A NEW NECK INJURY CRITERION CANDIDATE-
BASED ON INJURY FINDINGS IN THE CERVICAL SPINAL GANGLIA
AFTER EXPERIMENTAL NECK EXTENSION TRAUMA

O. Boström[1], M.Y. Svensson[2], B. Aldman[2], H.A. Hansson[3,5], Y. Håland[1],
P. Lövsund[2], T. Seeman[4], A. Suneson[5], A. Säljö[5], T. Örtengren[4]

[1]Autoliv AB, Research, S-447 83 Vårgårda
[2]Dept. of Injury Prevention, Chalmers University of Technology, S-412 96 Göteborg
Depts. of [3]Anatomy & Cell Biology and [4]Surgery, University of Gothenburg, S-413 90 Göteborg
[5]Dept. of Human Studies, National Defence Research Establishment, S-172 90 Sundbyberg

ABSTRACT

Neck injuries caused by swift extension-flexion motions of the cervical spine during car collisions result in human suffering and high societal costs. The symptoms of these patients are well documented but the actual site of the injury as well as the relation between this injury and the head-neck motion have not been established.

In earlier work by our group, experimental extension-flexion trauma to the neck of pigs has revealed signs of injury to the spinal ganglia that could explain many of the typical symptoms. As a part of that work, transient pressure changes were measured in the cervical spinal canal. An hypothesis, that such pressure changes would be the cause of the ganglion injuries was presented.

The present paper focuses on when, during the extension-flexion motion, this injury may occur and what neck motion parameters may be of relevance.
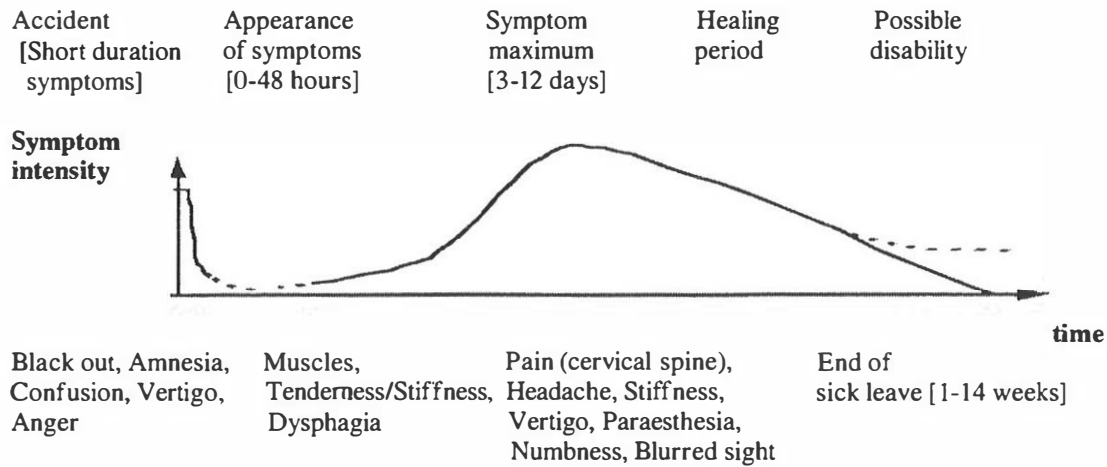
A mathematical model, based on Navier Stokes equations, was developed and validated against experimental data. The model predicts the pressure changes in the spinal canal as a function of the volume change inside the canal during neck bending in the x-z (sagittal) plane.

Further experiments on pigs were conducted. Preliminary results indicate that ganglion injuries, as well as pressure transients inside the spinal canal, seem to correlate to the phase shift when the neck passes an s-shape (or maximal retraction) during the rearward motion of the head. That is, when the upper neck quickly changes from a flexion to an extension shape. Static loading of the neck resulted in no signs of injuries to the ganglia.

With these findings established we present a possible candidate for a neck injury criterion. The criterion is based on the relative acceleration between the top and the bottom of the cervical spine. We also discuss a tolerance level based on the pig tests.

NECK INJURIES IN REAR-END COLLISIONS occur mostly at very low impact-velocities, typically less than 20 km/h (Kahane, 1982; Olsson et al., 1990) and are mostly classified as minor injury (AIS 1) on the abbreviated injury scale (AIS) (Foret-Bruno et

Fig.1 - The stages of the neck injury symptoms sustained in a rear-end collision
(adapted from Spangfort, 1985).

Accident [Short duration symptoms]	Appearance of symptoms [0-48 hours]	Symptom maximum [3-12 days]	Healing period	Possible disability

Symptom intensity

| Black out, Amnesia, Confusion, Vertigo, Anger | Muscles, Tenderness/Stiffness, Dysphagia | Pain (cervical spine), Headache, Stiffness, Vertigo, Paraesthesia, Numbness, Blurred sight | End of sick leave [1-14 weeks] | |

al., 1991; James et al., 1991; Ono and Kanno, 1993). In spite of this low AIS rating, these injuries lead to permanent disability (disability-degree>10%) in some 10% of the cases (Nygren, 1984). This can be compared with other AIS 1 injuries where the risk of permanent disability is 0.1% (Nygren et al., 1985).

According to Ono and Kanno (1993), 50% of all car-to-car accidents in Japan lead to neck injuries and the number of neck injuries are on the increase. In the Netherlands, the annual number of neck injuries increased by 54% during the period 1983 to 1991 (Kampen, 1993).

Women were found to be up to twice as vulnerable as men in rear-end accidents (Kihlberg, 1969; States et al., 1972; Kahane, 1982; Otremski et al., 1989; Foret-Bruno et al., 1991; vKoch et al., 1995; Spitzer et al., 1995).

Nygren et al. (1985) found that the use of head-restraints decreased the risk of neck injury in a rear-end collision on average by about 20%. Fixed head-restraints gave a 24% reduction and adjustable ones gave a 14% reduction. Similar findings have been presented by O'Neill et al. (1972) and by Huelke and O'Day (1975). However, Nygren et al. (1985) also found that the risk of neck injury was not reduced in newer cars. In fact the study disclosed great differences in the protective performance between different designs of seats and headrests, which is a clear indication of the need for further research in this area.

