Journal of the
National
Academy of
Forensic
Engineers®
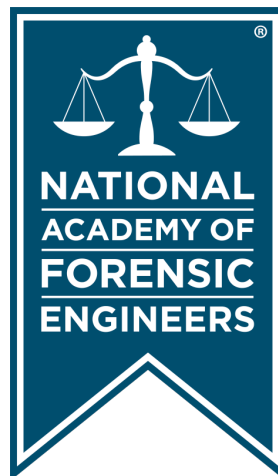
FE Analysis of Upper Extremity Nerve Entrapment Injury Mechanism as Related to Rear-End Collisions

By William E. Lee, PhD, PE (NAFE 655S)

Abstract

Nerve entrapments of the median nerve, i.e., carpal tunnel syndrome (CTS) and the ulnar nerve, i.e., cubital syndrome (CT) are relatively common, reflecting traumatic and atraumatic mechanisms. Claims of such injuries in relation to rear-end collisions (particularly low-velocity or < 10 mph collisions) are often contested by the defense, acknowledging that there is no obvious relationship between the collision and the claimed injuries. Of the collision types (frontal, side, rear-end), it is the least clear how a rear-end collision can establish mechanisms for such injuries. Direct blunt trauma to the carpal tunnel region or the cubital tunnel region are unlikely in a rear-end collision. Also, "stretch" injuries due to hypermotion of either the wrist or elbow are unlikely, reflecting occupant kinematics, vehicle interior geometry, and other factors. A case study involving CTS and CT claims as a result of a low-velocity rear-end collision will be presented.

Keywords

Nerve entrapment, rear-end collision, carpal tunnel syndrome, cubital tunnel syndrome

Introduction

The median, ulnar, and radial nerves are important motor and sensory nerves of the upper extremity. So-called compressive entrapment injuries can occur that have a negative impact on one's quality of life (on a societal scale, lost productivity) and may require surgical intervention in more severe cases. "Compressive entrapment injuries" or compressive neuropathies typically involve the creation of pressure on the associated nerve as a result of swelling or other medical conditions of the surrounding tissues, which decreases nerve function at that level.

Carpal tunnel syndrome (CTS), which involves a compressive neuropathy of the median nerve, is the most common such condition in the upper extremity. Within the United States, incidence (newly diagnosed cases/year) is estimated to be in the range one to three cases per 1,000 persons per year and a prevalence (total existing cases/year) of around 50 cases per 1,000 subjects per year (Bickel 2010). Nordstrom and co-investigators (1998) observed that the incidence of CTS increases with age. These statistics refer to the general population. For the industrial setting, prevalence has been estimated to be 5% to 15% of industrial workers (Neal and Fields 2010). Cubital tunnel

syndrome (CT), which involves a compressive neuropathy of the ulnar, is the second most common peripheral nerve entrapment. The incidence of CT has been estimated at 24.7 to 30.0 cases per 100,000 persons per year, significantly lower than the CTS incidence value (Assmus et al. 2015; Osel et al. 2017). Radial nerve compressive neuropathies are comparatively rare, with the incidence reported as less than 10% of the ulnar nerve compressive neuropathy value (Latinovic et al. 2006). Given the relative incidence values, this paper will focus on median and ulnar nerve compressive neuropathies.

CTS and CT are sometimes associated with vehicular collisions. As such, personal injury lawsuits may be based on a causal relationship between a documented peripheral compressive neuropathy and the vehicular collision. The underlying theory of causation usually is based on some blunt trauma event (e.g., a wrist contacting a steering wheel) or a nerve or associated tissue stretch event due to a tension load, often associated with a hyperextension or hyperflexion movement of some sort involving the wrist or elbow. Publications on this topic have been limited, and the biomechanics component of these publications is often unclear. Limited information suggests that rear-end
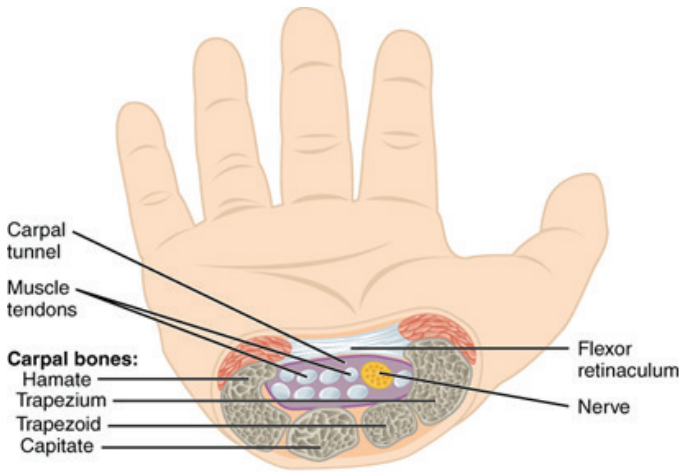
William E. Lee, PhD, PE, Department of Chemical and Biomedical Engineering, University of South Florida, Tampa, FL 33620, wlee2@usf.edu

Figure 1
Basic anatomy of the carpal tunnel (Open Stax College, 2013).

collisions are more frequently associated with such injuries, with frontal collisions a distant second (Coert and Dellon 1994). The purpose of this paper is to explore the extent to which any injury mechanisms are established for occupants of vehicles that are rear-ended in low-velocity impacts.

