



Pathology/Biology Section - 2014

G84 Investigation of a Disputed Mechanism of Diffuse Axonal Injury Following a Low-Speed Frontal Crash

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The goal of this presentation is to describe an unusual mechanism of a serious brain injury associated with a relatively low-speed frontal crash with an airbag deployment.

This presentation will impact the forensic science community by demonstrating a methodology for investigating an unusual injury mechanism.

Diffuse Axonal Injury (DAI) is a type of widespread traumatic brain injury that is typically associated with higher speed traffic crashes, falls from height, and assaults. The injury consists of shearing of axons in the brain that is associated with a high degree of angular acceleration (rotational forces).

This discussion concerns a single vehicle traffic crash involving a 36-year-old Asian male who was the unrestrained right front passenger of a 1994 Ford® Mustang®. For an unknown reason, the vehicle crossed over the opposite lane, impacted the curb adjoining the roadway, and subsequently struck several small trees on the west side of the sidewalk, coming to a complete stop. The impact speed was estimated to be less than 15mph and there was driver- and passenger-side front airbag deployment. The airbag deployed either during the impact with the curb or upon impact with the thin row of trees just beyond the sidewalk.

The front seat passenger was found unresponsive and was seated in the passenger's seat with his upper torso and head hanging outside the passenger door when police arrived. The passenger was bleeding copiously from a >10cm laceration over the left posterior aspect of his scalp. The windshield had a classic "spider" fracture on the passenger side, near the top of the windshield where it met the frame, and was bulging outward at the junction of the upper edge of the windshield and windshield frame. The victim was transported to the hospital, where he was diagnosed with a moderate to severe DAI. He was ultimately left with permanent and severe injuries.

An investigation by the insurer resulted in a denial of a claim for the injury based on the following assertion: because the location of the injury to the victim's head was posterior, and the kinematics of a frontal collision would typically involve interaction between the face and top of the head of a front seat passenger and the airbag, and possibly the dashboard and windshield, but *not* the posterior aspect of the head, it was *impossible* for the injury to have resulted from the crash. The alternative, therefore, was that the injury was caused by an assault that preceded the crash, for which there was no evidence. It was further asserted that, based on experimental primate studies, the speed change of the collision (<10mph) was insufficient to cause the injury.

In the ensuing investigation, it was discovered that there was a significant witness mark inside the vehicle at the top rail above the windshield, indicating a forceful vertically oriented loading of the top rail from below. A review of the CT scan taken on the day of the injury revealed a large laceration that was consistent with a high-velocity tangential load at the left posterior aspect of the head, resulting in a tearing of the scalp forward. Swelling over the right cheek was also documented.

The occupant kinematics and injury mechanism that best explained the evidence were reconstructed as follows: when the vehicle struck the curb, the victim was likely thrown forward and into the dashboard, but the airbag did not deploy until approximately one-half second later, when the vehicle struck the small trees. The deploying airbag then impacted the right side of the victim's face, and propelled his head and torso upward and into the windshield header, resulting in the tangential load that was high enough to deform the metal windshield frame, tear the victim's left posterior scalp, and induce rotational forces sufficient to produce the moderate-severe DAI. Not only was the explanation most consistent with the facts, it was the only plausible explanation for the vehicle damage and witness marks, as no other known mechanism existed to produce the observed damage to the interior of the vehicle. Although an unusual injury mechanism, it was not an impossible or improbable explanation for the evidence, and the assertion that the injury *must* have resulted from an alternative mechanism was rejected.

Diffuse Axonal Injury, Biomechanics, Forensic Epidemiology