The risk of neck injury to rear seat occupants was only about 50% of the risk of neck injury for front seat occupants in rear-end collisions (Kihlberg, 1969; States et al., 1972; Carlsson et al., 1985; Lövsund et al., 1988; Otremski et al., 1989).

The injury symptoms following neck trauma in rear-end collisions include pain, weakness or abnormal response in the neck, shoulders and upper back (parts that are connected to the central nervous system via the cervical nerve-roots) together with vision disorders, dizziness, headaches, unconsciousness, and neurological symptoms in the upper extremities (States et al., 1972; Nygren et al., 1985; Hildingsson, 1991; Watkinson et al., 1991; Spitzer et al., 1995). Spitzer et al. (1995) defined a new scale, WAD (Whiplash Associated Disorders), for the classification of these injuries into four categories. Spangfort (1985) used Figure 1 to describe the stages of the symptoms. Findings similar to those of Spangfort (1985) were reported by Deans et al. (1987).

According to Svensson (1993), a synthesis of findings by Mertz and Patrick (1967; 1971) and by McConnell et al. (1993) indicate that AIS 1 neck injuries during a rear-end

impact are prevented if the displacement between head and torso are eliminated. The injury can on the other hand occur without hyper extension of the complete cervical spine.

In a study presented by Svensson et al. (1993) and Örtengren et al. (1996) anaesthetised pigs were exposed to swift extension-flexion motion of the cervical spine in order to simulate the trauma to the neck of car occupants involved in rear impacts. In some animals, pressure was monitored in the CNS and other animals were histopathologically examined for signs of injury to the nervous system in the nerve-root region of the cervical and upper thoracic spine.

Signs of nerve cell membrane dysfunction were found in the spinal ganglia, particularly in the lower cervical region. These injuries could be an explanation for most of the symptoms that are typical for patients with an AIS 1 neck injury sustained in a rear-end car collision. Pressure pulses were registered in the CNS with magnitudes of up to about 150 mmHg (20 kPa) during the neck motion, Svensson et al. (1993). These results verified an 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 was in good agreement with the histopathological findings of the study by Svensson et al. (1993) and Örtengren et al. (1996). Absence of obvious abnormalities to vertebrae, ligaments and discs after the induced neck extension trauma indicated that adjacent vertebrae had 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 (Svensson, 1993).

Fig. 2 - The test set-up seen from above. The anaesthetised animal is lying right 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 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.
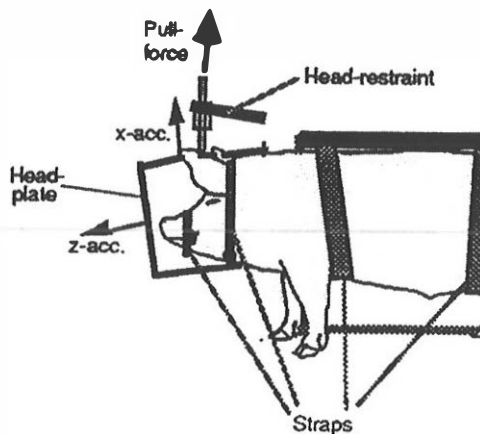

The first aim of the present study was to develop and verify a mathematical model of the transient pressure changes reported by Svensson et al. (1993). The second aim of the study was to investigate pressure phenomena and ganglion injuries at static neck extension loading and dynamic neck extension trauma with a head-restraint present.

MATERIALS AND METHODS

EXPERIMENTAL NECK EXTENSION TRAUMA - Eight anaesthetised pigs, males and females, with body weights of 20-25 kg were used in the present study (Table 1). The Svensson et al. (1993) test set-up was used with some minor modifications (Fig. 2). In some test-runs a head-restraint was introduced. The first animal (HS 01) was exposed to static loading of the cervical spine with load magnitudes corresponding to the dynamic loading

Fig. 3 - Schematic view of four parts of the head-neck motion during a rear-end collision; 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 (from Svensson et al., 1993). The vertical line represents a reference plane in rest.
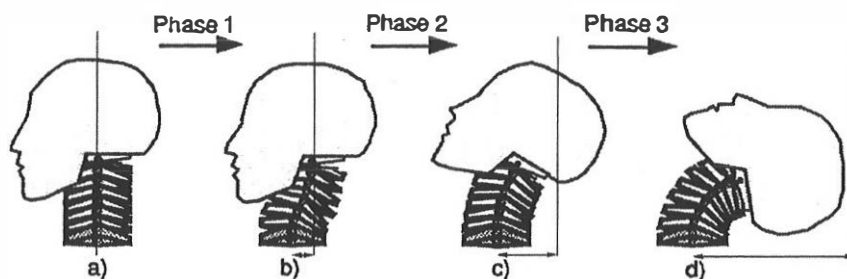
during the extension part of the neck motion of animals HW 01-08 (Svensson et al., 1993). During the first phase of the head-neck motion (Phase 1) the head tends to undergo translational rearward motion relative to the torso without angular displacement, that is retraction (McKenzie, 1993) (Fig 3). At the end of this phase the cervical spine has attained an s-shape, the upper half being fully flexed and the lower being fully extended. At this stage the rearward angular motion of the head is initiated by a combined horizontal (x-direction) shear force and a torque in the occipital joint. The head moment of inertia around the occipital joint was estimated to be 0.01 kgm^2 for a pig of this size and the angular acceleration of the head was estimated to be 840 rad/s^2 during the transition from Phase 1 to Phase 2 (Fig. 3) corresponding to a torque value of 8.4 Nm around the occipital joint. Based on these data the animal's head was pulled rearward, at a point close to the heads centre of gravity (CG), and was at the same time kept horizontal by a lever arm attached to the head plate of the test set-up (Fig. 2) to give the cervical spine a posture similar to that of Figure 3b and to attain the torque value of 8.4 Nm around the occipital joint. This torque was assessed by measuring the force applied to the lever arm 0.5 m from the occipital joint. Thereafter the strap pulling at the heads CG was removed and the animal's head was rotated rearward by means of the lever arm to a maximum extension angle (hyper extension) of 80 degrees. This angle corresponds to a typical maximum extension angle found in tests HW 01-08 in Svensson et al. (1993).

