their credit, there are a number of researchers in Sweden, some of whom are here, who have been developing a 50th percentile female whiplash dummy, called the EvaRID (Carlsson et al., 2014), and I applaud them for doing so.

To address these issues, we have tried to adapt our studies in different ways. First, we have tried to design studies that examine questions that are insensitive to (though not entirely free of) these problems. For instance, we have used female subjects and larger numbers of subjects in our studies to better capture the range of responses. Second, we expect large variability from our human subjects and try to interpret this variability in the context of a low transition rate to chronic whiplash injury. For instance, we have seen widely varying responses in the cervical multifidus muscles between subjects exposed to the same simulated rear-end crash (Siegmund et al., 2008). We know these muscles insert directly onto the facet capsular ligaments at C3 to C6 (Winkelstein et al., 2001; Anderson et al., 2005), and contraction of these muscles could exacerbate the kinematic-induced strains these ligaments experience during a rear-end impact. It may be those subjects with large multifidus responses we should be examining more closely. Another example is focusing on two of 13 cadaver specimens that showed exposure and tolerance responses that indicated an injury would have occurred in our tests (Siegmund et al., 2001). Rather than diluting these data with the other 11 specimens, we recognize that an injury rate of 15% in our admittedly small population was not too different from the rate of chronic injury in the exposed population. Over interpreting a minority of one's data comes with its own dangers, but my point here is that we need to look past the mean responses if we're going to unravel whiplash injury.

Another approach that we are only now developing is to test patients who actually have chronic whiplash pain. We have long been wary of going down this road, but these patients are motivated to help unravel whiplash injury. In addition to their neck morphology, we will be looking at neck muscle reflex responses during benign seated oscillations to learn whether their biomechanics and neuromuscular systems are different from controls. We won't know if some of the differences we observe were present before their collision or whether they are a result of remodeling after their collision, but we have to start somewhere. We're also exploring inducing pain in normal subjects to evaluate how they adapt their responses in the presence of pain. Stay tuned.

Counter-Intuition

One possible reason we have not advanced far towards preventing whiplash injury is our inherent belief in a monotonically increasing dose-response relationship for injury. This belief is readily deduced from our lived experiences beginning at a young age: the harder the punch, the greater the pain. Indeed this concept of more-force-equals-more-injury may be a key factor underpinning the prejudices against whiplash injury I talked about earlier. But more germane to our field is the fact that a monotonic relationship between dose and response is baked right into our injury risk functions. What if this assumption is wrong? What if this was one of those assumptions we did not realize we were making? What would that do to our vehicle design decisions? To our regulatory or insurance rating systems?

There is some evidence to support the idea that injury severity—typically measured by pain intensity and/or duration for whiplash injury—does not increase monotonically with impact exposure. The first piece of supporting evidence was published by Maria Krafft and her colleagues (Krafft et al., 2005): they showed that the risk for Grades 2 and 3 whiplash injuries (WAD 2 and 3 on the Quebec Task Force grading scale for whiplash-associated disorders) initially increases with collision speed change and then decreases at higher speed changes (Figure 2, left). Now before you point out that the sample size at these higher speed changes is too small to support this contention, let me say that I agree with you. I noted the pattern and initially dismissed it for that very reason. It wasn't until the second piece of evidence arrived 3 years later from Beth Winkelstein's lab that this concept came into focus for me. What they observed in their rat model was a relatively rapid return to normal pain responses when the facet capsular ligament was ruptured, but prolonged pain responses when the facet capsular ligament was stretched to lengths that caused only sub-catastrophic failures in the ligament, i.e., lengths consistent with a whiplash exposure (Lee et al., 2008; Figure 2 right).

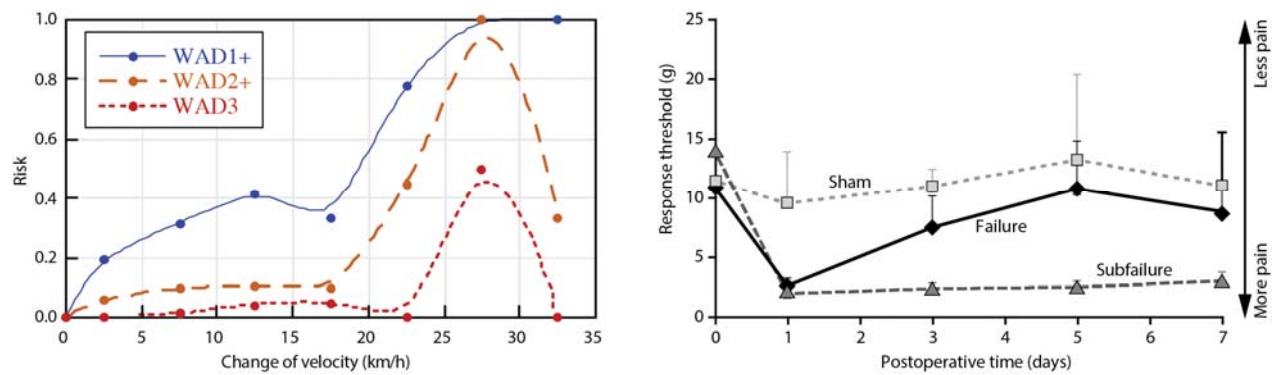


Figure 2. Potential evidence supporting a non-monotonic dose-response relationship. Left panel: Risk of WAD1+, 2+ and 3 injuries as a function of vehicle change in velocity (adapted from Krafft et al., 2005). Note the lower risk at higher speed changes. Right panel: Response thresholds for the rat forepaw using von Frey filament stimulation following sham, failure and subfailure of the facet capsular ligament (adapted from Lee et al., 2008). Note the prolonged period of lower response thresholds (greater pain sensitivity) for the subfailure condition.

