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 are 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 were initially confined to neck extension trauma, but more recently both forward flexion and lateral bending have been studied with similar findings.

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) has been developed in several steps and validated against volunteer test results. The new dummy is partly based on the Hybrid III dummy. It has 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.

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).

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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 body 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.

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 (Figs. 1b and 2b) 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.

The strategy of the neck-injury research carried out at Chalmers University of Technology, Göteborg, has been to address the problem of AIS 1 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

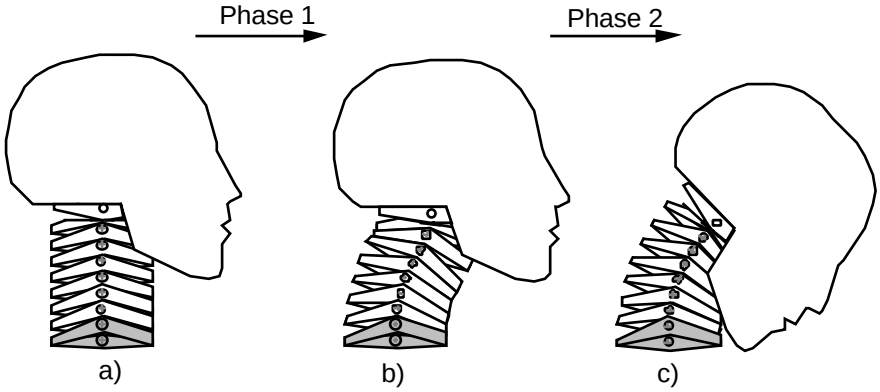

Figure 1: Schematic drawing of the head-neck motion during a frontal collision.
Phase 1: Protraction motion
Phase 2: Flexion motion

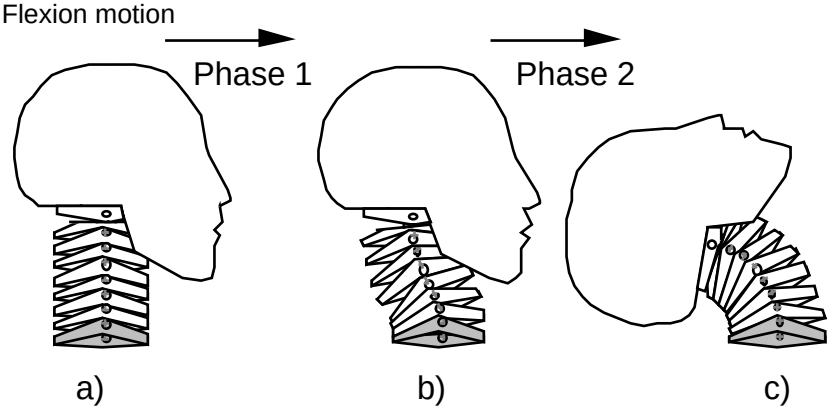

Figure 2: Schematic drawing of the head-neck motion during a rear-end collision.
Phase 1: Retraction motion
Phase 2: Extension motion

INJURY MECHANISM

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 volume compensation (Svensson et al., 1993). Blood volumes in the internal and external vertebral venous plexa, which communicate via vein bridges through the intervertebral foramina, can easily move to compensate for the change in inner volume in the spinal canal during the flexion-extension motion (Svensson et al., 1993).

During swift extension-flexion motion, pressure gradients along the spinal canal as well as across the intervertebral foramina may occur as a result of the blood flow in the vein plexa. Due to flow resistance and the acceleration effect on fluid mass, the pressure gradients may generate injurious stresses and strains to the exposed tissues, particularly in the intervertebral foramina.

Experimental neck trauma

Anaesthetised pigs were used in experiments (Boström et al., 1996; Örtengren et al., 1996; Svensson et al., 1993) approved by the local animal experimentation and ethics committee. A

first group of animals was used to measure pressure in the CNS during simulated rapid neck extension motion or neck flexion motion. A second group was used for histopathological examination. In the second group, some animals were exposed to simulated rapid neck motion and others served as sham-exposed controls. A schematic view of the test set-up for the experimental neck extension/flexion trauma is shown in Figure 3.