Two animals HC 08-09 were subjected to a slow and gentle extension-flexion motion of the neck.

The animals HW 12-15 were exposed to a single rearward head pull with head-restraint gaps of 100 mm or 130 mm (Table 1).

These animals, HS 01, HC 08-09 and HW 12-15, were treated and histopathologically examined according to the procedure described in Svensson et al. (1993).

Animal PW 03 was used for pressure measurements in the central nervous system (CNS) during

Table 1 - List of the test animals and the tests carried out in the present study. In the column Data, H and P mean Histopathological examination and Pressure measurements respectively (The neck flexion experiments PW 03.01-02 will be presented in a separate publication.)

Animal	Load type	Headrest gap [mm]	Pull force [N]	Data
HS 01	static	-	-	H
HW 12	extens. motion	100	600	H
HW 13	extens. motion	100	600	H
HW 14	extens. motion	130	600	H
HW 15	extens. motion	130	600	H
PW 03.03	extens. motion	100	600	P
PW 03.04	extens. motion	-	600	P
PW 03.05	extens. motion	130	600	P
HC 08	sham exposed	-	-	H
HC 09	sham exposed	-	-	H

Fig. 4 - Two possible critical phase-shifts (occurring at the middle positions) for a rearward motion of the head. In Figure 3, the shift from phase 1 to 2 is an s-shape phase shift. The arrows along the neck indicate the direction of flow.

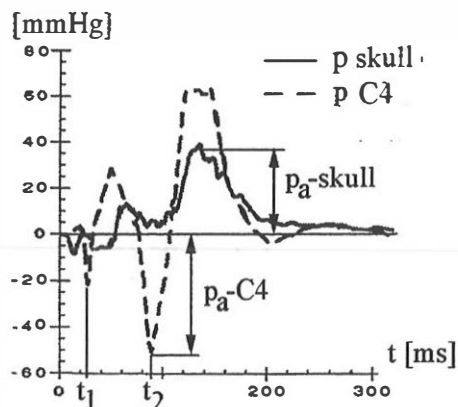Straight phase shift   S-shape phase shift
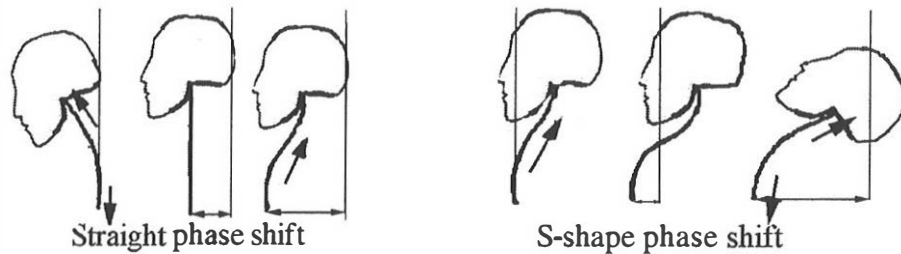
experimental neck extension motion. The animal was exposed to several experimental runs according to Table 1. Two pressure transducers were placed in the subarachnoid space in the cervical spinal canal at two levels according to the same procedure used by Svensson et al. (1993). A pressure transducer was not placed in the skull since this transducer position would interfere with the head-restraint. A head-restraint was placed behind the head. The gaps between the head and the head-restraint were chosen so that the head was arrested just before the neck reached full retraction (100 mm) or just after the head had passed the point of full retraction (130 mm).

MATHEMATICAL MODEL - For any one dimensional system of flowing medium, a quick change of flow direction at a significant flow velocity, may result in a transient pressure shift. In water pipe systems this is often called a "water hammer". For some systems, this shift may be violent enough to cause damage.

As described by Svensson et al. (1993), the blood in the internal venous plexus is most probably forced to flow along the inside of the canal (and it may also flow in and out of the canal via the intervertebral vein bridges) when the cervical spine is flexed or extended. Due to the swift change of volume inside the canal during a typical rearward head motion, blood flow-speed in the order of meters per second may result. Moreover, during the rearward motion of the head (relative to the torso), the neck may pass the phase shifts of the types "straight" and "s-shape" schematically described in Figure 4. This motion will result in a reversal of the flow direction during a time scale of milliseconds. This phenomenon probably caused the pressure transient along the cervical spinal canal seen in the pig experiments by Svensson et al. (1993). The maximum pressure magnitude was registered in the mid section (at the level of the fourth cervical vertebra, C4). The most reasonable estimate of the severity of this "blood hammer", is the amplitude of the pressure, p_a, just before the phase shift (Fig. 5). About 20 ms after the

Fig. 5 - A typical pressure curve at the C4 and skull level of a pig exposed to a rearward head pull (run no. 8 of the pig PW 02, Svensson et al. (1993)). The events of the phase shifts in Figure 4 as well as the amplitude p_a-C4 just before an s-shape phase shift and the response amplitude p_a-skull are shown.

Fig. 6 - The values of p_a-skull versus p_a-C4 for a phase shift of the s-shape type for 13 runs of pig PW 02, Svensson et al. (1993). See also Table 2.
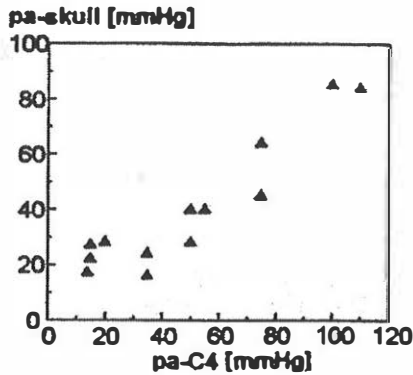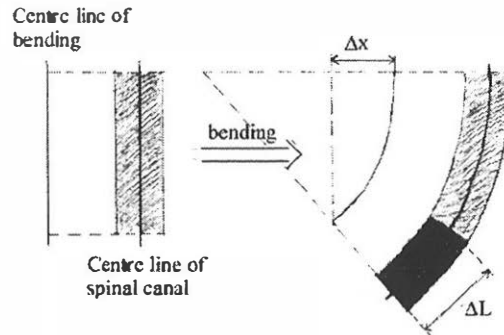