Basic Anatomy

The median and ulnar nerves are important nerves that affect finger/wrist motion and also provide sensory information from the same body area. The median nerve goes through what is known as the carpal tunnel (sometimes called the carpal canal) at the base of (or palmar side) the wrist (see **Figure 1**). Note that both flexor tendons (nine of them) and the median nerve pass through the carpal tunnel.

The tunnel is surrounded by carpal bones on the dorsal side and soft tissues throughout, including the flexor retinaculum (also called the transverse carpal ligament or anterior annular ligament) along the palmar side. The transverse carpal ligament is approximately 3 cm to 4 cm wide. The carpal tunnel is a narrow structure, with the narrowest section being approximately 1.6 cm^2 cross-sectional area (Schuenke et al. 2014) . The average width is around 25 mm, with the narrowest region being about 20 mm (Gillig 2016).

Wrist movement influences the geometry of the carpal tunnel, reflecting movement of the carpal bones as the wrist goes through various motions. Both wrist flexion and extension increase compression on the carpal tunnel. The median nerve itself can move up to almost 10 mm in wrist flexion (Ibrahim et al. 2012). The ulnar

nerve passes through a space of the dorsal medial elbow known as the cubital tunnel. The humeral medial epicondyle borders the cubital tunnel medially and the ulnar olecranon process laterally. With elbow flexion, the cubital tunnel becomes taunt; with elbow extension, it becomes lax.

The peripheral nerves employ anatomical features that may serve to protect the nerve fibers from mechanical damage, specifically from stretching or traction (tension) loads and compressive loads. The phenomenon called "nerve gliding" where the nerve can stretch is critical to prevent nerve damage while nerves are stretched during joint range of motion (Wehbe 2004). The "protection" comes from successive layers of connective tissue that make up the myelin sheath and also the nerve fibers themselves. In general, nerves have significant tension strength. For example, the median and ulnar nerves may be able to withstand 70 to 220 N and 60 to 150 N of tension loading, respectively (Rydevik et al. 1989).

These values refer to the nerve fibers themselves; other associated tissues may experience damage prior to nerve fiber injury. In general, the composite nature of nerve tissues, including the associated connective tissues and blood supply, makes it challenging to analyze from a biomechanics viewpoint. The maximal elongation (strain) of nerve fibers under tension is around 20% at the elastic limit (where the tissues would return to their pre-loading state). Complete structural failure may occur at around 30% strain.

In compression loading, functional changes may occur at about 30 mm Hg pressure. Pressure levels around 32 mm Hg have been recorded close to the median nerve in the carpal tunnel for CTS patients. The pressure in normal subjects was around 2 mm Hg (Rydevik et al. 1989). Investigators have explored the role of time duration of the pressure application, finding that the longer the pressure is applied, the higher the chance for nerve damage, possibly reflecting the viscoelastic properties of peripheral nerve tissue. As one example, investigators have found that maintaining direct nerve compression at 30 mm Hg for short durations (2 to 4 hours) may cause reversible changes; prolonged compression can cause irreversible nerve damage (Rydevic et al. 1981; Lundborg et al. 1982).

Carpal and Cubital Tunnel Syndromes

If the carpal tunnel becomes narrowed — or if one of the tendons or tissues of the carpal tunnel become inflamed, swollen, or fibrotic — this can apply pressure on the other tunnel structures, including the median nerve,

leading to carpal tunnel syndrome (also sometimes referred to as distal median nerve entrapment).

Among the possible causes of tissue swelling are various illnesses (including hypothyroidism, rheumatoid arthritis, and diabetes), obesity (BMI), repetitive motion, and blunt trauma of the carpal tunnel itself. Fractures of nearby bones can also cause CTS. Genetics may also play a role (Armstrong et al 2008; Bickel 2010). Median nerve pressure can lead to pain, numbness, tingling, and weakness of the hand. Pain may extend up the arm. CTS is often the result of the inflammation of tissues around the median nerve and not a problem with the nerve itself. If any resulting pressure on the median nerve continues over a long time, median nerve damage can result. However, neuropathic factors (such as diabetes, alcoholism, nutritional deficiencies, and toxin exposure) may affect the median nerve itself. It has been shown that diabetic patients have a higher tendency to develop CTS due to a lower threshold for nerve damage (Pourmenari and Shiri 2015). Obesity is also a recognized risk factor for CTS (Shiri 2015).