The head was pulled either in the posterior, anterior or lateral direction. In some extension-motion test runs a head-restraint was introduced. Gaps of 100-130 mm were used between head and head-restraint. A gap of 100 mm prevented the neck from reaching full retraction, and a gap of 130 mm allowed the neck to pass the point of full retraction but prevented it from reaching the maximum physiological extension angle.

The animals used for the pressure measurement experiments had catheter pressure transducers introduced into the subarachnoid space in the cervical spine. Pressure measurements were taken under various loading conditions. The pull force (Fig. 3) was varied from 150 N to 900 N.

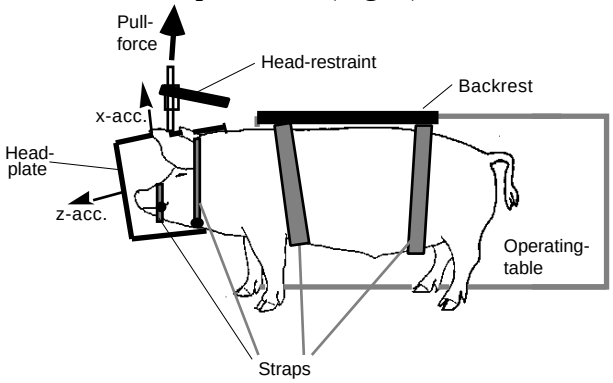

Figure 3: The test set-up seen from above. The anaesthetised animal is lying side down on the operating-table, strapped to the backrest. The head is strapped to the bolts in the horizontally movable head-plate. During the experiment a pre-tensed rubber-strap pulls the head-plate (in posterior or anterior direction) by the pull-rod. The pull force is active until the pull-rod is disconnected, and thereafter the head moves in the sagittal plane due to its inertia. In some tests, a head-restraint was introduced to limit the maximum rearward displacement of the head.

The animals in the group that underwent histopathological examination were given an intra-venous injection of Evans Blue (EB) dye (which normally conjugates to albumin in the blood) before the test. All animals in the histopathology group that underwent experimental neck trauma were exposed to a 600 N pull force. Lateral neck motion was not included for this group at this time. After the test, each animal was sacrificed. The brain and the spinal cord to about the T4 level were dissected. The spinal ganglia and proximal parts of corresponding nerves were identified and isolated (Örtengren et al., 1996). Cryostate microtome sections were prepared and examined in a fluorescence microscope according to a procedure described by Suneson et al. (1987).

The function of the EB is to show the damage sustained to the blood-brain barrier in the CNS. If, upon microscopical examination, EB can be detected outside the blood system, this indicates that the blood vessels have been damaged. Due to the fenestration of the capillaries in the spinal ganglia, however, EB will normally pass into the inter-cellular space, but not into the nerve cells. Thus EB inside the nerve cells indicates dysfunction of the nerve cell membranes and the satellite cells.

Results of the injury mechanism study

Pressure measurement results from one whiplash extension run is shown in Figure 4. The pull-force was 600 N, which is the same as that for the animals in the histopathological examination group. The angular displacements and linear X-displacements of the head, the x- and z-accelerations of the head-plate, and the readings from the three pressure transducers in the CNS are shown (Fig. 4). The onset of the angular motion of the head is delayed about 30 ms

compared to the linear X-displacement, indicating that the head moves mainly translationally during the first 30 ms. After about 60 ms the transformation from retraction motion to extension motion (Fig. 2) is completed and the head has reached its maximum rearward angular velocity (Fig. 4a). The general pattern of the pressure pulse in the spinal canal is the same for all degrees of pulling force, but the duration of the pulse becomes shorter and its magnitude higher with increasing pulling force (Svensson et al., 1993). The maximum angular head displacement occurs earlier and increases in magnitude with increasing pulling-force (Svensson et al., 1993).