Fig. 7 - When the upper half of the cervical spine is maximally flexed (in a retraction movement), the length is increased a distance ΔL.


event of the sudden transient in the neck, the pressure rises in the skull (Fig. 5). The relation between the value of p_a-C4 and the value of p_a-skull (for a phase shift of the s-shape type) actually seems to be linear according to Figure 6. From now on, the measure of the pressure transient severity will be the value of p_a at the C4-level. The rest of this section describes a mathematical model for the prediction of p_a-C4 for an s-shape phase shift in a pig exposed to a rearward head pull. Now, consider the relative "horizontal" motion in the x-direction (see Fig. 2) of the lowest versus the highest part of the neck (i.e. T1 and C1 respectively). The relative speed in the x-direction, v_{rel}, is then defined as,

$$v_{rel}(t) = \int_{t_0}^{t} a_{rel} dt, \qquad (1)$$

where a_{rel} is the relative acceleration in the x-direction (in the pig tests, a_{rel} is simply the acceleration of the head as the back of the pig is strapped), and t_0 is the time when the speed of flow along the neck is zero (In Fig. 5, t_0 equals $2 \cdot t_1$). The hydrodynamic model in this paper is based on three basic assumptions. Firstly, the blood is considered to flow one-dimensionally either along the spinal canal or in a transverse direction. Secondly, the relative horizontal head-torso displacement is assumed to be proportional to the volume change inside the spinal canal. Geometrically, this is obvious from a simple model of the neck, see Figure 7. Lastly, the speed of flow along the neck is assumed to be caused by the volume change and therefore proportional to its rate.

Consider the flow of blood before an s-shape phase shift (Fig. 4). As explained earlier, due to the co-operative effect of the volume decrease/increase of the lower/upper neck, the blood will flow along the neck "upwards" (as indicated in Fig. 4 and 8). However, there is a possibility of flow both "downwards" along the neck from the lower half, as well as "transversally" due to blood

Fig. 8 - A schematic view of the internal venous plexus of a retracting neck and possible directions of flow at various sites.
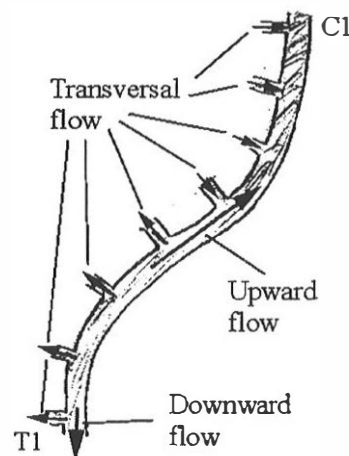
flow through the intervertebral foramina via vein bridges out to the external venous plexus, see Figure 8. Assume now that this "leakage" reduces the flow at the C4-level with a constant factor c_{leak}. Transversally, the capacity of the vein bridges is less than the capacity of the internal venous plexus and even further decreased because of vessel-collapse when the flow is above a certain flow rate (Fung, 1985). Downwards, the flow must be less than upwards just because of the co-operative effect at the middle part of the neck (see above). Therefore, the leak factor should be small, at least in the case of a considerable flow, which justifies the assumption of a constant flow-independent leak factor. Hence, before an s-shape phase shift (i.e. between the times $2 \cdot t_1$ and t_2 in Figure 5, to be explained later), the speed of flow along the neck at the mid section (at the level of C4) becomes,

$$u = c \cdot v_{rel}, \tag{2}$$

where

$$c = (1 - c_{leak}) \frac{A_{sc}}{A_{vp}} \frac{\Delta L}{\Delta x}. \tag{3}$$

The factor A_{sc}/A_{vp} is the inverse relative amount of vein blood in the spinal canal (A_{sc} and A_{vp} are the areas of a transversal section of the total spinal canal and venous plexus respectively). According to radiographs (Lippincott et al., 1976), this factor should be of the order 10. The factor $\Delta L/\Delta x$ is the maximal length change of the upper (or lower) half of the canal divided by the corresponding relative horizontal distance change, see also Figure 7. An estimate yields a value of 1/10. Altogether, the parameter c in (3) should roughly be of the order 1. The flow of blood along the neck may be both turbulent and non-Newtonian (with a non-constant viscosity). However, the time scales involved here are too low for turbulence to develop (Fung, 1984) and the contribution from the viscous nature of the blood is expected to be insignificant (shown later). So, the relevant equation which the blood obeys, should be the one dimensional Navier-Stokes equation for an incompressible non-turbulent Newtonian fluid,

$$\frac{\partial p}{\partial z} = -\rho\left(\frac{\partial u}{\partial t} + \frac{1}{2}\frac{\partial u^2}{\partial z}\right) + \mu\frac{\partial^2 u}{\partial z^2} + X, \tag{4}$$

Here, p is the pressure, z (positive upwards) and t are the spatial and time co-ordinates, μ is the viscosity, ρ is the density and X is the contribution due to the external pressure (including the gravitational force). We will neglect the last contribution mainly for two reasons. Firstly, the model *without* external pressure seems to be enough to explain the pressure curves. Secondly, static displacement comparable to the dynamic simulations was performed for PW 01 and 02, Svensson et al., (1993), without any significant pressure change.

For blood, ρ and μ are about 1050 kg/m³ and $3.5 \cdot 10^{-3}$ kgm⁻¹s⁻¹ respectively. Instead of explicitly solving equation (4), ∂z may be replaced with a typical length scale of 0.1 m (for the pig neck). With all this taken into account, the pressure change until the neck develops an s-shape (i.e. retracts maximally), $-p_a$ in Figure 5, is **roughly** estimated to be,

$$\Delta p \approx -0.1 \cdot 1050(a_{rel} + \tfrac{1}{2}(v_{rel})^2/0.1) + 0.0035 \cdot v_{rel}/(0.1)^2 . \tag{5}$$

Since a injurious motion necessarily involves relative speeds and/or accelerations of the order of meters per second and a number of g's respectively, the viscous contribution is negligible whilst the inertial part of the pressure dominates, according to equation (5). Consider now a relative velocity and acceleration of the head calculated at the time of the transient to be 2 m/s and 6g respectively. These, used in equation (5), give a value of the pressure change of,

$$\Delta p \approx -8 \ kPa \approx -60 \ mmHg, \tag{6}$$

roughly in agreement with the experimental values (see Fig. 5).

