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Neck injuries in car collisions — a review covering a possible injury mechanism and the development of a new rear-impact dummy

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Abstract

A review of a few Swedish research projects on soft tissue neck injuries in car collisions is presented together with some new results. Efforts to determine neck injury mechanisms was based on a hypothesis stating that injuries to the nerve root region in the cervical spine are a result of transient pressure gradients in the spinal canal during rapid neck bending. In experimental neck trauma research on animals, pressure gradients were observed and indications of nerve cell membrane dysfunction were found in the cervical spinal ganglia. The experiments covered neck extension, flexion and lateral bending. A theoretical model in which fluid flow was predicted to cause the transient pressure gradients was developed and a neck injury criterion based on Navier-Stokes Equations was applied on the flow model. The theory behind the Neck Injury Criterion indicates that the neck injury occurs early on in the rearward motion of the head relative to the torso in a rear-end collision. Thus the relative horizontal acceleration and velocity between the head and the torso should be restricted during the early head-neck motion to avoid neck injury. A Bio-fidelic Rear Impact Dummy (BioRID) was developed in several steps and validated against volunteer test results. The new dummy was partly based on the Hybrid III dummy. It had a new articulated spine with curvature and range of motion resembling that of a human being. A new crash dummy and a neck injury criterion will be very important components in a future rear-impact crash test procedure.

Keywords: Whiplash; Neck injury; Rear impact; Crash dummy; Spinal ganglia; Pig

1. Introduction

The symptoms of injury following neck trauma in rear-end collisions include pain, weakness or abnormal responses in the parts of the body (mainly the neck, shoulders and upper back) that are connected to the central nervous system via the cervical nerve-roots. Vision disorder, dizziness, headaches, unconsciousness, and neurological symptoms in the upper extremities are other symptoms that have been reported (Deans et al., 1987; Hildingsson, 1991; Nygren et al., 1985; Spitzer et al., 1995; Watkinson et al., 1991). The symptoms associated with soft-tissue neck injuries in frontal and side collisions appear to be very similar to those of rear-end collisions (Hildingsson, 1991).

During a rear-end car collision the struck vehicle is subjected to a forceful forward acceleration and the car occupant is pushed forward by the seat-back. The head lags behind due to its inertia, forcing the neck into a swift extension motion. In a later phase, the head moves forward relative to the torso and may stop with a somewhat flexed neck posture. This head and neck motion, commonly called 'whiplash motion', has been described by Ono and Kanno (1993) among others. The term 'whiplash' has also been used in the literature for the neck motion in frontal and side collisions.

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According to Svensson (1993) a synthesis of the findings by Mertz and Patrick (1967, 1971) and by McConnell et al. (1993) indicates that soft tissue neck injuries are prevented in a rear-end impact if the displacement between the head and the torso is avoided. But injury may occur in a rear-end impact even if the neck does not exceed the natural range of rearward angular head motion.

In frontal collisions, the neck usually experiences the same type of inertial loading from the head as it does in rear-end collisions. During the initial phase of these neck-loading situations, the head normally undergoes a horizontal translational displacement relative to the torso. This induces neck protraction motion in frontal collisions (Wismans and Spenny, 1984) and neck retraction motion in rear-end collisions (Eichberger et al., 1996; Geigl et al., 1995). The neck is exposed to significant mechanical loads when the end of the natural range of protraction or retraction of the neck is reached (1b2b) and neck injuries may well occur at this point (Deng, 1989). This may be one explanation why modern head-restraints do not provide better neck protection. They may simply come into play too late, after the neck has exceeded the maximum range of retraction motion and gone into extension.

Currently there is no adequate tool for testing the performance of car seats and head-restraints in rear impacts. The best available dummy is the Hybrid III. The neck and spinal structure of this dummy are stiff and unlikely to interact with the seat-back in the same compliant way as the human spine. Foret-Bruno et al. (1991) concluded that the human head can be moved relative to the torso with very limited stresses in the neck, but this is not the case for the Hybrid III. Scott et al. (1993), found that the human subject's torso appeared to ramp up the seat back while that of the Hybrid III did not.

Svensson and Lövsund (1992) developed and validated a Rear Impact Dummy-neck (RID-neck) that can be used on the Hybrid III dummy in low-speed rear-end collisions. Thunnissen et al. (1996) developed a new rear-impact dummy neck, the TRID-neck (TNO Rear Impact Dummy-neck) based partly on the RID-neck design. The TRID was subjected to a more extensive validation than the RID-neck, but for both of these necks the validation was restricted to the angular displacement between head and torso. The dynamic responses of the two neck types appear to be very similar.

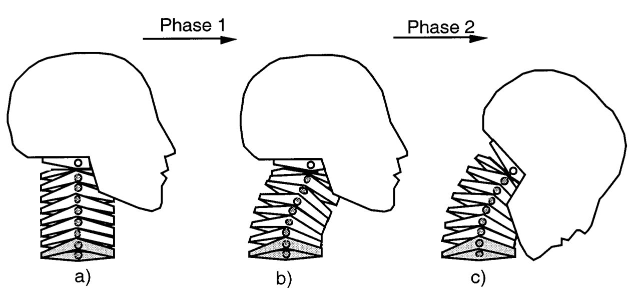

Fig. 1. Schematic drawing of the head-neck motion during a frontal collision. Phase 1: protraction motion. Phase 2: flexion motion

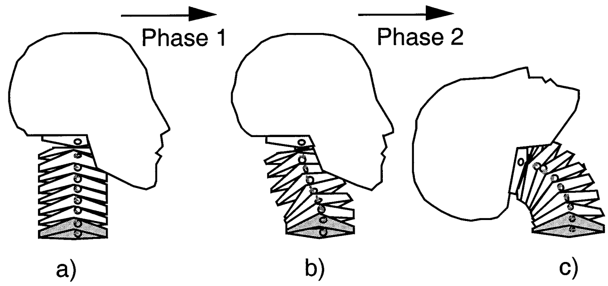

Fig. 2. Schematic drawing of the head-neck motion during a rear-end collision. Phase 1: retraction motion. Phase 2: Extension motion

The strategy of the neck-injury research carried out at Chalmers University of Technology, Göteborg, has been to address the problem of AIS 1 (Abbreviated Injury Scale) neck injuries in car collisions. The focus was originally on rear-end collisions at low impact velocities ($\Delta v < 20$ km/h). One aim was to find the injury mechanism that could explain the various long-lasting symptoms that result from soft tissue neck injuries and to establish how the risk of injury correlates to kinematic and kinetic parameters of the head-neck motion relative to the torso. The work originated from a hypothesis by Aldman (1986) postulating that injury could be induced in the cervical spinal nerve root region as a result of transient pressure gradients during a swift extension-flexion motion of the cervical spine. A second aim of the work was to develop and validate a crash test dummy for evaluation of the protective performance of car seat-systems in rear-end collisions at low impact-velocities.

2. Injury mechanism

2.1. Theoretical injury mechanism model

The inner volume of the cervical spinal canal increases at flexion and decreases at extension of the neck (Breig, 1978). All the tissues and fluids inside the spinal canal are virtually incompressible (Estes and McElhaney, 1970). This means that fluid transportation, to and from the cervical spinal canal, must take place during the flexion–extension motion of the cervical spine to compensate for the volume change. The fluid could be either blood in the venous plexus of the epidural space or cerebro spinal fluid (CSF). Due to the relatively high flow resistance in the subarachnoid space, flow of CSF was thought to be of minor importance compared to vein blood flow in this type of