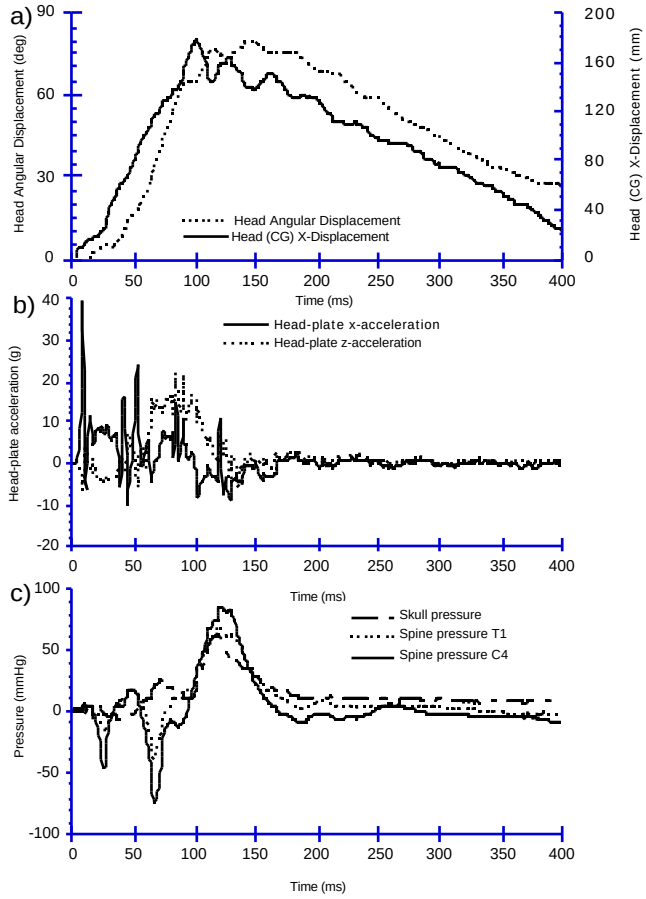

Figure 4: The results from one whiplash extension run with pressure measurements (25). The applied pull-force on the head-plate was 600 N.
a) Angular displacement and the linear X-displacement of the head CG (Centre of Gravity) versus time.
b) Accelerations of the head-plate versus time.
c) The pressure versus time in the CNS at three levels: skull, C4, and T1.

A comparison of the pressure readings in the spinal canal at C4 level between a test without a head-restraint (PW 03.04) and a test with a head-restraint positioned 100 mm behind the head (PW 03.03) are shown in Figure 5. The pressure pulse is drastically reduced after head to head-restraint contact at about 60 ms in test PW 03.03 (Fig. 5). With a 130 mm head-restraint gap the contact would occur at about 80 ms, which means that the deep pressure dip at about 70 ms would not be avoided.

The pressure readings from an experimental neck flexion trauma test are shown in Figure 6. The pull force was 300N and the magnitude of the pressure dip is in the same order as for an experimental neck extension trauma at the same pull force level (Svensson et al., 1993). Pressure readings from a lateral flexion experiment using 600 N pull force are shown in Figure 7. The results are very similar to those of a neck extension experiment (Fig. 4).

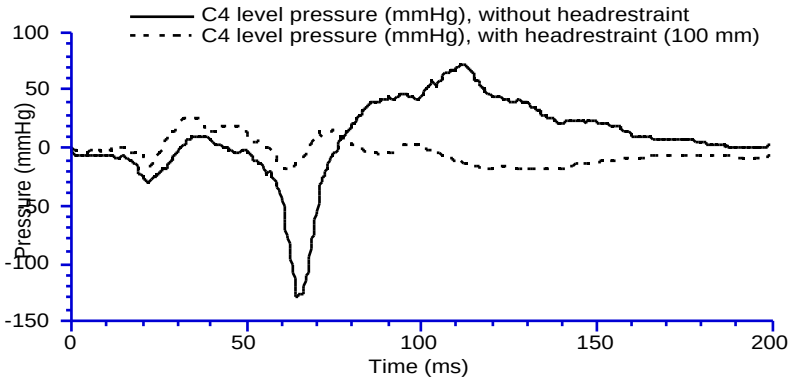

Figure 5: The pressures inside the spinal canal at the C4 level, with and without head-restraint, during swift extension motion at a pull force of 600N (3).

