Pressure Effects in the Spinal Canal during Whiplash Extension Motion : A Possible Cause of Injury to the Cervical Spinal Ganglia

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ABSTRACT

Whiplash extension trauma in rear impacts at low impact-velocities (<20 km/h) often cause pain in the neck region as well as a number of other neurological symptoms, most of which can be related to the nerve paths that pass through the cervical intervertebral foramina.

When the neck is flexed or extended in the sagittal plane the length of the cervical spinal canal alters but the cross-sectional area of the canal remains almost constant. During flexion-extension motion of the cervical spine, the size of the inner volume of the spinal canal will change. Since the tissues inside the canal can be considered incompressible, an alteration will take place of either the amount of cerebro spinal fluid or the amount of blood in the veinplexa of the epidural space, or both. This requires fluid transportation through the intervertebral foramina as well as along the spinal canal. During a whiplash extension motion, the flow velocity can be expected to rise far above physiologically normal levels and pressure gradients can thus be expected to occur. In turn, the soft tissues inside and around the cervical spine and particularly in the intervertebral foramina will sustain mechanical strain and stress.

Anaesthetised pigs were exposed to a swift extension-flexion motion of the neck while the pressure inside the spinal canal and the skull was measured. Pressure pulses of magnitudes up to 150 mmHg (20 kPa) were observed during the motion. The magnitude of pressure is for each moment dependent on the position of the neck, the velocity and the acceleration of the motion.

Plasma membrane dysfunction was indicated by the results from light microscopic analyses of the cervical and the three upper thoracic spinal ganglia revealing the staining of nerve cells and satellite cells by protein complexed to the Evans Blue dye.

INTRODUCTION

Neck injuries frequently occur in rear-end car accidents. Nygren (1984) found that 18% of all accidents in Sweden involving injured drivers were rear-end collisions and similar findings have been reported by others (States et al., 1972; James et al., 1991). Data published by Langwieder et al. (1981) and Kahane (1982) indicate that 80%-90% of those injured in rear-impacts sustained neck injuries. According to Foret-Bruno et al. (1991) and James et al. (1991) this type of injury is nearly always classified as ”minor injury” (AIS=1) in the abbreviated injury scale (AIS). In spite of this low AIS rating these neck injuries lead to permanent disability (disability degree ≥10%) in about 10% of the cases, which should be compared with 0.1 % of the cases for all other AIS 1 injuries (Nygren, 1984).

Neck injuries in rear impacts mostly occur at very low impact-velocities, typically less than 20 km/h (Kahane, 1982; Romilly et al., 1989; Olsson et al., 1990). The use of head-restraints decreases the risk of neck injury in a rear-end collision by about 20% on average (O'Neill et al., 1972; Huelke and O'Day, 1975; Nygren et al., 1985).

The motion of the head and neck that occurs during a rear-end car collision when the impacted vehicle accelerates and the torso is pushed forwards by the seat-back is in this paper defined as whiplash extension motion. In this situation the head lags behind due to its inertia and the result is a swift extension motion of the neck. The neck motion is be stopped by the anatomical structures of the neck (muscles, vertebra, discs and ligaments) and also, when applicable, by the head-restraint. In the next phase of the motion, the head and neck return to their upright position and might finally go into flexion. Results from simulated rear-end impacts using volunteers (Mertz and Patrick, 1967; Tarriere and Sapin, 1969; McConnell et al., 1993) show that this flexion motion is relatively slow compared to the initial extension motion.

The injury symptoms following whiplash extension trauma include pain, weakness or abnormal response 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 (Nygren et al., 1985; Watkinson et al., 1991). Vision disorders, dizziness, headaches, unconsciousness, and neurological symptoms in the upper extremities are other symptoms that have been reported (States et al., 1972; Nygren et al., 1985; Hildingsson, 1991; Watkinson et al., 1991).

The aim of the present study was to investigate a hypothesis presented by Aldman (1986) regarding hydro-dynamic pressure phenomena in the central nervous system (CNS) as a potential injury-causing factor in whiplash extension trauma. The present study was preceded by a pilot study (Svensson et al., 1989).

THEORETICAL BACKGROUND

The length of the cervical spinal canal alters when the neck is flexed or extended, it increases at flexion and decreases at extension (Breig, 1978) (Figure 1). In one case where the cervical spine of a human cadaver was moved from maximal flexion to maximal extension, the length of the spinal canal decreased by about 30 mm (Breig, 1978). The cross-sectional area of the cervical spinal canal decreases during neck extension since the ligamenta flava protrude into the canal (Breig, 1978) (Figure 1). This means that the inner volume of the spinal canal decreases during neck extension and increases during flexion of the neck. However, all the tissues and fluids inside the spinal canal are virtually incompressible (Estes and

McElhaney, 1971). This means that fluid transportation, to and from the cervical spinal canal, must take place during the flexion-extension motion of the cervical spine. The fluid moving to and from the cervical spinal canal during this motion could be either blood in the vein-plexa of the epidural space or cerebro-spinal fluid (CSF) (Figure 2).

According to Batson (1957) both the internal and the external vertebral venous plexa that communicate via vein bridges through the intervertebral foramina have a capacity by far exceeding that of the arteries supplying the tissues of the same region. Since these vein-plexa do not have any valves, the blood can easily move in any direction within the plexa, and also back and forth between the inner plexus and the outer plexus. This means that blood volumes can move along the inside of the spinal canal as well as between the inside and outside of the spinal canal and thus compensate for the change in inner volume of the spinal canal during the flexion-extension motion.

CSF can move up and down the spinal canal and the amounts of CSF in the nerve-root sleeves can also alter to compensate for the change in inner volume in the cervical spinal canal during the extension-flexion of the neck. Results by Löfgren et al. (1973) indicate that the flow resistance of CSF in the subdural space of the spinal canal is relatively high. Thus, the flow of CSF may in this context be of minor importance compared with the motion of the blood in the vein-plexa.

During the extension-flexion motion, particularly when the motion is rapid like for instance during a whiplash extension motion, pressure gradients along the spinal canal as well as across the intervertebral foramina can be expected. There are two separate superimposed effects that will cause this gradient. The first effect is the pressure gradient that occurs when a column of fluid accelerates and the pressure difference is proportional to the height of the fluid column as well as the magnitude of the acceleration. The second effect is the pressure gradient caused by the flow resistance in the vessels. Inside the spinal canal, both higher and lower pressures relative to the soft tissues surrounding the spine could be expected to occur during an extension-flexion motion of the cervical spine. The pressure gradients mentioned above can be expected to generate injurious stresses and strains to the exposed tissues.

MATERIALS AND METHODS

Fourteen male and female pigs of the Swedish landrace strain (body weight about 20-25 kg) were used in the experiments (Table 1). The animals were anaesthetised throughout the experiments. Ketamin™ was initially injected intramuscularly but thereafter administrated through intravenous infusion. The animals were intubated, and ventilated by means of a respirator. For the animal experiments, permission was granted by the ethical committee.

Treatments

Two of the animals (PW 01 and 02) were used for pressure measurements in the spinal canal and in the skull during simulated whiplash extension motion. Twelve animals were used for histopathological examination, eight of them (HW 01-08) being exposed to simulated whiplash extension motion and four (HC 01-04) served as sham-exposed controls.

Simulated whiplash extension motion

The animals that were exposed to whiplash extension motion were placed laterally on an operating-table with the right side of the body down. The table was equipped with a backrest to which the animals were strapped. In order to provide as good reproducibility of the test set-up as possible the backrest was made rigid. The upper (superior) end of the backrest was placed in level with the T1 spinous process.