There are many possible causes of acute CTS as reported in the literature. At a general level, the causes can be grouped into atraumatic and traumatic (Gillig 2016). Atraumatic causes includes inflammation, infections, circulatory issues, and tumors. Traumatic causes include wrist trauma such as fractures, dislocations, crush injuries, penetrating injuries, and distal radius fractures. Traumatic causes basically involve direct compression of the carpal tunnel (Jhattu 2012). Among the causes of wrist trauma are forward falls (slip and falls, trip and falls) and falls from height. Many of these fall events are associated with the so-called "fall on an outstretched hand" (FOOSH), the most common upper extremity injury mechanism seen in trauma medicine (Chung and Spilson 2001).

Women are approximately three times more likely than men to develop CTS, reflecting the anatomical fact that the carpal tunnel is smaller in females relative to males. Many studies focus on repetitive motions as a cause of CTS. For example, Hagberg and coworkers (Hagberg et al. 1992) looked at CTS as it related to 32 occupational or exposure groups, with the highest prevalence being for grinders, butchers, grocery store workers, frozen food factory workers, platers, and workers with high-force, high-repetitive manual movements.

As another example, Szabo (1998) notes that many studies claim CTS is related to workplace factors, although he also admits some studies find little evidence of the workplace relationship. Recently, Dale and coworkers (Dale et al. 2015) looked at repetitive motion and force exposures, identifying high/high, high/low, and low/low exposures. Their research showed that workers with high force/high repetitive jobs (examples from their study: dishwashers, brick masons, carpet installers, drywall installers, upholsterers, hand packers/packagers) had the highest prevalence of CTS. The current evidence to support the hypothesis that typing ("keyboarding") causes CTS is relatively weak, with most studies concluding that there is a weak association at best (Shiri and Falah-Hassani 2015).

As noted above, increased pressure within the carpal tunnel is a characteristic of CTS. McGorry and coworkers (2014) measured carpal tunnel pressure in association in healthy (no CTS) subjects while they performed various wrist activities with and without resistance and with and without gripping. Active gripping did not increase the pressure during wrist flexion (it often decreased), but did increase with wrist extension. The resting pressure was 3.5 mm Hg (standard deviation of 2.3 mm Hg) and increased to 7.7 to 10.6 mm Hg for activities involving grip and wrist flexion.

In one motion similar to dart throwing, going from no-grip to grip with the wrist extension movement otherwise being similar, the pressure only increased less than 1 mm Hg when a power grip was added to the activity (i.e, most of the pressure contribution was due to the extension movement). The McGorry study did not report the time duration of any of its test activities. Goss and Agee (2010) showed that intracarpal tunnel pressure increased with increasing grip strength. Keir and coworkers (2007) proposed a threshold of 25 to 30 mm Hg tunnel pressure as a workplace guideline. Studies of grip strength in subjects with various stages of CTS have shown that grip strength decreases with as the severity of CTS increases (Atalay et al. 2011; Baker et al. 2013).

The diagnosis of CTS may be challenging at times and associated with diagnosis errors. Assmus and coworkers (2015) have noted that CTS can be misdiagnosed as a C7 syndrome or as a circulatory issue (and vice versa). Witt and Stevens (2000) described 12 cases that were initially diagnosed as CTS, but were ultimately found to be a different neurologic disorder. Mireles and coinvestigators (2009) noted that most CTS misdiagnoses were false negatives, sometimes leading to unnecessary surgeries.

Following CTS, CT is the second most common entrapment neuropathy. It is usually the result of ulnar nerve

compression at the elbow as it passes through the cubital tunnel. CT can be caused by bone deformities, regional tumors, synovitis, and nerve enlargement (Cutts 2007; Wojewnik 2009). According to one proposed injury mechanism, repeatedly leaning on one's elbow (especially on a hard surface) may lead to CT (Nainzadeh 2011). Chronic repetitive microtrauma associated with repetitive elbow flexion/extension can cause local inflammation and perineural scarring, resulting in nerve compression. Repetitive traction can lead to CT as seen in boxers, baseball pitchers, weight lifters, and assembly line workers. Basically, anything that leads to a decrease in the volume of the cubital tunnel can lead to CT, including obesity, diabetes, inflammation, and direct blunt trauma to the inside of the elbow. Several studies have shown that occupational tasks that require relatively constant pressure on the elbow (including the cubital tunnel area) may be a risk factor for CT (Descatha et al. 2004).

Vehicular Collisions and
Upper Extremity Nerve Entrapment

Investigators Coert and Dello (1994) investigated injury mechanisms for CTS and CT as they related to vehicular crashes. In their study, they examined 68 subjects involved in collisions that included CTS and CT claimed injuries. Out of the 59 collisions, 13 were frontal, 34 were rear, 11 were side impacts, and one was considered other. Most of the subjects were using a seat belt, and only one of the collisions involved an air bag deployment.

According to their proposed injury mechanism, as the driver moves forward with hands on the steering wheel (or hands contacting the dash), a compressive force is applied between the palmar aspect of the wrist and the steering wheel or dashboard, causing the wrist to hyperextend and the median nerve to stretch. This can lead to a traction injury to the ulnar nerve within the post-condylar groove from hyperflexion of the elbow. Such forces could be established during a frontal collision or during the rebound portion of a rear-end collision. As such, the mechanism is one of compression (possibly related to blunt trauma), a traction injury, or a combination of both.