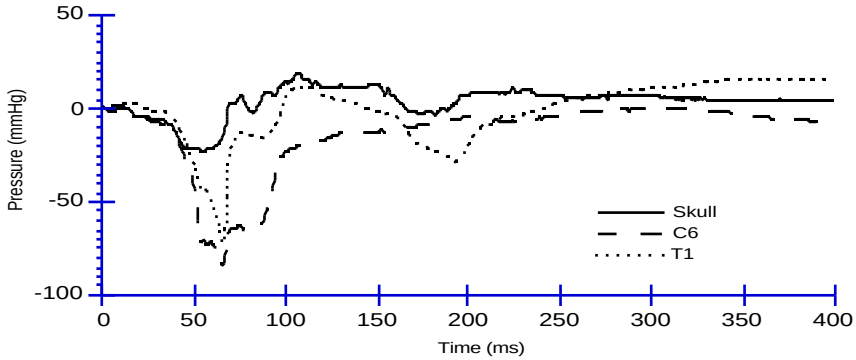

Figure 6: The pressures inside the skull and in the spinal canal at the C6 and T1 levels during swift flexion motion at a pull force of 300N (25).

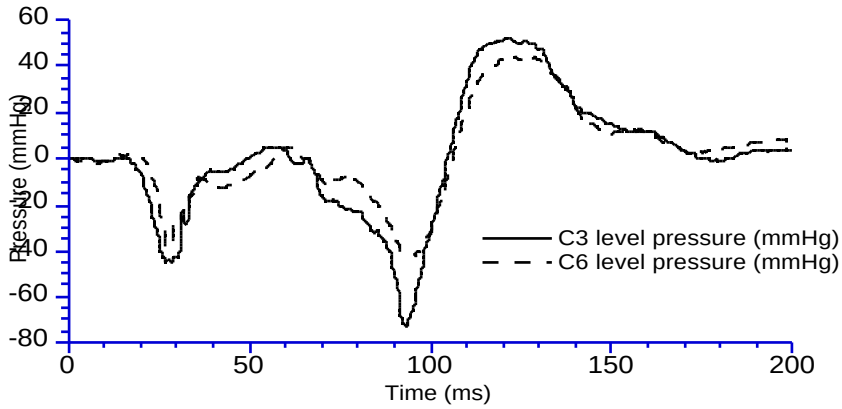

Figure 7: The pressure inside the spinal canal at the C3 and C6 levels during swift lateral flexion motion of the neck at a pull force of 600 N.

Macroscopical inspection during the autopsies of the animals exposed to trauma revealed no bleeding or fractures of vertebral structures, or ruptures of ligaments (Örtengren et al., 1996). However, fluorescence microscopic examination of the satellite cells and nerve cells in the spinal ganglia of the neck-extension trauma exposed animals (without head-restraint) disclosed red fluorescent material, indicating EBA leakage and thus membrane dysfunction. These findings were most obvious at the C6 - C8 levels. There was no sign of such leakage into the satellite cells or the nerve cells in the spinal ganglia from the sham-exposed animals (Örtengren et al., 1996).

In the tests with a head-restraint in place, there was no sign of EBA leakage at a 100 mm head-restraint gap, but for tests with 130 mm head-restraint gap, the frequency of leakage was the

same as in the animals where no head-restraint was used (Boström et al., 1996). Preliminary results indicate that animals exposed to neck flexion trauma had a similar frequency of EBA leakage as the neck-extension trauma exposed animals without head-restraint (Boström et al., 1996).

Static loading of the cervical spine under loading conditions resembling those caused by a posterior pull force of 600 N did not result in any pressure gradients or nerve cell membrane dysfunction (Boström et al., 1996).

A mathematical model and a neck injury criterion

A one-dimensional mathematical model of the flow and pressure phenomena in the spinal canal was developed (Boström et al., 1996). The model is based on the Navier Stokes equations and is built on the assumption that the flow along the cervical spinal canal is the most significant component causing the pressure gradients. According to the mathematical model, the two distinct pressure dips at 25 ms and 60 ms in Figure 4 are caused by a water-hammer effect as the flow along the spinal canal swiftly alters direction when the cervical spine changes its bending mode. The model predicts the magnitude of the negative pressure dip that occurs during a whiplash extension exposure at about the time of maximum neck retraction (after about 60 ms in Figure 4). Since the ganglion injuries were avoided when this pressure dip was excluded, by means of a head support (Fig. 5) or during static loading, it was assumed that the magnitude of the dip corresponded to the risk of injury. The model predicts the risk of injury in a human being according to equation [1] where NIC stands for Neck Injury Criterion, a_{rel} is the relative horizontal acceleration between T1 and the occipital joint, and v_{rel} is the relative horizontal velocity between T1 and the occipital joint.