The animals were exposed to whiplash extension-motion by means of a specially designed pull-rig (Figure 3).

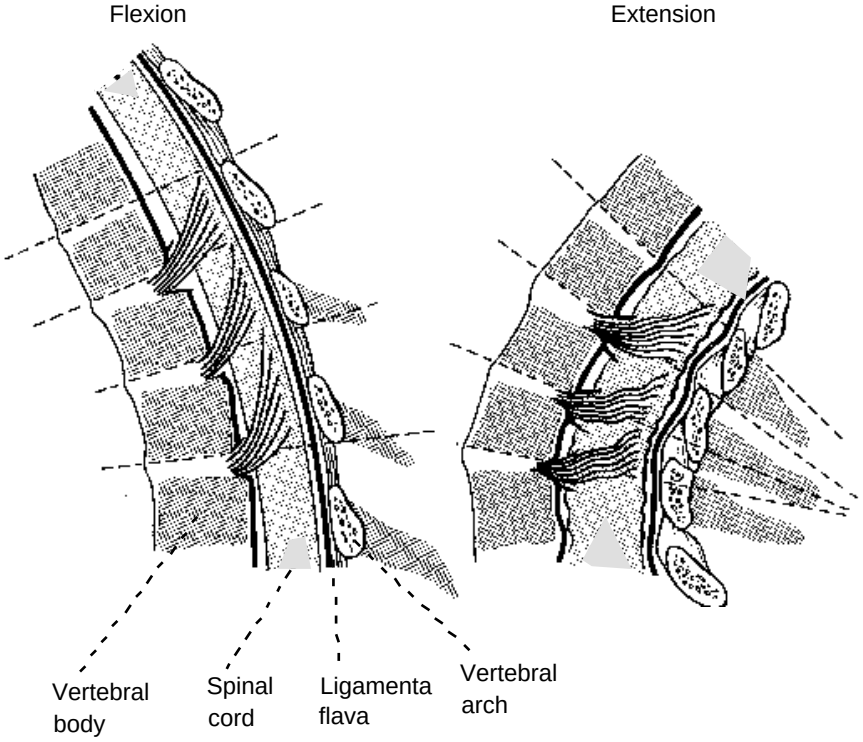

Figure 1: A sagittal cross-section of the lower cervical spine (C7-C3) in flexion and extension (adapted from Breig, 1978).

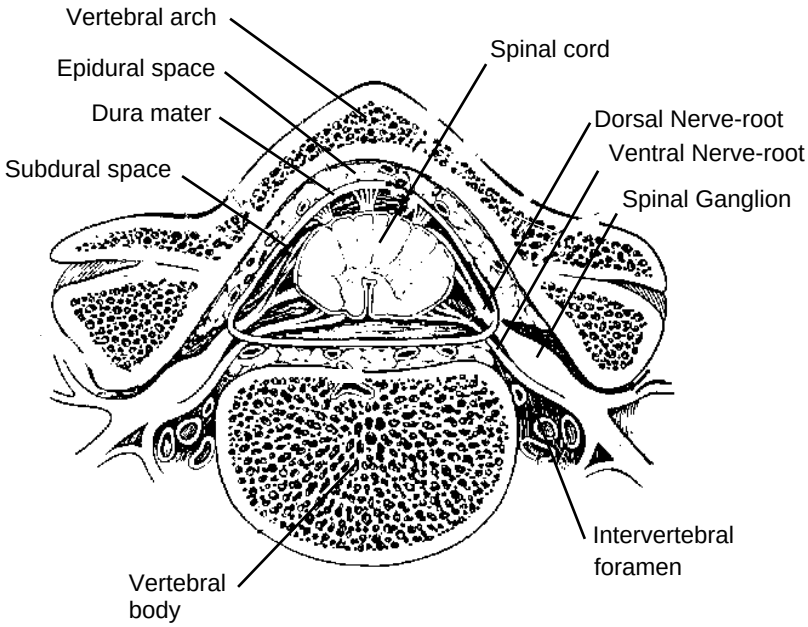

Figure 2: A horizontal cross-section of a cervical vertebra with the soft tissues of the spinal canal and intervertebral foramina (adapted from Sances et al., 1984).

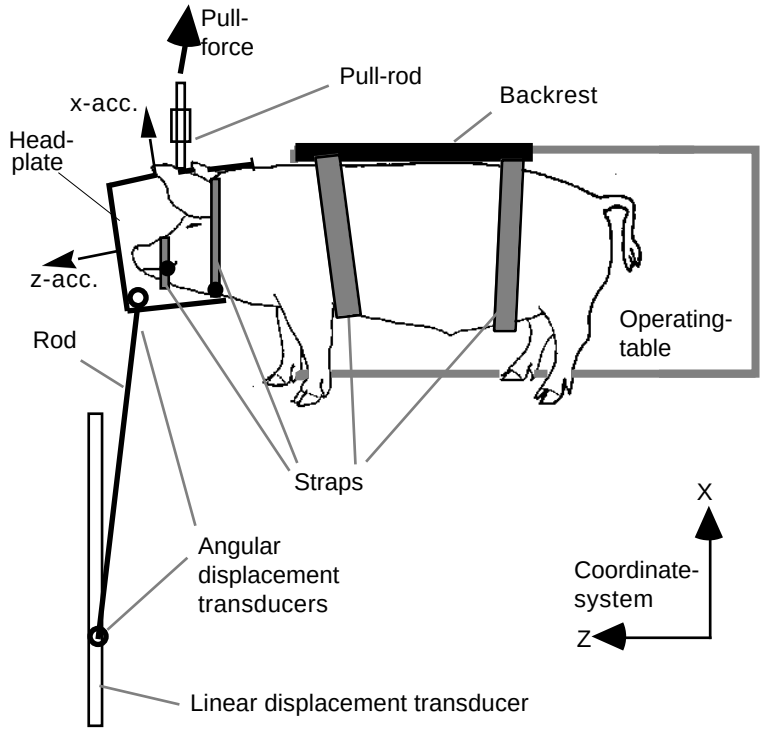

Figure 3: The test set-up seen from above. The anaesthetised animal is lying side down on the operating-table, strapped to the backrest. The head is strapped to the bolts in the horizontally movable head-plate. During the experiment a pre-tensed rubber-strap of a pull-rig pulls the head-plate by the pull-rod. The pull force is active until the pull-rod is disassembled and thereafter the head moves in the sagittal plane by its inertia.

The rig was equipped with a horizontal head-plate that was freely movable horizontally (both translational and angular motion). Two bolts were perpendicularly screwed on to the head-plate: One between the upper and lower jaw and one below the proximal part of the mandible. The head was firmly fixed by straps to these two bolts.

The pull force was generated by a pre-tensed long rubber-strap that provided an almost constant pulling-force during the whole pulling distance. The pull force could be set to up to 900 N with an accuracy of ±20 N (with exception of HW 01-04 where the accuracy was ±100 N) . The force was transmitted from the rubber-strap to the head-plate by means of a pull-rod. This rod was made of two aluminium tubes connected so that they disassembled after a chosen pull-distance, after which the head moved in the sagittal plane restricted only by the neck. The pull-distance was set to 135 mm in all the tests.