Wiechel and coworkers (2006) noted that in rear end impacts, the occupant would not experience impact to the wrist, especially during the initial backward-directed movement (they did not address the rebound phase). They also observed that to require a contact force to the carpal tunnel requires a forward motion of the occupant into the dash or steering wheel coupled with alignment of the palmar aspect of the wrist collinear with the direction of force

applied to the vehicle — something they opined was extremely difficult to accomplish. They further noted that if the wrist is stretching, the median nerve results in a strain that is well below any identified tension threshold injury level.

In a retrospective study, Ames (1996) examined 96 patients diagnosed with CTS involved in vehicular collisions. The type(s) of collision was not addressed. Ames developed a proposed mechanism that was based on gripping the steering wheel, more specifically as "blunt trauma" established when the driver moves forward, applying force to the wrists that are in contact with the steering wheel. Note that the driver was presumably already holding onto the steering wheel. He also noted that similar blunt trauma can occur when an occupant moves forward and contacts the dash with the wrist(s). Guyton and Honet (1977) hypothesized that CTS was due to momentary hyperflexion or hyperextension of the wrist while tightly grasping the steering wheel or bracing for impact. Haas and Nord (1981) similarly hypothesized that CTS was due to interactions between the hands with the steering wheel.

In a more recent study, Melhorn and coworkers (2009) examined the literature that explored causation of CTS by motor vehicle accidents. They considered 290 articles that focused on acute trauma using an evaluation protocol that incorporated temporality, strength of association, dose-response relationships, coherence, consistency, specificity, plausibility, predictive performance, and other epidemiological factors. From their conclusions:

> Currently there is "insufficient evidence" based on the above method of assessment and analysis of the current medical literature to support the hypothesis that there is causality between motor vehicle accidents and the onset of carpal tunnel syndrome in individuals who have not had a sufficient traumatic event such as fracture of the same wrist at the time of injury.

They noted that acute compression of the median nerve at the wrist can occur as a secondary result to wrist trauma (for example, wrist fractures and joint capsule tears) and also can be related to infectious, rheumatologic, and hematologic issues.

The preponderance of human subject rear-end impact studies shows the initial backward movement of an occupant due to the impact followed by a forward "rebound" movement (for example, McConnell 1995;

Howard 1999; Siegmund 1997; and Viano 2013). Many of these studies showed that the "rebound" phase was associated with a significantly reduced velocity and acceleration relative to the initial backward movement. One limitation of most of these studies: They typically focused on the description of head/neck motions along with general body motions, with little to no attention to upper extremity movement. However, a study reported by Furbish and co-investigators (2011) did investigate upper extremity motions during low-speed (ΔV from 5.3 to 7.2 mph) rear-end collisions.

Two conditions were included in the study — one where subjects were unaware of the time of impact and one where they knew the impact was about to occur (all subjects experienced both conditions). In three of the 18 tests, only one hand was placed on the steering wheel pre-impact. Otherwise, both hands were placed on the steering wheel post-impact for both the aware and un-aware conditions. The study found that unaware subjects did not maintain a controlled grip on the steering wheel at impact while otherwise moving backward into the seat, but re-established a controlled grip approximately ½ to 2 seconds following the impact. Aware subjects maintained a controlled steering wheel grip throughout the impact phase.

For aware subjects, steering wheel loading indicated tension and downward loading for the first approximately 150 seconds (backward occupant movement) followed by a period of compression and upward loading for the following approximately 150 seconds (forward occupant movement). Aware subject experienced a higher peak mean tension load (760 N) versus the unaware subjects (424 N). Aware subjects experienced a mean compression load of 756 N. Unaware subjects displayed "indiscernible" compressive loads while re-establishing steering wheel contact after initially losing grip. The investigators also observed that aware subjects loaded the seatback at a level significantly less than unaware subjects. Aware subjects braced more effectively, which restricted their general body motion relative to unaware subjects.

A study by Bruno and coinvestigators (2019) reported on elbow kinematics during low to moderate speed rear-end impacts. Their study employed Hybrid III 50th percentile male anthropomorphic test devices (ATD) in the restrained driver position. They observed that elbow excursion during the initial backward movement was more significant than the subsequent forward excursion during the rebound phase. Their video frames showed the ATD losing grasp with the steering wheel during the initial backward movement.

Discussion

The discussion above indicates that any acute injuries to the median or ulnar nerves (specifically CTS and CT) require the application of force to the region of interest. For CTS, there must be a direct application of force to the carpal tunnel region. It has been established in the medical literature that wrist injuries associated with vicinity fractures or ligament or other soft tissue damage can lead to CTS. One should therefore be vigilant in situations where CTS is claimed, and there are no other significant injuries of the carpal tunnel-related structures.