[1] $$NIC = 0.2a_{rel}+v_{rel}^2 \quad \text{(calculated at full retraction)}$$

A preliminary estimation yielded a NIC value that should not exceed 15 m^2/s^2 if injuries with long-term consequences are to be avoided. The estimation is based on the scaling of the pig anatomy to that of the human and comparing with results from volunteer tests (Boström et al., 1996). The validity of the NIC was further supported by Boström et al. (1997) and Eichberger et al. (1998).

Discussion of the injury mechanism study

The overall anatomy of the cervical spine of the pig is similar to that of the human being even though the dimensions and the detailed shapes of different tissues differ somewhat between the two species. The spine and head of the pig serve as a qualitative substitute of the corresponding parts of the human body and have served as guidance in terms of what kinematic and kinetic parameters are related to the risk of injury. Repeatability and reproducibility was found to be adequate in the test set-up, and the loading conditions and time history of the neck trauma experiments were considered relevant (Svensson, 1993).

A key finding for the group of animals used in the histopathological examination was the observation that the spinal ganglia from whiplash-exposed animals showed an increased frequency of EBA-stained nerve cells as compared to the sham-exposed controls. Further, the uptake of EBA-complex within the nerve cell cytoplasm and nucleus was distinct and striking in the whiplash-exposed animals in contrast to the controls.

It is tempting to presume that the pressure gradients induced during the whiplash extension motion (Fig. 4) constitute an important factor in the pathogenesis of the observed change. To verify the relationship between the pressure gradients and the observed change, an experimental set-up would be needed in which a stationary animal is exposed to pressure gradients of the same type as in our whiplash experiments. Olmarker et al. (1989), using a different loading

condition, demonstrated that nerve roots can be damaged by pressures of less than 50 mm Hg, particularly when the onset rate is high. The loading conditions in their study were, however, somewhat different to those of the present work.

Crushing or transection of a peripheral nerve, e.g. the sciatic nerve, results in reactive changes in corresponding spinal ganglia nerve cells (Sunderland, 1991) with an initial loss and subsequent restoration of their afferent input (Woolf et al., 1992). Adaptive as well as aberrant patterns of synaptic connections are established in the deeper laminae in the dorsal horn of the spinal cord concomitant with the regeneration of the injured peripheral nerve. Tentatively, the whiplash-related changes observed in the spinal ganglion neurones could be sufficient to cause similar loss and rebuilding of the afferent synaptic connections within the laminae in the posterior horn of the spinal cord. That could contribute to the exacerbated clinical symptoms reported by patients even weeks postwhiplash injury. This working hypothesis, however, requires further investigation.

Symptoms similar to those incurred during rear-end collisions also occur in patients that have been involved in frontal impacts (Hildingsson, 1991; Larder et al., 1985; Maimaris et al., 1988) though the relative injury risk appears to be smaller in the latter circumstance (Temming and Zobel, 1998). The lower risk in frontal impacts could possibly be explained by the fact that car occupants in frontal collisions usually are aware of the impending impact and brace their neck muscles. This will in turn mitigate the relative motion between the head and the torso thereby reducing the transient pressure gradients in the spinal canal. The posterior neck muscles that resist forward head motion are also stronger compared to the anterior neck muscles and this will further increase the difference in injury risk between frontal and rear-end collisions. Pressure measurements in the CNS during swift experimental flexion motion of the neck revealed only negative pressures (Fig. 6), but these were of similar magnitudes as in the extension motion experiments (Svensson et al., 1993). The positive histopathological findings from experimental swift flexion motion indicate that the negative portion of the pressure readings corresponds to the occurrence of nerve cell membrane dysfunction.

The data presented in Figure 4 clearly indicates that pressure pulses do occur in the pig during this type of motion. Such pressures are not likely to be induced by other mechanisms or to be due to a measurement artefact (Svensson et al., 1993).