The head-plate accelerations were measured in booth the x-direction and the z-direction using two accelerometers (Entran, EGC-500DS-100SC). For the HW 01-04 the head-plate was equipped with light emitting diodes and the head motion was monitored by a video camera according to a method by Kullgren et al. (1992). For the PW 01-02 and HW 05-08 animals the head motion was monitored by means of one linear displacement transducer (Baureihe LWG 600) and two angular displacement transducers (Baureihe P 4500). One of the angular displacement transducers was fixed to the head-plate and was connected via an aluminium rod to the other angular displacement transducer, which in turn was mounted to the linear displacement transducer (Figure 3). All transducer signals were amplified

(Johne+Reilhofer 8 MV 1) and stored in a Macintosh II computer equipped with data acquisition hardware (NB-MIO-16, National Instruments). The signals were low-pass filtered (1 kHz). From the displacement transducer data the translational and angular displacements of the head in the sagittal plane could be calculated.

The sham-exposed control animals (HC 01-04) were subjected to a slow and gentle extension-flexion motion of the neck. First the neck was bent to full extension and thereafter to full flexion. This was done by hand.

Pressure measurements

The two animals used for the pressure measurement experiments (PW 01-02) were equipped with three pressure transducers, one transducer mounted in the frontal bone of the cranium measuring the CSF pressure inside the cranium and two transducers placed subdurally in the spinal canal.

A hole was drilled through the frontal bone and through the dura mater at eye level about 20 mm to the side of the sagittal line. Saline was poured into the hole to prevent air entry while the pressure transducer (Endevco 8510-100, pressure range: -0.1 to +0.7 MPa, frequency range: 0-10 kHz) was fastened into the hole.

Laminectomy was done on one of the lowest thoracic vertebra, a small hole was cut in the dura mater and the two catheter-tip pressure transducers (p^{P}G HD-33-002-9001, pressure range ±40 kPa, frequency range 0-20 kHz) were operated into the subdural space and pushed into place, one at cervical level and the other in level with the upper thoracic region.

Each of the two animals used for the pressure measurements (PW 01-02) were exposed to several whiplash extension motions with various degrees of pulling force and with various positions for the pressure transducers along the spinal canal (Table 1). The state of the animal was observed after each run. Animal PW 01 died after the fifth run.

Histopathological examination

The twelve animals (HW 1-8 and HC 1-4) were used to investigate whether whiplash extension motion could cause dysfunction of the membranes of neural spinal ganglia cells. These animals were given an injection of the Evans Blue dye, conjugated to albumin (EBA) (1 ml of a 4% solution injected intravenously per kg body weight; Suneson et al., 1987) 10 minutes prior to the simulated whiplash extension motion. The animals were anaestethized for an additional two hours prior to fixation by transcardial perfusion with buffered formalin solution after an initial rinsing with buffered saline. The brain and the spinal cord to about the T4 level were dissected and fixed in buffered formalin. The spinal ganglia and proximal parts of corresponding nerves were identified and isolated.

Table 1: Listing of the complete set of tests in the present study.

Animal	Test run no.	Pulling force (kN)	Comments	Pressure transducers
PW 01	01	0.15	-	Skull, C6, T2
	02	0.15	-	Skull, C6, T2
	03	0.30	-	Skull, C6, T2
	04	0.45	-	Skull, C6, T2
	05	0.60	The animal died	Skull, C6, T2
	06	0.30	Postmortem	Skull, C6, T2
PW 02	01	0.15	-	Skull, C1, T1
	02	0.15	-	Skull, C1, T1
	03	0.15	-	Skull, C1, T1
	04	0.15	-	Skull, C4, T1
	05	0.15	-	Skull, C4, T1
	06	0.15	-	Skull, C4, T1
	07	0.60	-	Skull, C4, T1
	08	0.45	-	Skull, C4, T1
	09	0.30	-	Skull, C4, T1
	10	0.15	-	Skull, C4, T1
	11	0.75	-	Skull, C4, T1
	12	0.90	-	Skull, C4, T1
	13	0.15	-	Skull, C4, T1
	14	0.15	Data lost	Skull, C6, T1
HW 01	01	0.60	-	-
HW 02	01	0.60	-	-
HW 03	01	0.60	-	-
HW 04	01	0.60	-	-
HW 05	01	0.60	-	-
HW 06	01	0.60	-	-
HW 07	01	0.60	-	-
HW 08	01	0.60	-	-
HC 01	-	-	-	-
HC 02	-	-	-	-
HC 03	-	-	-	-
HC 04	-	-	-	-

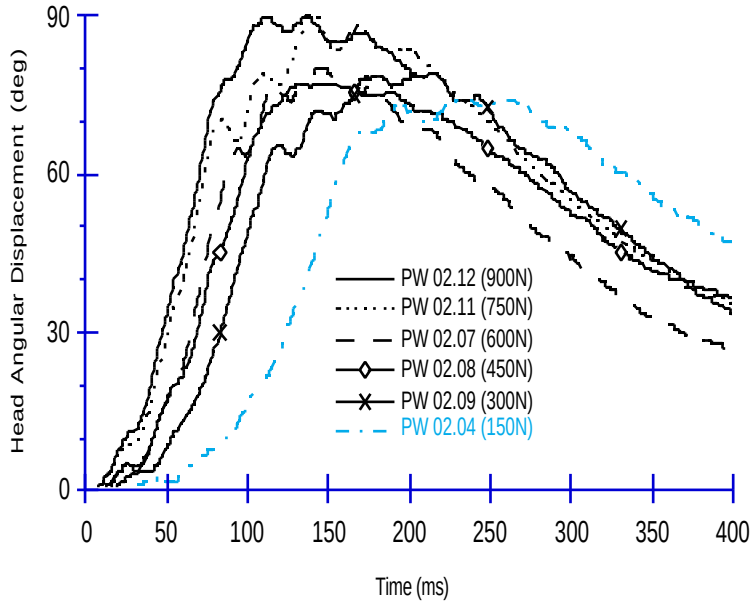

Figure 4: The angular displacements versus time of the head at simulated whiplash extension motion at different magnitudes of pulling-force using the same animal (PW 02).

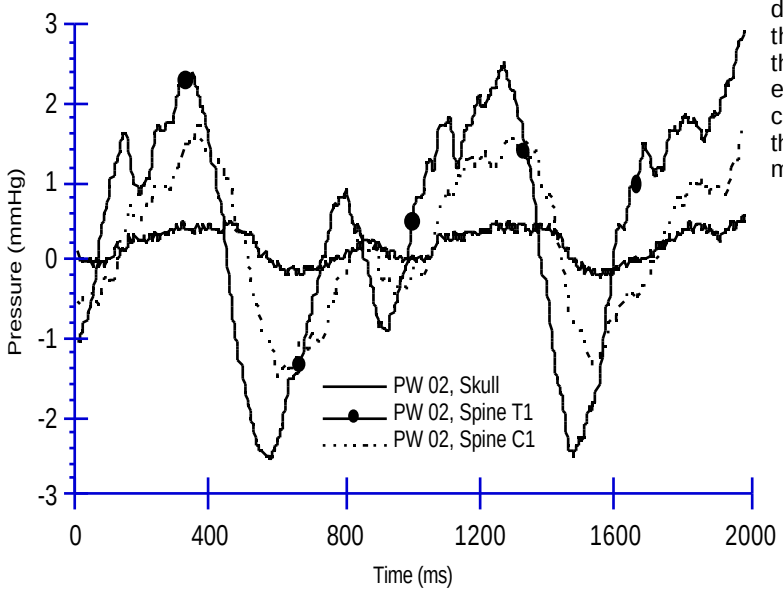

Figure 5: The pressure versus time during two seconds at three levels of the CNS in the PW 02 animal before the first simulated whiplash extension exposure. The zero-level corresponds to the mean-pressure of the CSF at the three different measurement sites.

Cryostate microtome sections with a normal thickness of 5-8 µm were prepared and examined in a fluorescence microscope equipped for epiillumination and with appropriate filters for evaluation of red fluorescence (Suneson et al., 1987).