Fig. 9 - Mathematical versus experimental value of p_a (C4, s-shape phase shift) for 13 consecutive runs of pig PW 02, Svensson et al. (1993).
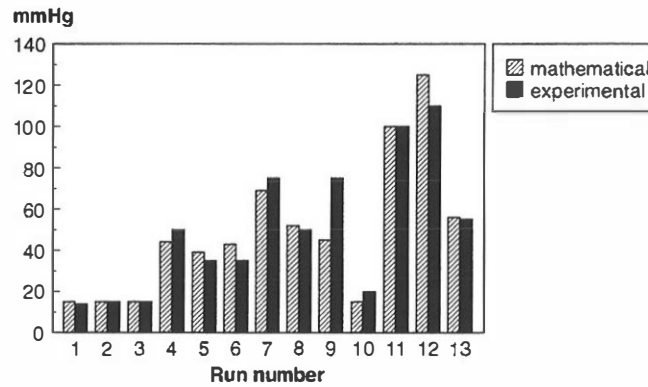
A more strict use of equation (4), provides a mathematical estimate of the pressure transient, the amplitude of pressure at the C4-level just before the occurrence of the "blood hammer", to be,

$$p_a = 1100(0.1 \cdot a_{rel} + \tfrac{1}{2}(v_{rel})^2). \tag{7}$$

Here, the factor 0.1 multiplied with the relative acceleration as well as the factor 1100 in front of the total expression is of the expected order, see the previous estimates, but tuned to agree with the experimental values. In order to evaluate the pig tests in a convenient systematic way, this may be expressed in a different fashion. Consider a pigs head pulled rearward by an almost constant force F, through both types of phase shifts (Fig. 4). The times of the passages of the straight and s-shape phase shifts are named t_1 and t_2 respectively, see also Figure 5. According to the model, the flow at the mid section is zero at the time $2 \cdot t_1$. If there is no straight phase shift, t_1 equals zero. Moreover, the relative acceleration approximately equals the pulling force divided with the effective pull-mass, m_{eff}. Then, the relative speed at the occurrence of an s-shape phase shift, equals $(F/m_{eff}) \cdot (t_2 - 2 \cdot t_1)$. Finally, (7) becomes,

$$p_a \approx 550 \tfrac{F}{8}(0.2 + \tfrac{F}{8}(t_2 - 2t_1)^2). \tag{8}$$

A comparison of the model with the experimental values of p_a-C4 for 14 consecutive runs of one pig, PW 02, (Svensson et al. 1993), is presented in Figure 9, see also Table 2 (data was lost for run PW 02.14). Note that for runs 1-6, 10 and 13 the pull force was the same, however, for no. 4-6 and 13 there was no straight phase shift. The starting position of the neck for the others was a slight flexion resulting in a straight phase shift which actually *lowered* p_a for the s-shape phase shift to its minimal value, the acceleration part (as the speed of flow was zero)

Table 2 - Data and pressure predictions at the mid section of the cervical spine for 13 extension motions of pig PW 02 by Svensson et al. (1993). The evaluations are for an s-shape phase shift (a passage of a maximal retraction).

no	Pull force [N]	t_1 [ms]	t_2 [ms]	v [m/s]	p_a-mod. [mmHg]	p_a-exp. [mmHg]	p_a-skull [mmHg]
1	150	67	114	0	15	14	17
2	150	65	130	0	15	15	22
3	150	65	130	0	15	15	27
4	150	0	140	2.6	44	50	28
5	150	0	128	2.4	39	35	24
6	150	0	139	2.6	43	35	16
7	600	25	67	1.3	69	75	64
8	450	26	88	2.0	52	50	40
9	300	28	106	1.9	45	75	45
10	150	140	160	0	15	20	28
11	750	22.5	70	2.3	100	100	85
12	900	22.5	70	2.8	125	110	84
13	150	0	168	3.2	56	55	40

RESULTS

EXPERIMENTAL NECK EXTENSION TRAUMA - Histopathological examination of animal HS 01, exposed to static loading, revealed no signs of membrane leakage in the cervical spinal ganglia. The same result was found in the two sham-exposed animals HC 08 and HC 09.

The animals in the neck extension experiments, HW 12 and HW 13, with a head-restraint gap of 100 mm, exhibited no signs of membrane leakage in the cervical spinal ganglia. The animals in the neck extension experiments, HW 14 and HW 15, with a head-restraint gap of 130 mm, exhibited membrane leakage in the neurons of the cervical spinal ganglia. The distribution of injured neurons between the spinal levels was very similar to the findings of Örtengren et al. (1996) in both animals.

For the tests of PW 03 without head-restraint and with a head-restraint gap of 100 and 130 mm, the pressure pulses were very similar, except that the pressure went to almost zero at the same time as the rearward head motion was stopped by the head-restraint (Fig. 10). The results of PW 03 without head-restraint are in good correlation with the results of PW 01 and PW 02 by Svensson et al. (1993).

MATHEMATICAL PREDICTION - For the 13 test runs with PW 02 by Svensson et al. (1993), the mathematical value (7) of the negative pressure magnitude just before an s-shape phase shift (Fig. 4,5),

$$p_a = 1100(0.1 \cdot a_{rel} + \tfrac{1}{2} \cdot (v_{rel})^2),$$

correlates well with the experimental values (Fig. 9) as well as with the following pressure rise in the skull (Fig. 6). See also Table 2.

DISCUSSION

EXPERIMENTAL NECK EXTENSION TRAUMA - The static loading of the neck in test HS 01 (Table 1) did not result in any signs of injury to the spinal ganglia. This indicates that the presence of a dynamic component in the head-neck loading is needed to induce ganglion injuries. The finding supports the hypothesis (Aldman, 1986) that the ganglion injuries are caused by the transient pressure gradients measured in the central nervous system during experimental extension trauma (Svensson et al., 1993).