In the neck-extension trauma experiments, the negative part of the pressure readings was avoided only when the head-restraint was at the closest distance, 100 mm behind the head. Only at this narrow head-restraint gap were the injuries to the spinal ganglia avoided. This is another indication that the negative part of the pressure readings are responsible for the nerve-cell membrane dysfunction. The findings also indicate that a head-restraint, in order to be effective, must interact with the head motion early on, before the point of maximum neck retraction has been reached.

Conclusions of the injury mechanism study

1) The Aldman-hypothesis (Aldman, 1986) regarding transient pressure gradient during swift neck-bending motion was supported by the pressure recordings during experimental whiplash motion.

2) Spinal ganglion nerve-cell membrane dysfunction was revealed after experimental whiplash trauma. Injury to this region was predicted by the Aldman-hypothesis (Aldman, 1986). These findings could very well explain many of the symptoms that are connected to whiplash associated disorders and that are related to afferent nerves passing through the cervical spinal ganglia.

3) It seems possible that the negative pressure readings observed during experimental whiplash trauma could be the cause of the ganglion injuries. Should this assumption turn out to be correct, the pressure gradient injury mechanism could explain the similarity in symptoms for different crash directions as well as the reason for the poor effectiveness of current head-restraints that are usually placed too far behind the head to prevent the neck from reaching maximum retraction.

4) The NIC indicates that improved neck protection in rear-end car collisions would require that the relative horizontal acceleration and velocity between the head and the torso be kept low or that the head be caught by the head-restraint very early on in the crash event.

REAR IMPACT DUMMY DEVELOPMENT

Dummy design

A new dummy for rear-end collision testing at low velocity changes was developed (Davidsson et al., 1998a) (Figure 8) in a joint project involving Chalmers, Autoliv, Saab Automobile and Volvo Car Corporation. The dummy has been given the name Biofidelic Rear Impact Dummy (BioRID). It has a new torso, arm attachments, articulated spine, neck muscle substitutes and pelvis, to be used with Hybrid III legs, arms and head.

In order to resemble the human spine, the BioRID spine consists of 7 cervical, 12 thoracic and 5 lumbar vertebrae. In seated posture the neck has a lordosis. The thoracic spine has a kyphosis and the lumbar spine is straight to emulate the human in seated posture (Robbins, 1985) (Figure 8). The vertebrae are made of durable plastic and connected with pin joints which only allow for angular motion in the sagittal plane. The spine curvature can be changed thereby, enabling different initial seating postures. The choice of static joint characteristics in the cervical, thoracic and lumbar spine were based on MADYMO simulations (Davidsson et al., 1998a; Linder et al., 1998a).