RESULTS

The angular displacements for six whiplash extension experiments with the same animal (PW 02) at six different degrees of pulling force are shown in Figure 4. The maximum angular displacement occurs earlier and increases in magnitude with increased pulling-force (Fig. 4).

Pressure measurements

Pressure fluctuations in pace with the heart rate in the CNS at the three levels, skull, C1, and T1 were registered when the animal (PW 02) was lying on the right side prior to the first whiplash extension test (Fig. 5). The zero level in this diagram corresponds to the mean pressure level of the CSF at the sites of the transducers.

One whiplash extension run with animal PW 02 is shown in Figure 6. The pulling force is 600 N which is the same force as in the simulated whiplash extension exposures for the animals in the histological examination. 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 in the figure. The start of the angular motion of the head is delayed about 30 ms compared to the linear X-displacement, which indicates that the head is moving mainly translationally during the first 30 ms. The peak values for the pressure in the spinal canal at C4 level are about -70 mmHg and +85 mmHg (-9.3 kPa and +11.3 kPa) in this test.

The general pattern of the pressure pulse at C4 level in the spinal canal is the same for all degrees of pulling force but the pulse becomes shorter in duration and

higher in magnitude with increased pulling force (Figure 7). Pressure peak values of down to -110 mmHg (-14.6 kPa) and up to +145 mmHg (+19.3 kPa) relative to the mean pressure at rest were measured.

The pressures at three different levels of the spinal canal are shown in Figure 8 for six consecutive whiplash extension experiments at 150 N pulling force using the same animal (PW 02). In the first three experiments measurements were done at the C1 and the T1 level, and in the last three experiments at C4 and T1. The measurements at the T1 level functioned as a reference and the pressure at this level had a very similar profile throughout all six tests. The three C1 level pressure profiles were very alike and so were those of the C4 level. However, the pressure profiles were significantly different between the C1 and the C4 levels, particularly in the time interval 50 - 150 ms were the profiles are almost reversed. In the interval 50-100 ms the pressure increases some 10 mmHg at C4 level and decreases some 15 mmHg at T1 level, and in the interval 100-150 the C4 pressure decreases about 40 mmHg while the T1 pressure fluctuates around 10 mmHg.

A comparison was made of the pressure profile in the CNS between two different animals (PW 01 and PW 02) during simulated whiplash extension motion using 300 N pulling force (Fig. 10). This comparison gives an indication of the reproducibility of the pressure pulses between two different animals subjected to the

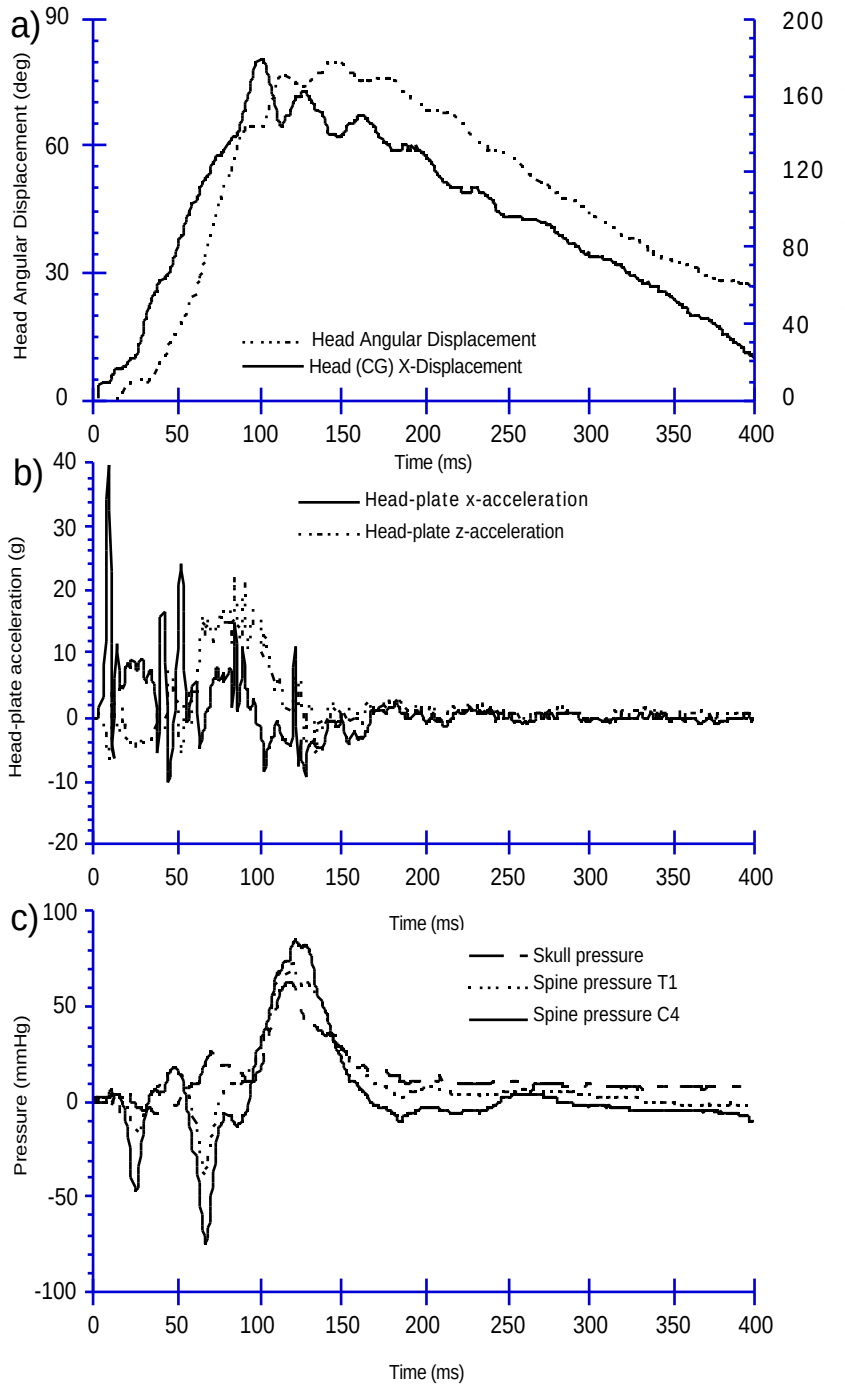

Figure 6: The results from one whiplash extension run (PW 02.07) with pressure measurements. The applied pulling force on the head-plate was 600 N.
a) Angular displacement and the linear X-displacement of the head versus time.
b) Accelerations versus time of the head-plate versus time.
c) The pressure versus time in the CNS at three levels, skull, C4, and T1.