Regarding rear-end collisions, it is unclear how any significant force can be applied to the carpal tunnel region during the collision phase. As the work of Furbish et al showed, in cases of unaware drivers, they lost contact with the steering wheel during the impact phase. Therefore, the question of gripping force or any other carpal tunnel area applied forces becomes irrelevant. Note that while one is gripping the steering wheel, the force is actually not being applied directly to the carpal tunnel region; with the fingers grasping the steering wheel, the carpal tunnel region may be a short distance (perhaps 1 to 2 inches) proximal to the steering wheel-palmar side of hand contact area. The occupant is initially moving backward during the collision phase; any loading of the carpal tunnel would be unlikely. During the subsequent rebound phase, the force experienced by the occupant is significantly mitigated (relative to the initial backward movement). This was demonstrated in the work of Furbish et al, specifically regarding the upper extremity movements.

Simple "stretch" injuries reflecting the exceeding of some joint(s) range of motion appear to be unlikely. Such stretching basically applies a tension load to the nerve structures. Nerve structures are designed to withstand strain associated with body motions, specifically joint motions including both elbow and wrist or hand motions. In the forward motion (rebound phase) of a rear-end collision, it would be very difficult to establish any hyperextension movements of the wrist. First, the force of impact with some vehicle structure would be very low since the rebound phase of a rear end collision is significantly less forceful than the original backward movement (which is also not forceful in a low-velocity rear-end collision). Also, it would be very difficult to position a wrist on a steering wheel where the arm otherwise wants to "go through" the steering wheel (while the body is otherwise moving

	Women (mean±sd)	Men (mean±sd)
Flexion (deg)	152.5 ± 4.1	148.4 ± 4.1
Extension (deg)	11.9 ± 6.2	11.9 ± 7.5
Total ROM (deg)	140.6 ± 6.5	136.5 ± 9.0

Figure 2
Flexion, extension, and total range of motion (ROM)
for adult men and women (Chapleau et al. 2013).

forward), and the fingers remain firmly gripping the steering wheel. The geometry of many steering wheels simply will not allow this to occur.

Similarly, there must be a direct application of force to the cubital tunnel region to possibly cause CT. Given that the cubital tunnel region of the elbow is located medially, it would be difficult to apply direct force to the region during the body motions of a typical rear-end collision. It is possible that any "flailing" motions or other motions of the body may establish incidental contact between the lateral aspect of the elbow and a vehicle interior structure (for example, the vehicle door). Again, the cubital tunnel is located medially and therefore experiences a high degree of protection from such lateral contacts. As with the carpal tunnel, damage to associated structures (including nearby fractures) can lead to CT (Li 2013). Prior fall events and other blunt trauma events may be responsible for such CT injuries as opposed to a vehicular collision where no such injuries were documented.

As further criticism of the proposed mechanism of Coert and Dellon (focusing on the elbow motion), it should be noted that the normal range of motion for males and females is as follows (see **Figure 2** above). For the elbow to hyperflex, it would have to exceed the mean values for flexion shown in the table. Referencing activities of daily living, the elbow goes through 89 to 107 degrees of flexion while cutting with a knife, 85 to 128 degrees putting a fork in one's mouth, and 43 to 136 degrees while using a phone (Zuckerman and Matsen 1989). This seems unlikely to occur in a rear-end collision. In videos of human subject rear-end collisions, there are two scenarios: 1) the driver continues to grasp the steering wheel through the entire backward-forward movement during the collision phase; or 2) the driver lets go of the steering wheel at some point.

These two scenarios were described by Furbish and coworkers (2011). Obviously, the question of injury mechanism is irrelevant if the driver lets go of the steering wheel during the collision phase. If the driver continues to grasp the steering wheel, the range of motion that the

elbow goes through as the driver moves backward into the seat and followed by any rebound motion is well within normal range of motion. This elbow movement serves to reduce any loading of the hands or wrists while maintaining a grasp on the steering wheel. This discussion also is not applicable to the occupants other than the driver because any associated elbow region movement during the impact phase would be limited and within normal range of motion, to the extent that there is even any elbow movement. Note that most such occupants would not normally be bracing during a rear-end collision, especially if the impact is unanticipated (Furbish 2011, Lee 2000).

For wrist flexion/extension, the normal values have been reported as 85 to 90 degrees for flexion and 75-80 degrees for extension (Stuchin 1989). There is only a slight difference between male and female values. Note for the wrist to hyperextend, it would have to exceed approximately 85 to 90 degrees. In terms of the total arc of motion (total flexion + extension), 121 degrees with a range of 84 to 169 degrees has been reported (Sarrafian 1977). For reference, the wrist goes through almost 43 degrees total arc to use a phone and 63 degrees total arc to rise from a chair (Stuchin 1989). As noted above, it is unclear how any hyperextension of the wrist can occur during a rear-end collision.

This analysis has focused on the forward-backward movement of the upper extremities during rear-end collisions. In some instances, there may be a flailing motion of the upper extremity during the collision phase. This may reflect the lower arm(s) resting on the lap or the arm rest of a door.