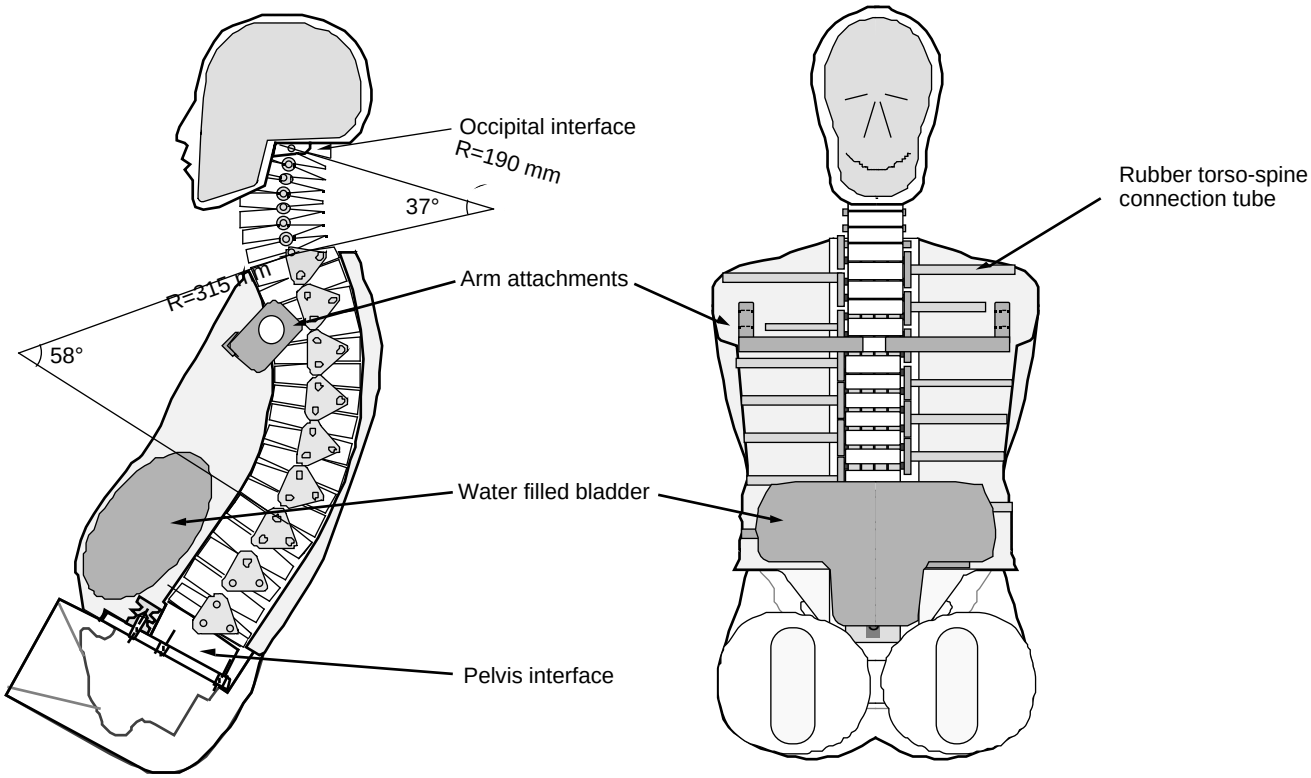

Figure 8: Schematic drawing of the BioRID-dummy torso, arm attachments, spine, neck and modified pelvis with Hybrid III head (Davidsson et al., 1998a).

In order to better replicate the human head and neck retraction motion (head-lag) and thus more precisely predict injury risk, the new neck is equipped with muscle substitutes. These consist of wires originating from the head, in the front and in the back of the occipital joint, and guided through the cervical vertebrae, terminating at the T1. At the T1 the wire load is transferred via nylon coated steel wires and wire housing to a spring in parallel with a damper (Linder et al., 1998b).

The torso consists of chest and abdomen and is moulded in a soft silicon rubber. The torso surface contour resembles a seated 50% male (Robbins, 1985). The spine is contained in a curved rectangular container inside the torso. A total of 15 steel tubes with a diameter of 10 mm connect the rubber torso to the spine (Figure 8). In order to reduce the bending resistance of the rubber torso, a water filled bladder (volume 2.05 litres) is enclosed in the abdominal region of the torso (Figure 8). The bottom of the rubber torso is attached to the pelvis.

In the BioRID, the original Hybrid III pelvis anterior-superior iliac spine height is decreased to conform with the modifications to the Advanced Anthropometric Test Dummy prototype (Schneider et al., 1992). The original pelvis front flesh is removed to allow the abdomen to bulge forward. The pelvis flesh is modified to reduce femur joint flexion/extension resistance. The BioRID was dressed in two layers of elastic nylon/Lycra shirt and pants to mimic the low friction observed between the human skin and normal clothing.

A new modified version, BioRID P3, of the original BioRID A (BioRID I) (Davidsson et al., 1998a) was tested. The BioRID P3 had a somewhat modified spine stiffness and rubber-torso stiffness compared to the BioRID A.

Dummy validation

The validation data used in this work was from 5 volunteer tests, a subset of a larger series of rear-end impact volunteer tests (Davidsson et al., 1998b). The angular displacements of the dummy head, T1 and head relative to T1 are compared to volunteer data (Figure 9). The T1 angular displacement and angular velocity for the BioRID were similar to those of the volunteers for the first 250 ms. The BioRID maximum T1 rearward angular displacement was well within the volunteer corridor while the maximum T1 rearward displacement was about 9° less for the Hybrid III. The head relative to T1 angle for the BioRID P3 stayed within the volunteer corridor, except for the less pronounced early head flexion between 70-120 ms (Figure 9), and had a significantly improved time history compared to the Hybrid III.