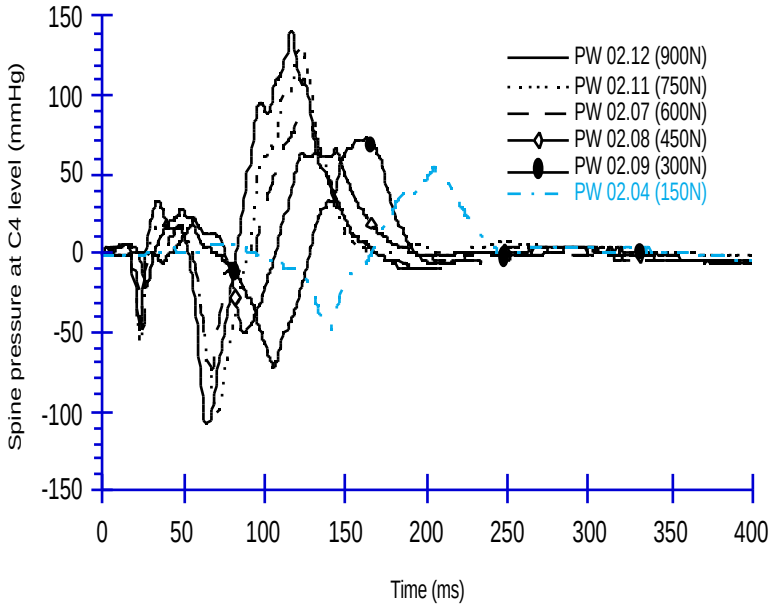

Figure 7: The pressure versus time in the spinal canal at C4 level in animal PW 02 for various pulling forces.

same pull-force. The kinetic energy of the motion is virtually identical in the two tests but there is some difference in the distribution between angular and linear motion (Fig. 10a). Nevertheless the pressure profiles show very similar patterns (Fig. 10b). The timing of the different pressure events is almost identical and the magnitudes of the pressure peaks are similar. Comparison between the two animals is restricted by the fact that the pressure transducers in the spinal canal were not placed at exactly the same spinal levels and

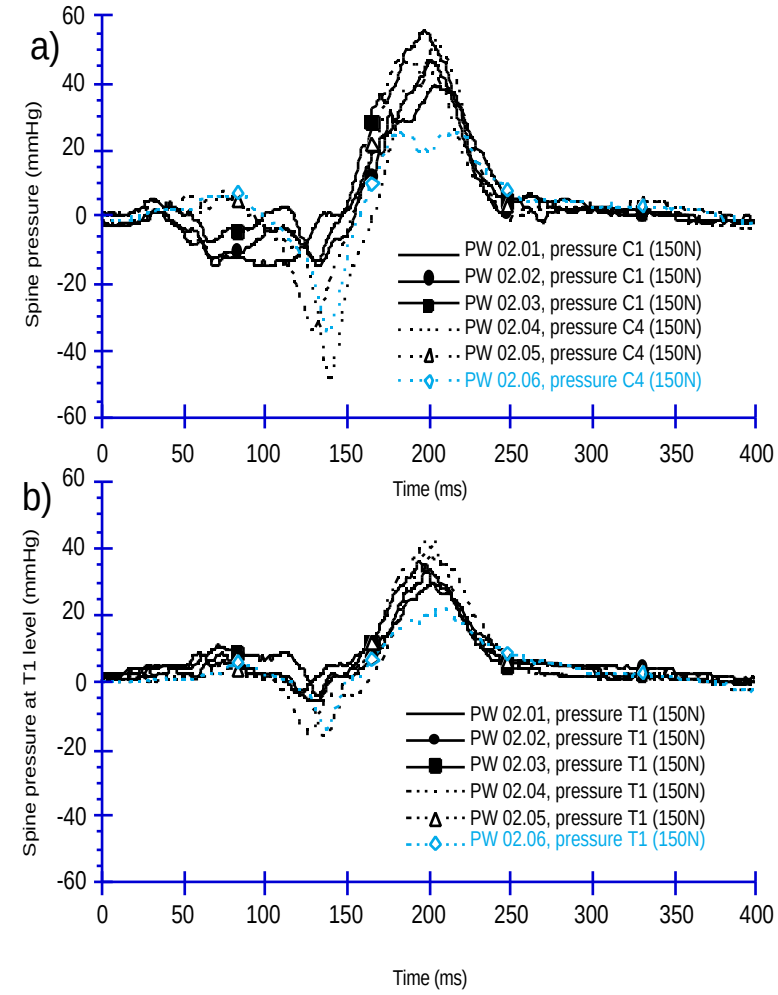

Figure 8: Six consecutive whiplash extension runs were done with one animal (PW 02) using 150 N pulling force.
a) Pressure versus time at C1 level for the first three runs and at C4 level for the three last runs
b) Pressure versus time at T1 level for all six runs.

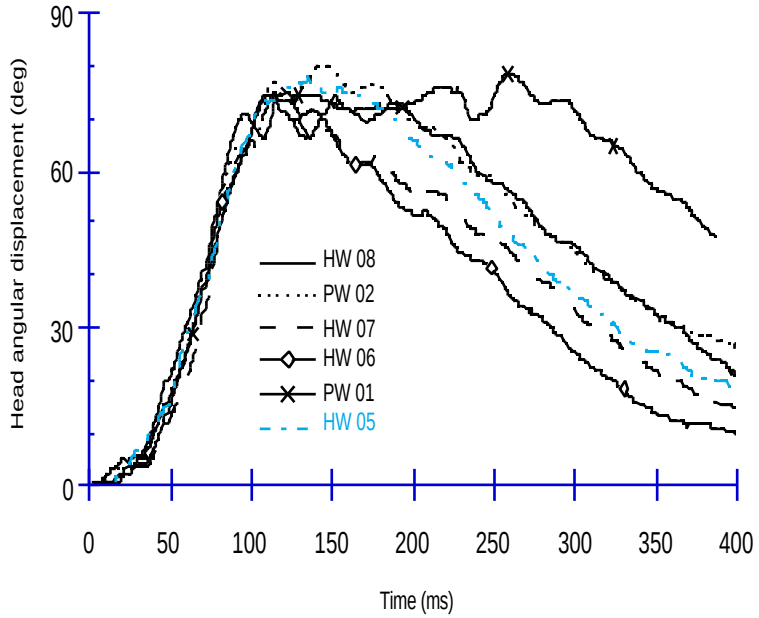

Figure 9: The angular displacement versus time of the head in six simulated whiplash extension runs with six different animals (600 N pulling-force).

that the pressure measurement in the skull failed in animal PW 01.

The angular motion of the head in six experiments involving six different animals at the 600 N pull-force level illustrates the reproducibility of the experiments (Figure 9). The loads to the neck can be expected to be the greatest during the first 150 ms where the angular motions are almost identical for the six tests. A resonance vibration of about 40 Hz occurred in the rod connecting the two angular displacement transducers (Fig. 3) and this disturbance was superimposed on all the displacement results in this study.

Histopathological examination

The macroscopical inspection of the nervous tissue from the 8 and 4 pigs in groups HW and HC respectively revealed no abnormalities. There were no bleedings, fractures of vertebral structures or ruptures of ligaments.

Fluorescence microscopic examination disclosed that satellite cells and nerve cells in the spinal ganglia contained red fluorescent material, indicating leakage of the protein-dye complex and thus membrane dysfunction. These findings were most obvious at the C6 - C8 levels and diminished in both cranial and caudal direction. There were no signs of EBA leakage into the satellite cells or the nerve cells in the spinal ganglia from the sham-exposed animals. Detailed morphological and biochemical data will be published separately (Örtegren et al., 1993).