The results of HW 12-15 show that the injuries to the cervical spinal ganglia occur early on in the extension motion, at just about the point of maximum retraction. Provided that the pressure pulse in the spinal canal is the cause of the ganglion injuries, the pressure measurements from the tests

Fig. 10 - The pressure at the C4 level in the cervical spinal canal with a 100 mm head-restraint gap, PW 03.03, and without a head-restraint, PW 03.04.
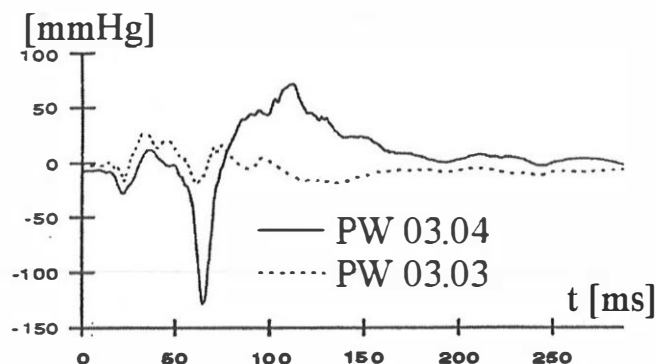
with animal PW 03 show that the injury is caused by the pressure transient[i] that occurs as the neck passes the point of full retraction (67 ms in Fig. 10).

Provided that ganglion injuries of the type found in the present study are identical to the injuries that cause WAD-symptoms in patients following neck extension trauma, the findings of the present study indicate that a head-restraint, in order to be effective, must stop the rearward head-neck motion very early on, before the point of maximum retraction is reached.

CRITERION CANDIDATE FOR A PIG - With the information of the pull force and a time parameter, we were able to predict the pressure amplitude, p_a, at the point of maximal retraction (s-shape), see Figure 5 and equation (8). We also succeeded theoretically explaining the "pull-force & time" formula (8) with a simple but natural hydrodynamic model. We may say that the basic assumptions and the rough estimates of the neck-specific parameters, were justified by this possible explanation. With this theoretical explanation in hand, we may propose a generalisation (7) for other types of motions (with non-constant acceleration). Moreover, the value of p_a seems to predict the risk of ganglion nerve cell injury. Therefore, we propose a candidate for a neck injury criterion for a pig to be,

$$NIC_{pig} = a_{rel} \cdot 0.2 + v_{rel}^2, \tag{9}$$

where a_{rel} and v_{rel} are related to the horizontal C1-T1 motion at the time of a presumed passage of the s-shape. To associate this with something more familiar, compare with the energy of a mass (weight m) 0.1 m above ground with the velocity v,

$$E = mg0.1 + \frac{1}{2}mv^2 = \frac{m}{2}(g0.2 + v^2), \tag{10}$$

where g is the gravitational acceleration. Consider now the tolerance level. In total 11 pigs were exposed to a pulling force of 600 N (Örtengren et al., 1996). All 11 animals revealed clear signs of injury to the spinal ganglia at the histopathological examination. Results of PW 02, Table 2, indicates that the p_a-C4 value would have been about 75 +/-15 mmHg corresponding to a NIC_{pig} value of 18 +/-4 m^2/s^2 (roughly estimated). Histopathological examinations was *not* combined with pull forces less than 600. However, in the tests of the pigs HW 12 and 13 (Table 1) the head was pulled with 600 N through a phase shift of the straight type, but arrested just before the s-shape type. The pigs were not injured and the p_a-C4 value for the straight phase should have been about 45 +/-12 mmHg corresponding to a NIC_{pig} value of about 11 +/-3 m^2/s^2 (according to the tests PW 02, Svensson et al., (1993)). Hence, an exposure leading to an amplitude of about $p_a < 40$ mmHg corresponding to a value of $NIC_{pig} < 10$, seems to be a non-injurious motion.

CRITERION CANDIDATE FOR A HUMAN BEING - Svensson et al. (1993) discussed the validity of the pig as a model of the human head-neck system. They concluded that the pig is a good qualitative model but that it is difficult to make quantitative comparisons. It is for instance difficult in the present study, to make any assumptions about the relation between the pull force to the pig's head and a corresponding Δv in a real rear-end collision. The time from the start to the maximum rearward angular head displacement is similar, about 100 ms, in the pig experiments at 600 N pull force and in volunteer tests in the Δv range 8-16 km/h (Mertz and Patrick, 1967; Tarriere, 1969). The maximum rearward angular displacement is, on the other hand significantly higher in the pig experiments (70-80 degrees) compared to the volunteer tests (40-60 degrees) indicating a higher severity in the pig experiments. With the NIC criterion it is however possible

---

[i]Throughout this study we have assumed the pressure rise following the pressure dip to be the cause of injury. However, if it is the fast pressure dip itself causing the injury (or a combination with the rise), the parameter p_a-C4 and the following criterion-discussion remain relevant.

to make assumptions about the pressure magnitudes in the spinal canal of a human being for a given differential horizontal acceleration between the head and the upper torso, at the moment of maximum retraction of the head.

Therefore, the form of the mathematical expression of the pressure amplitude for a pig (7), should most likely be valid for a human being. A neck injury criterion for a human being could be of the form,

$$NIC = a_{rel} \cdot L + v_{rel}^2, \qquad (11)$$

where L is a length parameter. As the constants appearing in the model, eq. (3), happen to be almost scale insensitive and the

Fig. 11 - In this diagram, a straight line will split up the injured from the not injured, if the NIC

lengths of pig necks are of the same order as for human beings, the value of L for human beings in (11) should be of the same orders as that found for pigs.

When evaluating a rear-end collision, the head, (if it not makes immediate contact with the head rest), more or less *stays at rest* until the neck is retracted maximally (full s-shape), see phase 1 in Figure 3. Therefore, as an approximation, the relative acceleration and velocity in (11) simply becomes the acceleration and velocity of the bottom part of the neck (T1 level).