For me, this upending of the monotonic dose-response relationship was another revelation in my thinking about whiplash injury. It suggested that the dose-response relationship between loading and the degree of ligament rupture (the engineering outcome) might be monotonic, but that the dose-response relationship between the degree of ligament rupture and the duration of pain (the clinical outcome) was not. This counter-intuitive finding provides a possible explanation for how less severe collisions could generate injuries with worse clinical outcomes than those following more severe collisions. Since we don't see many diagnoses of full ligament ruptures following a rear-end collision, the rat model may not provide a complete explanation of whiplash injury; but nevertheless the concept of lower exposures being related to worse clinical outcomes may still be relevant to whiplash injury.

The toxicology world routinely deals with non-monotonic dose-response relationships in biological systems, something they call hormesis (Calabrese and Baldwin, 2001). They have many instances where an increasing dose of a drug (or even some toxins) initially improves health (or some other biological measure) at low doses, but then harms health as the dose continues to rise. I'm not proposing that low-severity collisions could be good for our health; what I am proposing is that we think more critically about the relationships between the dose and response that underlie our injury risk functions. I believe we're on relatively solid ground when the dose and response metrics are closely connected, e.g., applied force and structural failure. I am less assured when the connection between the dose and response metrics grows more distant, e.g., vehicle speed change and symptom duration for whiplash injuries. A risk function linking speed change and symptom duration consists of the superposition of multiple intermediate dose-response relationships (and other transfer functions), some of which may not be monotonic or relevant across the entire exposure spectrum. For instance, it is not hard to imagine that the risk of a particular response or injury might diminish or be eliminated by some discrete event like seat back failure. Considerably more work is needed to explore these concepts, but they may help us to better understand and delineate whiplash injury outcomes.

The Future is Coming

Most of what I've said so far has involved looking back. I'd like to close with a few comments on the future. There are three areas that I see as key to moving our understanding of whiplash injury forward.

The first is computational modeling. Computer models (e.g., Brolin et al., 2015; Cronin, 2014) are the only realistic option for running the kinds of parametric studies that we need to unravel injuries that suffer from some of the problems I've discussed. Yes the models will need to improve, and yes they will need to contend with issues like sub-catastrophic tissue failures and hormetic dose-response functions, but there is no way to systematically explore the parameter space using cadavers, humans or dummies.

The second area is imaging and diagnostics. This area lies mostly outside our expertise, but there is some fascinating work being done by Jim Elliott and his colleagues using diffusion tensor imaging to detect fatty infiltrates in muscles shortly after a whiplash injury (Elliot et al., 2010). This work, and other works like it, has the potential to identify chronic patients well before they become chronic, and provide the foundation on which to build more effective treatment paradigms.

The third and perhaps most important area is prevention. Some folks see self-driving cars as the solution to all automobile-related injuries, but I don't (yet) share their optimism—at least not in the immediate future. Given the complexity and unpredictable nature of our road environment and other road users, I think we'll be seeing collisions and injuries for some time to come. We should continue to work on understanding injury mechanisms and continue developing safety systems that eliminate or attenuate these mechanisms. In our lab, we've been working on an active and adaptive car seat that attenuates key occupant kinematics by more than 50% compared to the current crop of anti-whiplash seats. We've also explored acoustically startling occupants immediately prior to impact to evoke their startle response before the crash, causing the impact to occur during the startle response's refractory period. I don't know whether either idea will be adopted, but our collective role is to reduce traumatic injuries, and this is what we've set out to do.

Closing

At the outset of my career, I had no idea that taking a job at a fledgling forensic engineering company would (or could) lead to standing at this podium sharing my thoughts on whiplash injury and research with some of the world's leaders in injury and impact biomechanics. But opportunities are everywhere it seems. And you never know where they will take you. The last insight I'll share with you is one that my mentor shared with me early on: try a lot of things and keep what works. I know it sounds trite, but with a little reflection and discernment about whether something is indeed "working" for you, it remains solid advice.

I. ACKNOWLEDGEMENT

There are many people and organizations whose contributions and funding deserve more credit than I have space for. Thank you to all of my coauthors and collaborators for their patience, insights and hard work. A special thanks to the Science Council of British Columbia, a now defunct funding agency, who helped fund our first human subject study. Other funding sources include the Natural Sciences and Engineering Research Council of Canada, the Canadian Institute for Health Research, Auto21, BC Chiropractors Association, The Whitaker Foundation, Michael Smith Foundation for Health Research, WorkSafe BC, and the Canadian Chiropractic Research Foundation. And perhaps most importantly, thanks to my partners at MEA Forensic for sharing the view that research, new knowledge and giving back to our community is both a responsibility and in the long term interest of the company.

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