DISCUSSION

The results in Figure 9 show that the test set-up in this study offered a good reproducibility of the head-neck motion between the different pigs in the critical interval from 0 ms until 150 ms. The results in Figure 10 indicate that the reproducibility of the pressure pulses in the CNS between the different animals in the

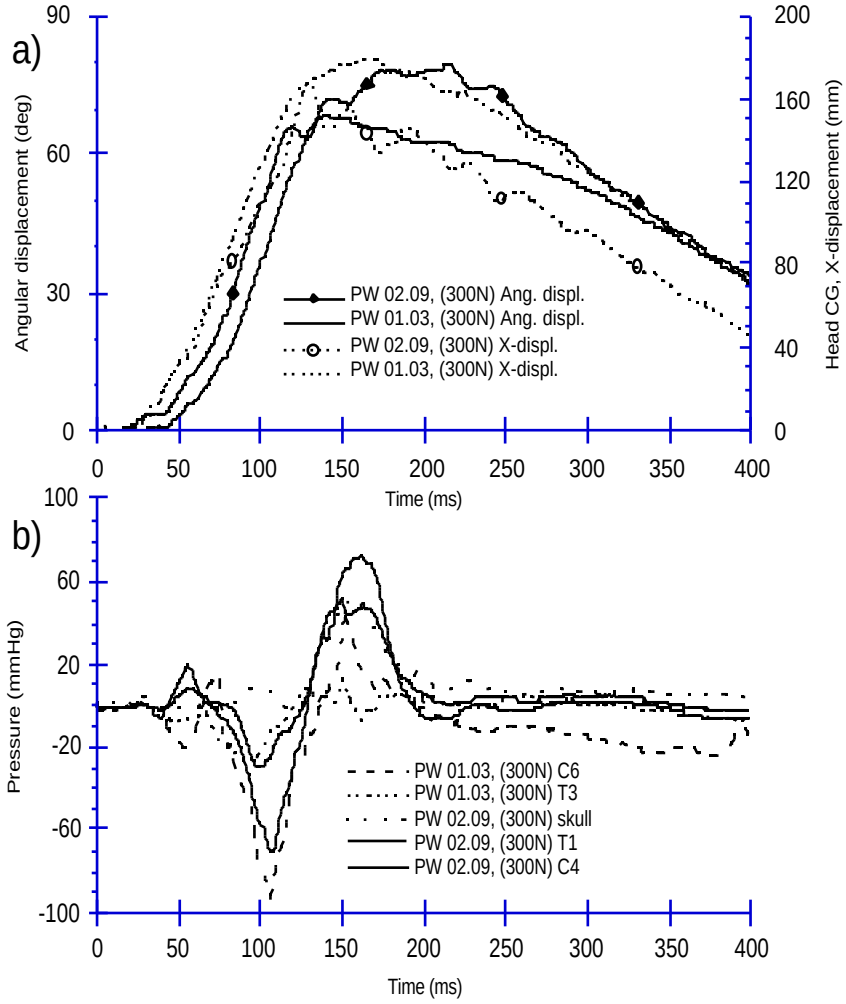

Figure 10: The head motion (a) and pressure in the CNS (b) versus time is compared in two animals (PW 01 and PW 02) at 300N pulling force. (The skull-pressure measurement failed in PW 01.)

study is acceptable. The repeatability when the same animal is used under identical conditions in repeated test runs is also acceptable (Fig. 8). It was difficult to control the exact initial angular position of the animals head in the test set-up of this study. This may explain the spread in the pressure measurement results between identical repeated test runs. The fact that the animals' neck structures may have been affected by each whiplash extension exposure and thus gradually changed their mechanical properties is another possible cause of spread in the results.

The loading conditions on the head and spine in the experiments in this study and in the real world whiplash extension motion differed to some extent. In this study the head was pulled rearwards in one point close to the head centre of gravity while the torso remained fixed. In a real accident, the torso is pushed forwards and the head and neck lags behind resulting in a swift extension motion of the neck. The inertial loading of the head and the neck segments go in opposite directions in the two cases. This probably means that the "s-shape" of the spine (Figure 11b) in the initial stage of the motion develops differently and that the pressure pattern during this phase may differ slightly. McConnell et al. (1993) undertook staged rear-impacts using volunteers and reported z-acceleration of the head due to the straightening of the chest-kyphosis during the acceleration of the torso. Since in the present study the torso is not accelerated forward during the experiments this type of head acceleration does not occur. However, the loading conditions and the head-neck motion in the experiments were considered to be close enough to a real accident situation to serve as a relevant model.

The method of measuring the head displacements by means of a video camera (Kullgren et al., 1992) proved to function well. The displacement transducer method was introduced since the motion was restricted to one plane and to a very limited motion range. With the latter method the analysis became less time-consuming and the time resolution of the measurements was also improved. Unfortunately a vibration in the rod connecting the two angular displacement transducers added an artefact to the measurement signal.

The pressure readings have very similar contours at the three CNS levels (Figs. 6c, 8, and 10b), which indicates that the readings are not caused by artefacts like for instance direct mechanical loads (clamping) to the pressure transducers. The pressure transducers are insensitive to acceleration and since no correlation between the pressure readings and the acceleration readings can be found this artefact can be excluded. The catheter-tip pressure transducers may be displaced somewhat along the spinal canal during the whiplash extension motion due to the alteration of the canal length. At the upper cervical spine the transducer movement could possibly reach up to 15 mm during the extension motion. Thus the transducer levels listed in Table 1 are approximate.

The overall anatomy of the cervical spine of the pig is similar to that of the human though the dimensions and the detailed shape of different tissue differs somewhat between the two species. The spine and head of the pig serve well as a qualitative model of the corresponding parts of the human body. It is more difficult to make quantitative comparisons as for instance try and predict the pressure magnitudes in the human spinal canal during an extension-flexion motion of a certain velocity or trying to predict the threshold magnitude of pressure for injury to the spinal ganglia from the corresponding level for the pig. Nevertheless this qualitative model will hopefully serve as guidance in terms of what kinematic and kinetic parameters are related to the risk of injury.

The swift extension motions that the animals are exposed to in the present study have a realistic time history when compared to other whiplash extension experiments. The time from start to maximum extension angle is in the range 100 ms to 250 ms. McConnell et al. (1993), in a staged rear-end collision with a volunteer in the driver's seat of the impacted vehicle at a ΔV of 7.8 km/h, measured a time of 140 ms from the initial stage of the head-neck motion to the maximum extension angle. This ΔV was considered to be at the limit where neck injury starts occurring. The seat in that experiment was equipped with a head restraint that stopped the angular displacement at about 45 degrees. During rear-impact sled tests, Svensson et al. (1993) attained maximum extension angles of about 40° and 70° after between 105 ms and 150 ms at 12.5 km/h ΔV and 5 km/h ΔV respectively using a Hybrid III-dummy equipped with a RID-neck (Svensson and Lövsund, 1992) seated in a rear-seat without a head-restraint.

In the present experiments the shape of the pig spine altered during the simulated whiplash extension motion in a way similar to that seen in experiments with the RID-neck (Svensson and Lövsund, 1992). At the start time the neck is straight (Fig. 11a) and during the first phase (Phase 1) of the motion the head is moving rearwards relative to the torso without any angular motion which means that the spine is formed into an "s-shape" (Fig. 11b). Thus the upper cervical spine is undergoing a flexion motion and the lower cervical spine is undergoing an extension motion. This phase resembles the time period 0 ms to about 100 ms in Figure 12 where the pressure curves show a small pressure rise at the C4 level below which the spinal canal becomes shorter and a pressure decrease at C1 where the spinal canal becomes longer.

At the end of Phase 1 the linear rearward motion of the head is abruptly decelerated at the same time as the head starts rotating backwards. This is explained by the fact that the upper cervical spine reaches its limit for maximum flexion while the lower cervical spine reaches the limit for full extension. In the next phase (Phase 2) the angular head motion is accelerated while the upper cervical spine goes from full flexion into extension and at the same time the lower cervical spine goes from full extension to a less extended posture. This phase resembles the time period 100-150 ms in Figure 12. During this period there is a clear dip in the C4 pressure since the lower cervical spinal canal is lengthening from its initially fully compressed position. The C1 curve also displays a small pressure dip although the spinal canal is shortening at this level. This might be explained by the fact that the effect of the volume expansion in the lower cervical spine dominates the compression of the upper cervical spine and superimposes on the pressure at C1 level.