Figure 3 on page 7 presents one situation where the right lower arm is initially resting on a front seat passenger lap, then rotates outward ("flails") toward the door. Note that the carpal tunnel does not directly contact any door structures; the same is true of the cubital tunnel. The author considered a variety of flailing motions, including both a "resting on lap" and "resting on arm rest" initial position. The flailing contact point was either the arm rest or the door region above the arm rest. In all cases, there was no direct contact between either the carpal tunnel region or the cubital tunnel region. Therefore, no blunt trauma mechanisms regarding the carpal tunnel or cubital tunnel were established during such door structure contact.

In the discovery process regarding a personal injury case in a rear-end collision, it is important to understand how the claimant was positioned within the vehicle, and (if

Figure 3
Front seat restrained passenger right lower arm initially resting on the lap (left). The arm then rotates outward ("flails")
toward the door (right). There is no direct contact to either the carpal tunnel or cubital tunnel.

the driver) what the hand positioning was on the steering wheel, extent to which the arms are bent prior to the impact, and body movements during the impact phase, noting any claimed body contacts with vehicle interior surfaces. One should also understand the occupational history of the claimant. Medical risk factors (such as obesity or diabetes) would normally be addressed by the independent medical examination physician, who would also address any prior medical history issues of relevance.

Case study

A 1994 Mercury Sable was driven by a female claimant that was rear-ended while the vehicle was stopped at a stop sign; her vehicle was first in line. The rear-ending vehicle, a 2013 GMC Terrain, was initially stopped behind the Mercury Sable when the driver took her foot off the gas, causing the GMC Terrain to move forward and contact the rear of the claimant's vehicle. No passengers were present within either vehicle. The plaintiff claimed carpal tunnel syndrome and ulnar nerve entrapment at the elbow, both of which required surgery. She testified that her hands were initially on the steering wheel

(approximately 9 and 3 o'clock positions) and may have come off the steering wheel during the impact. She also testified that her left elbow hit the driver's door. Otherwise, there were no body-interior surface contacts. Through demonstration, she described that her elbows were at 1,200 to 1,300 (1,800 would be straight arms) at the time of impact. An accident reconstruction provided a delta-v to the plaintiff's vehicle of 4.3 mph + 0.3 mph, with an average acceleration of 1.6 g + 0.13 g and no vehicle rotation (minimal offset impact). The plaintiff was 42 years old at the time, 5 feet 8 inches tall, and weighed 361 pounds. She had worked in the food service area for the previous 20 years. **Figure 4** on the page 8 presents two views of an exemplar 1994 Mercury Sable along with a close-up of the vehicle steering wheel.

The defense expert opined as follows: details on the accident reconstruction as described above; the plaintiff's body would have moved backward into the seat, followed by a slight rebound or forward motion; and no mechanisms for medial or ulnar nerve entrapment injuries of the type claimed were established during the collision. There would

Figure 4
Two views of the interior of an exemplar 1994 Mercury Sable along with a close-up view of the steering wheel.

be no significant tension loading or "stretch" of either the medial or ulnar nerves, especially if hands-steering wheel contact is not maintained. Any wrist or elbow motions that occurred would have been within normal range of motion for the two joints. There would be no significant compressive loading of the carpal tunnel region at any point. Any impact of the left elbow with the driver's door would have been at a lateral location of the elbow.

Since the cubital tunnel is located medial to the elbow, there would be no direct blunt trauma to the cubital tunnel region. Finally, the overall forces experienced by the occupant were comparable to the forces associated with so-called Activities of Daily Living (ADL). The latter references to ADLs was more to assist the jury in understanding what 1 g, 2 g, 3 g, etc means; it was not the opinion of the expert that any ADL modeled a rear-end collision (see Lee 2012; Lee 2014).

The independent medical exam (IME) physician noted that the plaintiff was morbidly obese and suffered from diabetes — both significant risk factors for peripheral nerve entrapment neuropathies. The IME physician also noted that the plaintiff had presented for treatment for the same symptoms at least 12 times since 1997 prior to this vehicular collision. While testifying in court, the plaintiff stated that she did not feel that the collision of interest aggravated in any way any prior carpal tunnel or elbow entrapment-related issues. In fact, she testified that she was not experiencing any such issues prior to the incident.

This case resulted in a defense verdict. The jury found there was no causation regarding the claimed injuries and the vehicle collision of interest.

Conclusions
This study focused on the relationship between rear-

end collisions and claimed median nerve entrapment (CTS) and ulnar nerve entrapment at the elbow (CT). Claims of such injuries in relation to low-velocity rear-end collisions are not unusual and are often contested by the defense, acknowledging that there is no obvious relationship between the collision and the claimed injuries. Of the collision types (frontal, side, rear-end), from a biomechanics viewpoint, it is the least clear how a rear-end collision can establish mechanisms for such injuries.