NIC AND ACCIDENT & VOLUNTEER TEST DATA - So far, the NIC criterion seems promising. Yielding seats are known to be better from an neck injury point of view than non-yielding seats (Martinez, 1968; Foret-Bruno et al., 1991; Warner et al., 1991; Saczalski et al., 1993; Thomson et al., 1993; Digges et al., 1993; Parkin et al., 1995; Håland et al., 1996). A yielding seat automatically lowers the NIC value. Women, who on average have less weight than men, are more often injured. A decrease of the seat-back (recliner) stiffness or an increase of the occupants weight, means lower NIC values (regardless of the values of the length parameter L and of the tolerance level). Also, the observed impacted car-Δv correlates to the neck injury (Krafft, 1996).

Because of this, the theory seems to have a logical support in the accident statistics. Nevertheless, the "NIC-theory", or more specific, a realisation of equation (11), now has to be either falsified, validated or corrected. Instead of investigating biomechanical data from further pig tests or other sorts of laboratory experiments, there is now a chance of trying another approach. Namely, to evaluate NIC values from accident and volunteer test data. For example, consider the diagram in Figure 11 with the relative velocity squared on one axis and the relative acceleration on the other. Further, consider an exhaustive plotting in this diagram of all sorts of motions from accident data, volunteer or dummy tests. These are known to be either injurious or non-injurious from a particular aspect, for example high or low risk of permanent disability (according to accident statistics or case study results or volunteer test results). Suppose that a straight line (with some tolerance of broadness) will split the injurious from the non-injurious motions. Then, the NIC theory is relevant and the tolerance level as well as the value of L is found. If not, the nature of injury has to be re-categorised (e.g. if motions with temporary muscle discomfort were treated as injurious) or the theory has to be abandoned. There have been some efforts in plotting data. Motions consisting of some volunteer tests as well as dummy-interactions with seats from cars that statistically have different risk of injury (according to research data from Folksam Insurance Company (vKoch et al., 1995)) have been
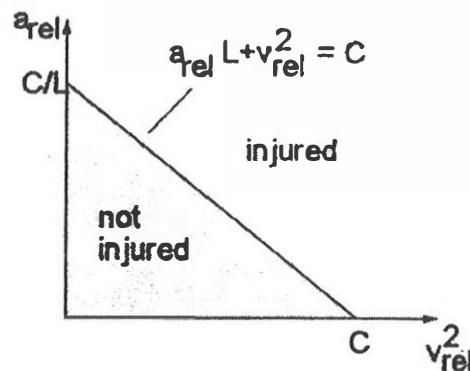
plotted. The result seems promising but will be presented later, see also (Håland et al., 1996). A **preliminary estimate** of the exact form of (11) and a tolerance level yields,

$$NIC = a_{rel} \cdot 0.2 + v_{rel}^2 < 15 \text{ m}^2/\text{s}^2. \tag{12}$$

Please regard this proposal as a starting point for validation, falsification or correction.

In this work, the rearward motion of the head (relative to the torso) has been the focus. A "blood hammer" may well occur also during a forward motion of the head. There will be a phase shift, if for example the neck is forced through a maximal protraction (a forward s-shape). However, the s-shape phase shift (the passage of a maximal retraction) treated in this work seems to be the most important considering the high neck injury incidence in low velocity rear-end collisions. Nevertheless, the phase shift in protraction *can be* relevant in frontal collisions.

ACKNOWLEDGEMENTS

REFERENCES

Aldman, B. (1986): An Analytical Approach to the Impact Biomechanics of Head and Neck. Proc. 30th Annual AAAM Conf., pp. 439-454, LC 64-1965

Carlsson, G.; Nilsson, S.; Nilsson-Ehle, A.; Norin, H.; Ysander, L.; Örtengren, R. (1985): Neck Injuries in Rear End Car Collisions. Biomechanical considerations to improve head restraints. Proc. Int. IRCOBI/AAAM Conf. Biomech. of Impacts, Göteborg, Sweden, pp. 277-289

Digges, K. H., Morris, J. H., Mallaris, A. C. (1993): Safety Performance of Motor Vehicle Seats. SAE paper no. 930348, SAE/SP-93/963, pp. 183-191, ISBN 1-56091-348-7, LC 92-63161

Deans, G.T, Magalliard, J.N.; Kerr, M.; Rutherford, W.H. (1987): Neck sprain - a major cause of disability following car accidents. Injury, Vol. 18, pp. 10-12

Fung, Y.C. (1984): Biodynamics: Circulation. Springer Verlag, New York Inc., ISBN 0-387-90867-6

Foret-Bruno, J.Y.; Dauvilliers, F.; Tarriere, C.; P. Mack (1991): Influence of the Seat and Head Rest Stiffness on the Risk of Cervical Injuries in Rear Impact. Proc. 13th ESV Conf. in Paris, France, paper 91-S8-W-19, NHTSA, USA, DOT HS 807 991, pp. 968-974

Hildingsson, C. (1991): Soft Tissue Injury of the Cervical Spine. Umeå University Medical Dissertations, New Series No 296, ISSN 0346-6612, ISBN 91-7174-546-7

Huelke, D.F.; O'Day, J. (1975): The Federal Motor vehicle Safety Standards: Recommendations for Increased Occupant Safety. Proc. Fourth Int. Congr. on Automotive Safety, pp. 275-292, NHTSA, USA

Håland, Y.; Lindh, F.; Fredriksson, R.; Svensson, M. (1996): The influence of the car body and the seat on the loading of the front seat occupant's neck in low speed rear impacts, Proc. 29th ISATA Conf., Florence, Italy, pp. 21-30

James, M.B.; Strother, C.E.; Warner, C.Y.; Decker, R.L.; Perl, T.R. (1991): Occupant Protection in Rear-end Collisions: 1. - Safety Priorities and Seat Belt Effectiveness. Proc. 35th Stapp Car Crash Conf., pp. 369-377, SAE/P-91/251, SAE paper no 912913, ISBN 1-56091-190-5

Kahane, C.J. (1982): An Evaluation of Head Restraints - Federal Motor Vehicle Safety Standard 202. NHTSA Technical Report, DOT HS-806 108, National Technical Information Service, Springfield, Virginia 22161, USA

Kampen, L.T.B. van (1993): Availability and (Proper) Adjustment of Head Restraints in The Netherlands. Int. IRCOBI Conf. on the Biomech. of Impacts, Sept. 8-10, Eindhoven, The Netherlands, pp. 367-378