In the last sequence (Phase 3 in Figure 11) the whole cervical spine goes into full extension and the motion stops and turns. During this phase the spinal canal shortens and a clear pressure peak is seen in Figure 12.

Following this extension motion the whole cervical spine returns towards its initial posture by means of the elastic energy that is stored in the neck structures at the fully extended posture. It should be

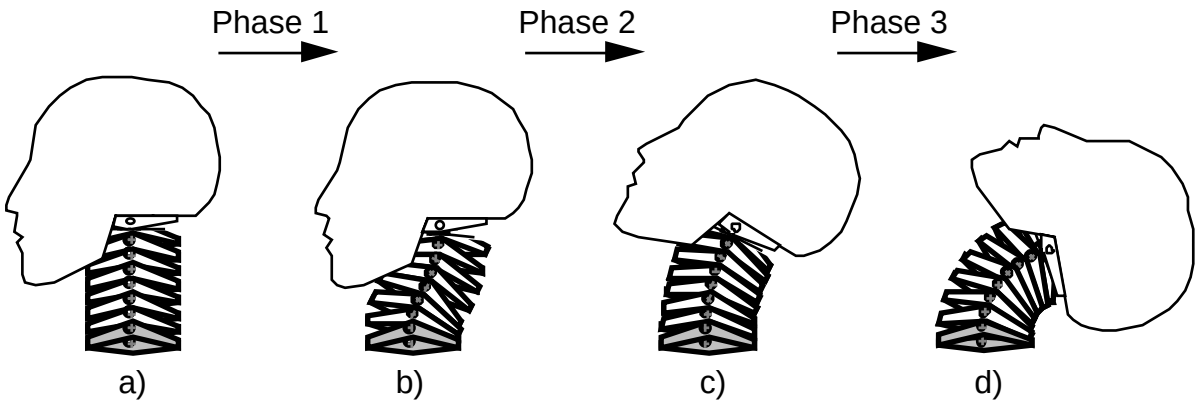

Figure 11: Schematic view of four parts of the whiplash extension motion; a) initial posture; b) maximum rearward translational displacement of head; c) Maximum rearward angular velocity of the head is reached, d) Maximum extension angle of the neck is reached.

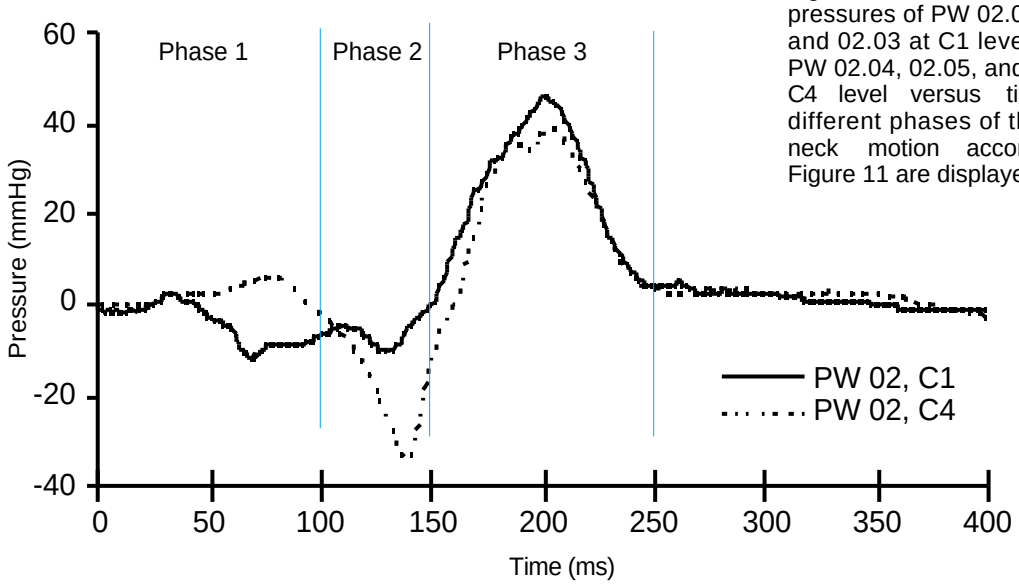

Figure 12: The average pressures of PW 02.01, 02.02, and 02.03 at C1 level and of PW 02.04, 02.05, and 02.06 at C4 level versus time. The different phases of the head-neck motion according to Figure 11 are displayed.

emphasised that there are no clear and exact borders between the different phases described above. The upper thoracic vertebrae probably follow the motion of the lower cervical vertebrae during all the five phases though they have a more restricted range of motion than the cervical vertebrae have.

There is a time-history correlation between the head-neck motion and the pressure profiles at the three different pressure transducers. This clearly indicates that the pressure pulses measured in this study during extension and flexion motions of the neck are caused by the hydro-dynamic effects that result from the change in inner volume in the spinal canal according to Aldman's (1986) hypothesis.

Pressure gradients could also arise in the CNS due to the accelerations of the head and the individual segments of the neck during the whiplash extension motion. This pressure effect though, would give rise to pressures that are proportional to the acceleration of the individual body segment. Since this type of correlation was not found in the pressure measurements of this study the acceleration of the different body segments probably only contributed mildly to the pressures measured.

The soft tissue of the neck is virtually incompressible and has viscous properties. As a result of the swift deformation of the the tissue during a whiplash extension motion, pressure changes inside the tissue can thus be expected to occur. Since the spinal canal is situated close to the centre of the neck where these soft tissue deformations are small, this soft tissue deformation is probably not an important factor in this context.

The pressure in the CNS when the animal is at rest (Fig. 5) is independent of the posture in the sagittal plane of the head and neck. This was shown in a pilot study (Svensson et al., 1989) where the pressure inside the skull and in the spinal canal at the upper thoracic spine was monitored during extension-flexion motions of the neck done by hand. When the neck was in movement the pressure was influenced by the motion but whenever the motion was stopped at an arbitrary neck angle, from full extension to full flexion, the pressure immediately reverted to the same rest-level, similar to the situation in Figure 5.

A typical mean level of the CSF pressure in a dog would be in the range 5-15 mmHg (0.7-2.0 kPa) (Löfgren et al., 1973) and it was assumed to be of the same order of magnitude in the pig. The magnitudes of the pressures measured in the CNS in the present study reached about 150 mmHg (20 kPa) which was about 10 times higher than the normal pressure.

The results from the pressure measurements indicate that the pressure gradients generated during the simulated whiplash extension motions are of greater magnitude across the inter-vertebral foramina than along the spinal canal. The pressures had the largest values in the lower half of the cervical spine. This correlates with the findings of the histopathological examination, where tissue damage was found in the spinal ganglia that are situated in the inter-vertebral foramina, and the injuries to the ganglia were most severe at the lower half of the cervical spine. The injuries to the cervical spinal ganglia are also corroborated by many of the known whiplash extension symptoms. Pain and sensory disturbances to the parts of the body that are associated with the cervical dorsal nerve-roots are common and vision disorders could be caused by injury to the upper cervical spinal ganglia (Hildingsson et al., 1991). Headaches could also be caused by injuries to the nerve root region (Hildingsson et al., 1991). The injuries to the spinal ganglia found in this study are likely to be caused by mechanical stresses and strains to the ganglia and surrounding tissues. These mechanical loads to the ganglia could be caused by deformation of the intervertebral foramina in turn caused by motions between adjacent vertebrae exceeding the normal range of motion. The deformations of the intervertebral foramina could be of compressive, tensile or shearing type. The loads to the ganglia could also be caused by the pressure gradients between the inside and the outside of the spinal canal according to the hypothesis by Aldman (1986). The fact that no signs of injury to vertebrae, discs or ligaments could be detected during the excision of the CNS specimens indicates that displacements between adjacent vertebrae only marginally exceeded the normal range of motion.

