# Injury Biomechanics of the Cervical Spine in Car Collisions - Some needs for further research

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**Background**

The incidence of soft tissue neck injuries in car collisions has been on the increase during the last two decades and these injuries cause enormous costs to the society and the insurance business today. This is the reason behind the intensified research efforts and the great number of important publications in this field in recent years. Youganandan and Pintar (2000a) present a condensed overview of the latest development in the field.

Accident data have been collected in a number of studies and we are beginning to get a clearer view of the injury incidence as a function of various parameters. The injury risk appears to be the greatest in rear-end collisions but frontal collisions as well as side collisions stand for a significant portion of these injuries (Krafft, 2000; Temming and Zobel, 1998; Jakobsson et al., 2000b). The neck injury symptoms appear to be very similar for all impact directions (Minton et al., 2000). The injury risk differs between seating positions. In rear-end collisions the rear seat generally is safer (Lövsund et al., 1988; Jakobsson et al., 2000a). The risk of initial neck symptoms increases with velocity change (delta-v) but among those initially injured the acceleration pulse appears to be more important than the delta-v as a predictor of risk of long term consequences (Krafft, 2000; Kullgren et al., 2000). The neck injury risk differs significantly between different car models of similar size in rear impacts (Krafft, 2000). Taller drivers have an increased injury risk in rear impacts (Jakobsson, 2000) and the injury risk increases if the vehicle occupant is unaware of the impending accident (Ryan et al., 1994).

A number of experimental studies on volunteers and post mortem human subjects have been reported. There is a relatively good view of the overall body kinematics in different crash directions. The typical neck loading in a car accident is caused by the acceleration of the torso resulting in a neck bending motion illustrated in Figure 1. The thoracic spine normally undergoes some type of bending motion in this type of event. In rear end collisions the thoracic kyphosis is straightened resulting in an elevation and a rearward tilt of the T1 vertebra (Davidsson et al., 1998; van den Kroonenberg, 1998; Ono et al., 2000). Several studies have focused also on the detailed motion of the cervical spinal segments during rear-end impact loading (Ono et al., 1997; Panjabi et al., 1999; Winkelstein et al., 1999; Youganandan and Pintar, 2000b; Deng et al., 2000). The intervertebral motion appears to deviate from normal physiologic human neck bending motion.

Two new crash dummy prototypes for rear-end collision testing have been presented, BioRID (Davidsson, 1999; Davidsson, 2000; Davidsson et al., 1999) and RID2 (Cappon et al., 2000). The dummies enable more detailed assessment of the human body kinematics in rear end impacts. This will for instance improve the prediction of the contact conditions between the head and the head-restraint. In the future, with this type of dummy and with appropriate dummy instrumentation, it will hopefully be possible to measure injury criteria that correlate to neck injury risk.

Soft tissue injuries have been found in several different structures and locations in the neck region in experimental studies and autopsy studies. In a recent study Yoganandan et al. (2000) reported injuries to several ligaments, the intervertebral discs and the facet joint structures. Siegmund and Brault (2000) and Brault et al. (2000) presented indications of muscle injury due to eccentric muscle loading in the early phase of the neck motion in rear impacts. Taylor et al. (1998) reported interstitial haemorrhage in cervical dorsal root ganglia in an autopsy study of victims who had sustained severe inertial neck loading during impacts to the torso or to the head. The structures around the ganglia were mostly uninjured. These findings correlate to experimental findings in pigs of nerve cell membrane dysfunction in cervical spinal root ganglia reported by Svensson et al. (2000).

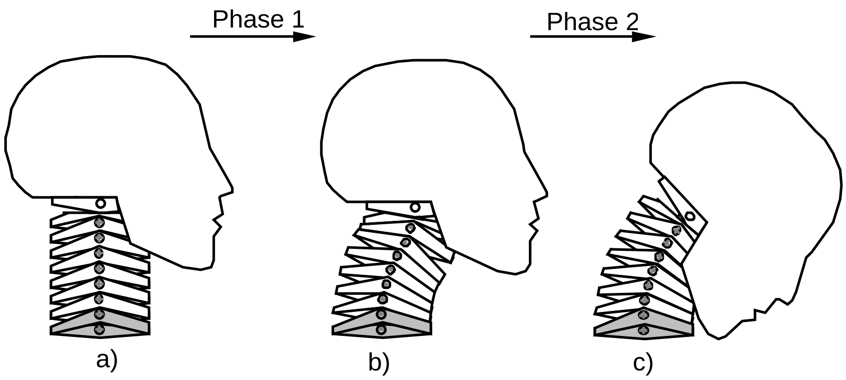

Figure 1a: Schematic drawing of the head-neck motion during a frontal collision.
Phase 1: Protraction motion
Phase 2: Flexion motion