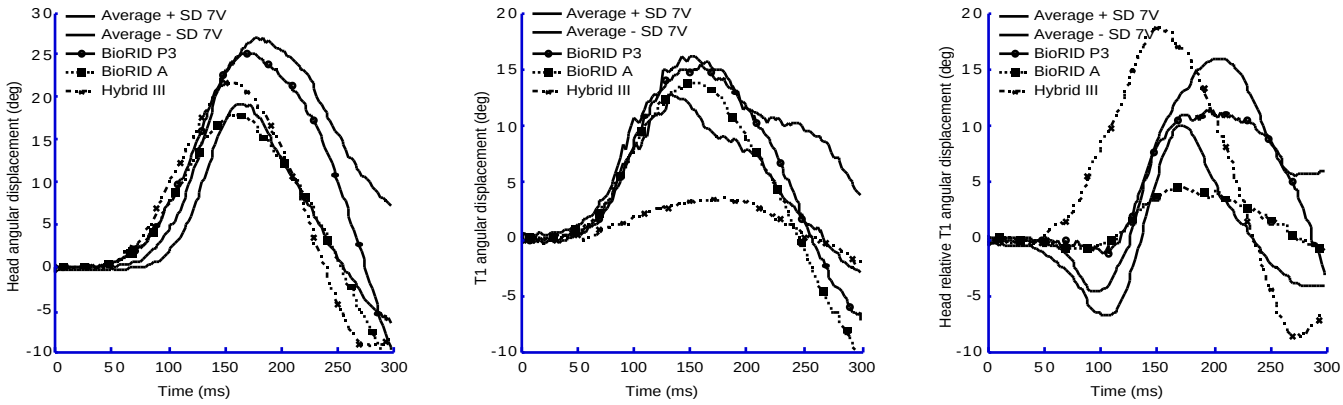

Figure 9: Rearward angular displacements of the head, T1 and head relative to T1 compared to the volunteer response corridors (average ± standard deviation) for the Hybrid III, BioRID A and the new BioRID P3 at ΔV=7 km/h.

Between 50 ms and head-restraint contact, at about 95 ms after impact, the Hybrid III head was accelerated forward more than was the average volunteer, while the BioRID head x-acceleration was almost negligible prior to head-restraint contact. The response of the BioRID P3 was generally closer to the volunteer results than was the response of BioRID A (Fig. 9). Further results will be published elsewhere.

Discussion of the rear impact dummy project
The head relative T1 angle had a good time history response and magnitude. The Hybrid III head relative to T1 angle was larger than those of the volunteers and the BioRID P3. The rearward rotation of the Hybrid III head relative to T1 started 40 ms before the volunteers and the BioRID. The data demonstrate that the Hybrid III thoracic spine was too stiff in sagittal bending and that the Hybrid III neck retraction resistance was too high.
Svensson and Lövsund (1992) reported translation motion without angular displacement for the head centre of gravity relative T1 in the validation of the RID-neck. It was concluded that a larger head lag was possible if the RID-neck design was supplemented with anterior and posterior muscle elements. A later validation study by Geigl et al. (1995) indicated that the head lag is too small with the RID-neck in rear-end impacts. Therefore, the BioRID neck was fitted with posterior and anterior muscle substitutes connecting the occipital interface and the T1. The design is similar to that of the next generation frontal impact dummy (Eppinger et al., 1994).

Conclusion of the rear impact dummy project
The design proved to be repeatable and reproducible (Davidsson et al., 1998a).The T1 and head angular displacements of the BioRID were close to those of the average volunteer while the Hybrid III T1 displacement was significantly different from that of the average volunteer.

CONCLUSION

This work has resulted in the finding of an injury that could explain many of the most common neck injury symptoms caused by car collisions. Indications of a new injury mechanism have been found that could explain how these injuries may be a result of transient pressure gradients in the spinal canal. The new neck injury criterion (NIC) predicts the risk of injury as a function of the head-neck motion, and the new biofidelic rear impact dummy BioRID offers a significantly more human like head-neck performance compared to earlier crash dummies in rear-end crash tests. Together these findings will contribute more effective development and testing of new car designs for improved neck protection, primarily in rear-end collisions, but possibly also in other impact directions.

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