When the tests with animal PW 02 were completed after 14 runs the animal was still in good condition and thus an additional test consisting of two runs (PW 02.15 at 300 N and PW 02.16 at 450 N) was made. In

this test the animal was turned around so that its left side was facing down and it was then placed in the same test set-up as for the extension experiments (Fig. 3). The head was now pulled forwards causing a swift flexion motion of the head and neck.

The torso was not as firmly fixed in this experiment as in the whiplash extension experiments which may partly explain the relatively large maximum linear X-displacement in experiment PW 02.15 (Fig 13a). The accelerations of the head-plate were very similar to the corresponding accelerations of the whiplash extension experiments at the same pulling force (300 N) (Fig. 13b). The inner volume of the spinal canal changes in a very different way during the forward flexion motion compared to the whiplash extension motion. This explains why the pressure readings of the neck flexion runs (Fig. 13c) show a very different pattern compared to those of all the whiplash extension experiments. The maximum negative pressure was -85 mmHg at C6 level which is similar to the -70 mmHg measured at C4 level in the whiplash extension experiment PW 02.09 at the same pull-force (300 N). The data from PW 02.16 were lost due to an error in the computer software.

If we assume that the pressure phenomena observed in the CNS in the present study are the indirect cause of the injuries to the cervical spinal ganglia and that these injuries are identical to the neck injuries that are typical for car occupants that have been involved in rear impacts, this will have implications for the methods devised for preventing these injuries. It will not be sufficient with a head-restraint that prevents hyperextension of the neck. The rearward motion of the head and the neck during the rear impact must either be stopped very early on, probably before Phase 1 (Fig. 11) is completed or else the velocity of the motion must be limited to keep the pressure magnitudes in the spinal canal below the injury threshold.

SUMMARY AND CONCLUSIONS

In order to simulate the trauma to the neck of car occupants involved in rear impacts, anaesthetised pigs were exposed to a swift extension-flexion motions of the cervical spine. Pressure was monitored in the CNS and the animals were histopathologically examined for signs of injury to the nervous system in the nerve-root

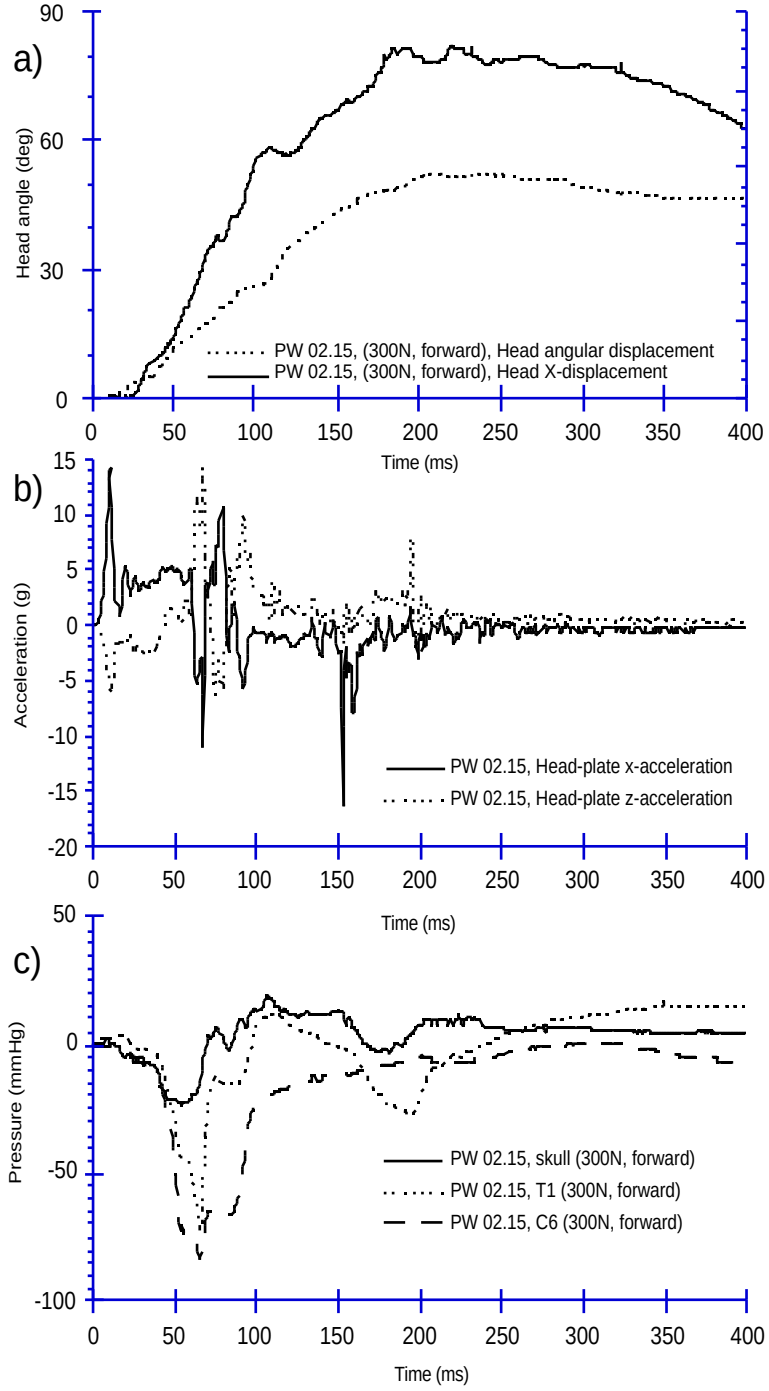

Figure 13: The head and neck of the animal (PW 02) were subjected to a swift flexion motion.
a) The angular and translational head motion versus time.
b) The accelerations versus time of the head-plate.
c) The pressure versus time in the CNS at three levels.

region of the cervical and upper thoracic spine.

Pressure pulses in the CNS with magnitudes of up to about 150 mmHg (2.0 kPa) were registered during the neck motion. The magnitude and the sign of the local pressure seems to correlate to the velocity and direction of the angular motion between adjacent vertebrae near the location of the transducer. Extension motion causes a pressure rise and flexion motion causes a corresponding pressure drop.

Injuries to the spinal ganglia particularly in the lower cervical region were found. These injuries could explain most of the symptoms that are typical for patients with an AIS=1 neck injury sustained in a rear-end car collision.

The results verify a hypothesis by Aldman (1986) predicting that the volume changes inside the spinal canal during an extension-flexion motion of the cervical spine would result in transient pressure changes in the CNS during a swift head-neck motion in the sagittal plane. Aldman (1986) also hypothesised that these pressure effects could induce injurious mechanical loads to the tissues inside the intervertebral foramina and this agrees well with the histopathological findings of the present study. The absence of obvious abnormalities to vertebrae, ligaments and discs after the induced whiplash extension trauma indicates that adjacent vertebrae have not been displaced relative to each other much further than the physiological range of motion. It is thus plausible that the injuries to the spinal ganglia were

caused by the transient pressure effects rather than the deformation of the intervertebral canals.

The methods used in the present study could be further developed and used to increase understanding of the causes of the injuries to the spinal ganglia and to get better isight into the working of these injuries and into the healing process. Better knowledge of how the nervous function is affected could also be aqiured. This in turn may provide knowledge that can be used to improve the medical treatment of whiplash patients and to improve the injury preventive measures in future vehicles. The animal model will however not provide any quantitative information about injury threshold levels for kinematic or kinetic parameters of the head-neck motion of the human being.

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