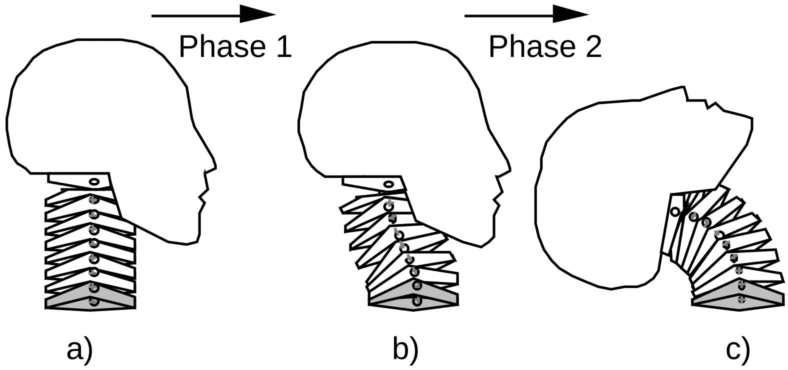

Figure 1b: Schematic drawing of the head-neck motion during a rear-end collision.
Phase 1: Retraction motion
Phase 2: Extension motion

Several neck injury mechanisms and neck injury criteria have been proposed during recent years. Two criteria, Nij (Kleinberger et al., 1998) and Nkm (Muser et al., 2000), use combinations of neck loads to predict the risk of injury to the skeletal spine. The IV-NIC (Panjabi et al., 1999) uses the angular displacement between adjacent vertebrae to estimate the risk of injury to various structures of the intervertebral joints. The correlation between these three injury criteria and the risk of long term soft tissue neck injury has not (to the author's knowledge) yet been established. The Neck Injury Criterion (NIC) (Boström et al., 2000) uses differential horizontal acceleration between the head and the T1 vertebra to assess the neck injury risk. The NIC was initially based on experimental injury findings summarised by Svensson et al. (2000). NIC would also function as a predictor of other types of injury mechanisms and indications of correlation between NIC and long term neck injury risk have been presented (Boström et al., 2000).

The injury symptoms have been described in a large number of papers (e.g. Sturzenegger et al., 1995; Spitzer et al., 1995). It is important to distinguish between initial symptoms and long term symptoms (Krafft, 2000). Long term (chronic) whiplash symptoms appear to be associated with central pain sensitisation (Sheather-Reid and Cohen, 1998; Johansen et al., 1999). The exact origin of this pain sensitisation has not been established. Successful treatment methods could possibly provide a clue. Byrn et al. (1993) reported significantly reduced symptoms during a time period after sub-cutanious sterile water injections on the back of the neck. Bogduk (2000) reported pain relief in about 50 percent of the patients after coagulation of the small nerves that innervate the facet joint that is associated with the painful dermatome.

**Synthesis**

It appears likely that several types of neck injury may appear as a result of a whiplash trauma (muscles, ligaments, facet joint, discs, nerve tissue etc.). Several injury types may be present in the same patient at the same time. The relation between these possible injuries and the large set of known whiplash symptoms (neck pain, head ache, shoulder pain, neurological symptoms etc.) is unclear. It would be of particular interest to know which one (ones) of these injuries that would result in long term symptoms and central pain sensitisation. It would then also be of interest to know which injury mechanism is responsible for this particular injury.

At the initial symptom stage, arm pain and high symptom intensity seem to correlate to an increased risk of long term consequences (Sturzenegger, 1995; Karlsson et al., 2000). The apparent influence of the crash pulse on the risk of long term consequences in patients with initial symptoms (Krafft, 2000) indicate that there could be a separate injury and a separate injury mechanism behind the long term symptoms. This particular injury could in the acute stage often co-exist with other injuries that normally heal without causing residual pain. Sturzenegger et al. (1995) found a higher risk of long term symptoms in those patients that were injured in a rear end collision and this may indicate that one particular injury (which may cause long term symptoms) is more likely to occur in a rear impact.

In more peripheral parts of the body most of these injury types (tissue types) normally recover without long term pain and central pain sensitisation. Is there something special about the neck region that makes one or several of these injuries result in long term pain? Cavanaugh (2000) for instance, explained that the facet joint capsules are particularly rich in nerve endings why an injury at this point would be a likely reason for long lasting pain. This pain may cause referred pain in e.g. the shoulder region. Facet joint capsule strain and pinching has been shown in post mortem human subjects in rear impact testing (Yoganandan and Pintar, 2000b; Deng et al., 2000). It is however not known whether the same type of mechanisms may occur also in side impacts and frontal impacts. Is there some type of structure that is unique for the neck? The spinal nerve root ganglia would be an example of such a structure. Cavanaugh (2000) explained that injury to the dorsal root ganglia is likely to cause radiating pain to dermatomes of for instance the shoulders and the arms. These symptoms are, as mentioned earlier, known to correlate to increased risk of long term consequences. Cervical dorsal root ganglion injuries have been observed in various impact directions (Svensson et al., 2000, Taylor et al., 1998) and would explain the similarity in symptoms between different impact directions.

It can be concluded that we have a lot of exciting research work ahead of us before we get a complete understanding of the whiplash problem. But once we have this understanding, we will have a much better chance of developing accurate diagnosis methods, effective treatment and good preventive measures in vehicles.

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