Direct blunt trauma to the carpal tunnel region or the cubital tunnel region is unlikely in a rear-end collision. The occupant is initially moving backward into the seat, away from possible surfaces for body contact. Also, so-called "stretch" injuries due to hypermotion of either the wrist or elbow are unlikely, reflecting occupant kinematics, vehicle interior geometry, and other factors such as the low forces involved in such a collision. While the forensics engineer can address the presence or lack of such mechanisms (general causation), a physician may assign specific causation to a past injury event that established prior damage to the area of interest (for example, wrist or elbow fractures) or a consequence of the presence of significant risk factors such as obesity or diabetes. Certain occupations (for example, high repetition/high force) may also provide a significant contribution.

References
Ames EL. Carpal tunnel syndrome and motor vehicle accidents. JAOA. 1996;96(4):223-226.

Assmus H, Antoniadis G, and Bischoff C. Carpal and cubital tunnel and other, rarer nerve compression syndromes. Dtsch Arzteb Int. 2015;112:14-26.

Armstrong T, Dale AM, Franzblau A, and Evanoff BA. Risk factors for carpal tunnel syndrome and

median neuropathy in a working population. JOEM 2008;50(12):1355-1364.

Atalay NS, Sarsan A. Akkaya N, Yildiz N, and Topuz O. The impact of disease severity in carpal tunnel syndrome on grip strength, pinch strength, fine motor skill and depression. J Phys Ther Sci. 2011;23:115-118.

Baker NA, Moehling, KK, Desai AR, and Gustafson NP. Effect of carpal tunnel syndrome on grip and pinch strength compared with sex- and age-matched normative data. Arthritis Care & Res. 2013;65(12):2041-2045.

Bickel K.D. Carpal tunnel syndrome. J. Hand Surgery. 2010;35(1):147-152.

Bruno A, Toney-Bolger M, George J, Koller J, Filatov A, and Olberding J. Evaluation of occupant kinematics in low-speed frontal and rear-end motor vehicle collisions. SAE Technical Paper 2019-01-1226.

Chapleau J. et al. Demographic and anthropometric factors affecting elbow range of motion in healthy adults. J Shoulder Elbow Surg. 2013;22:88-93.

Chung KC and Spilson SV. The frequency and epidemiology of hand and forearm fractures in the United States. J Hand Surg Am. 2001;26(5):908-915.

Coert JH and Dellon AL. Peripheral nerve entrapment caused by motor vehicle crashes. J Trauma. 1994;37(2):191-194.

Cutts S. Cubital tunnel syndrome. Postgrad. Med. J. 2007;83:28-31.

Dale AM. et al. General population job exposure matrix applied to a pooled study of prevalent carpal tunnel syndrome. Am J Epidem. 2015;181(6): 431-439.

Descatha, Al, Leclerc, A, Chastang, JF, and Roquelaure, Y. Incidence of ulnar nerve entrapment at the elbow in repetitive work. Scan J Work Environ Health. 2004;30(3):234-240.

Ford DJ. Acute carpal tunnel syndrome – complications of delayed decompression. J Bone Joint Surg. 1986;68-B(5):758-759.

Furbish C, Ivory M, Hoffman M, Anderson R, and

Anderson R. Steering column loads and upper extremity motions during low speed rear-end collisions. SAE publication 2011-01-0275.

Gillig JD, White SD and Rachel JN. Acute carpal tunnel syndrome. A review of current literature. Orthop Clin North Am. 2016;47(3):599-607.

Goss BC and Agee JM. Dynamics of intracarpal tunnel pressure in patients with carpal tunnel syndrome. J Hand Surg. 2010;35A:197-206.

Guyton MA and Honet JC. Carpal tunnel syndrome or trigger finger associated with neck injury in automobile accidents. Arch Phys Med Rehabil. 1977;58(7):325-327.

Hagberg M, Morgenstern J, and Kelsh M. Impact of occupations and job tasks on the prevalence of carpal tunnel syndrome. Scand J Work Environ Health. 1992;18: 337-345.

Haas DC and Nord SG. Carpal tunnel syndrome following automobile collisions. Arch Phys Med Rehabil. 1981;62:204-206.

Howard RP, Harding RM and Krenric SW. The biomechanics of "whiplash" in low velocity collisions. SAE publication 1999-01-3235.

Ibrahim I, Khan WS, Goddard N. and Smitham P. Carpal tunnel syndrome: a review of the recent literature. Open Ortho. J. 2012;6:69-76.

Jhattu H, Klaassen S. Ying C, and Hussain MA. Acute carpal tunnel syndrome in trauma. Eur J Plast Surg. 2012;35:639-646.

Keir PJ, Bach JM, and Rempel DM. Guidelines for wrist posture based on carpal tunnel pressure thresholds. Human Factors 2007;49:88-99.

Latinovic R, Gulliford MC and Hughes RA Incidence of common compressive neuropathies in primary care. J Neurol Neurosurg Psychiatry. 2006;77:263-265.

Lee WE III. Target vehicle occupant body motion reflex in anticipated rear-end collisions. 44th Ann Proc AAAM. 2000; 517-519.

Lee WE III. Forensic engineering analysis in injury

event reconstruction and causation analysis: references to activities of daily living. J Nat Acad Forensic Eng. 2012;29(2):31-41.

Lee WE III. Subject ability to characterize g's in relation to activities of daily living. Paper presented at the Biomedical Engineering Society Annual Meeting, San Antonio, Texas, October 22-24, 2014.

Li YL et al. Review of literature of radial nerve injuries associated with humeral fractures – an integrated management strategy. Plos One. 2013;8(11):e78576. doi:10.1371/journal.pone0078576.

Lundborg G et al. Median nerve compression in the carpal tunnel: the functional response to experimentally induced controlled pressure. J Hand Surg. 1982;7(3):252-259.

McConnell WE, Howard RP, Van Poppel J, Krause R, Guzman HM, Bomar JB, Raddin JH, Benedict JV and Hatsell CP. Human head and neck kinematics after low velocity rear-end impacts – understanding "whiplash". SAE publication 952724 (1995).

Melhorn JM, Wilkinson LK, and Seaman SM. Causation: an analysis of carpal tunnel syndrome and motor vehicle accidents. Current Orthop Practice. 2009;20(2):184-190.

Mireles MC, Miller JA, and Paske WC. Misdiagnosis of carpal tunnel syndrome – A systematic misclassification or error of omission. J. Clin. Eng. 2009;34(2):99-102.

Nainzadeh NK, Ilizarov S, and Piligian G. Ulnar neuropathy at the elbow in computer keyboard operators. Work 2011;39:93-101.

Norstrom DL, DeStefano F, Vierkant RA, and Layde EM. Incidence of diagnosed carpal tunnel syndrome in a general population. Epidemiology 1998;9(3):342-345.

Open Stax College – Anatomy & Physiology, Connexions Web site: http://cnx.org/content/col11496/1.6/ June 19, 20-13. CC by 3.0.

Osel DA, Broves AP, Bommarita K, and Ray WZ. Cubital tunnel syndrome: incidence and demographics in a national administrative database. Neurosurgery.

2017;80(3):417-420.

Pourmemari MH and Shiri R. Diabetes as a risk factor for carpal tunnel syndrome – a systematic review and meta-analysis. Diabetic Med. 2016;3:10-16.

Rydevik B. Lundborg G and Bagge U. Effects of graded compression on intraneural blood flow. An in vivo study on rabbit tibial nerve. J Hand Surg. 1981;6(1):3-12.

Rydevik B, Lundborg G and Skalak R. Biomechanics of peripheral nerves. In: Basic biomechanics of the musculoskeletal system (2nd ed.), M Nordin and VH Frankel eds., Lea & Febiger, 1989.

Sarrafian SK, Melamed, JL, and Goshgarian GM. Study of wrist motion in flexion and extension. Clin. Orthop. Relat Res. 1977;126: 153-159.

Schuenke M, Schulte E. and Schumacher U. Thieme atlas of anatomy: general anatomy and musculoskeletal system. New York: Thieme, 2006.

Shiri R and Falah-Hassani K. Computer use and carpal tunnel syndrome: a meta-analysis. J Neurological Sciences. 2015;349:15-19.

Shiri R, Pourmemari MH, Falah-Hassani K. and Viikari-Juntura E. The effects of excess body mass on the risk of carpal tunnel syndrome: a meta-analysis of 58 studies. Obesity Rev. 2015;16:1094-1104.

Siegmund GP. Head/neck kinematic response of human subjects in low-speed, rear-end collisions. SAE Trans. 1997;106(6):3877-3905.

Stuchin, S. Biomechanics of the wrist. In: Basic biomechanics of the musculoskeletal system (2nd ed.), M Nordin and VH Frankel eds., Lea & Febiger, 1989.

Szabo RM. Carpal tunnel syndrome as a repetitive motion disorder. Clin. Orthop. Related Res. 1998;351:78-89.

Viano DC, Parenteau CS, and Burnet R. Rebound after rear impacts. Traffic Injury Prev. 2013;14(2):181-187.

Wehbe MA, Schlegal, JM. Nerve gliding exercises for thoracic outlet syndrome. Hand Clin. 2004;20(1):51-55.

Wiechel J, Morr D and MacDowell A. Review of vehicle impact induced carpal tunnel injury mechanisms. J. Biomech. 2006;39(Suppl 1):S162.

Witt JC and Stevens JC. Neurologic disorders masquerading as carpal tunnel syndrome: 12 cases of failed carpal tunnel release. Mayo Clin. Proc. 2000;75:409-413.

Wojewnik B and Bindra R. Cubital tunnel syndrome – review of current literature on causes, diagnosis and treatment. J Hand Microsurg 2009;1(2):76-81.

Zuckerman JD and Matsen FA III. Biomechanics of the elbow. In: Basic biomechanics of the musculoskeletal system (2nd ed.), M Nordin and VH Frankel eds., Lea & Febiger, 1989.