Kihlberg, J.K. (1969): Flexion-Torsion Neck Injury in Rear Impacts. Proc. 13th AAAM Ann. Conf., The Univ. of Minnesota, Minneapolis, USA, pp. 1-17

vKoch. M.; Kullgren, A.; Lie, A.; Nygren, Å.; Tingvall, C. (1995): Soft tissue injury of the cervical spine in rear-end and frontal car collisions. Proc. Int. IRCOBI, Conf. on the Biomech. of Impacts, pp. 273-283

Krafft, M.; Kullgren, A.; Lie, A.; Nygren, Å.; Tingvall, C. (1996): Whiplash associated disorder-Factors influencing the incidence in rear-end collisions. Proc. 18th ESV Conf. in Melbourne, Australia, paper 96-S9-O-09

Lippincott, J.B. et al. (1976): Clinical Orthopaedics and Related Research, No. 115, 1976.

Lövsund, P.; Nygren, Å.; Salen, B.; Tingvall, C. (1988): Neck Injuries in Rear End Collisions among Front and Rear Seat Occupants. Proc. Int. IRCOBI Conf. on the Biomech. of Impacts, Bergisch-Gladbach, F.R.G., pp. 319-325

Martinez, J. L. (1968): Headrest and seat back design proposals. STAPP 12 Car Crash Conf. 1968.

McConnell, W. E.; Howard, R. P.; Guzman, H. M.; Bomar, J. B.; Raddin, J H.; Benedict, J. V.; Smith, L. H.; Hatsell, C. P. (1993): Analysis of Human Test Subject Responses to Low Velocity Rear End Impacts. SP-975, SAE paper no. 930889, pp. 21-30, SAE Inc., ISBN 1-56091-360-6

McKenzie, R. A. (1990): The cervical and thoracic spine, mechanical diagnosis. Spinal Publications Ltd, New Zeeland, ISBN 0-9597746-7-X

Mertz, H.J.; Patrick, L.M. (1967): Investigation of the Kinematics and Kinetics of Whiplash. Proc. 11th STAPP Car Crash Conf., Anaheim, California, USA, pp. 267-317, SAE Inc., New York, USA, LC 67-22372

Mertz, H.J.; Patrick, L.M. (1971): Strength and Response of the Human Neck. Proc. of 15th Stapp Car Crash Conf., pp. 207-255, SAE Inc., New York, LC 67-22372

Nygren, Å. (1984): Injuries to Car Occupants - Some Aspects of the Interior Safety of Cars. Akta Oto-Laryngologica, Supplement 395, Almqvist & Wiksell, Stockholm, Sweden, ISSN 0365-5237

Nygren, Å; Gustafsson, H., Tingvall, C. (1985): Effects of Different Types of Headrests in Rear-End Collisions. 10th International Conference on Experimental Safety Vehicles, pp. 85-90, NHTSA, USA

Olsson, I.; Bunketorp, O.; Carlsson, G.; Gustafsson, C.; Planath, I.; Norin, H.; Ysander, L. (1990): An In-Depth Study of Neck Injuries in Rear End Collisions. Proc. 1990 Int. IRCOBI, Conf. on the Biomech. of Impacts, Bron, Lyon, France, pp. 269-282

O'Neill, B.; Haddon, W.; Kelley, A.B.; Sorenson, W.W. (1972): Automobile Head Restraints: Frequency of Neck Injury Insurance Claims in Relation to the Presence of Head Restraints. Am J Publ Health, Vol. 62, No. 3, pp. 399-406

Ono, K. and Kanno, M. (1993): Influences of the Physical Parameters on the Risk to Neck Injuries in Low Impact Speed Rear-end Collisions. Int. IRCOBI Conf. on the Biomech. of Impacts, Sept. 8-10, Eindhoven, The Netherlands, pp. 201-212

Örtengren, T.; Hansson, H.-A.; Lövsund, P.; Svensson, M.Y.; Suneson, A.; Säljö, A. (1996): Membrane Leakage in Spinal Ganglion Nerve Cells Induced by Experimental Whiplash Extension Motion: A Study in Pigs. J. Neurotrauma, vol.13, no. 3

Otremski, I.; Marsh, J.L.; Wilde, B.R.; McLardy Smith, P.D.; Newman, R.J. (1989): Soft Tissue Cervical Spinal Injuries in Motor Vehicle Accidents. Injury, Vol. 20, pp. 349-351

Parkin, S.; Mackay, G.M.; Hassan, A.M.; Graham, R. (1995): Rear end collisions and seat performance -To yield or not to yield, 39th annual proceedings-AAAM, pp. 231-244

Saczalski, K.J.; Syson, S.R.; Hille, R.A.; Pozzi, M.C. (1993): Field Accident Evaluations and Experimental Study of Seat Back Performance Relative to Rear-Impact Occupant Protection. SAE paper no. 930346, SAE/SP-93/963, pp. 129-142, ISBN 1-56091-348-7, LC 92-63161

Spangfort, E. (1985): Klinisk Bedömning av Whiplash patienten. In: Nackskadesymposium, Åre Sjukhus, 12 febr., pp. 71-77, FOLKSAM FoU, R 0619, S-106 60 Stockholm, Sweden.

Spitzer, W.O. et al. (1995): Scientific Monograph of the Quebec Task Force on Whiplash-Associated Disorders: Redifining "Whiplash" and Its Management. Spine, Supplement, Volume 20, Number 8S, ISSN: 0362-2436

States, J.D.; Balcerak, J.C.; Williams, J.S.; Morris, A.T.; Babcock, W.; Polvino, R.; Riger, P., Dawley; R.E. (1972): Injury Frequency and Head Restraint Effectiveness in Rear-End Impact Accidents. Proc. 16th Stapp Car Crash Conf., pp. 228-245, Soc. of Automotive Eng., New York, LC 67-22372

Svensson, M.Y. (1993): Neck Injuries in Rear-End Car Collisions - Sites and Biomechanical Causes of the Injuries, Test Methods and Preventive Measures. Dept. of Injury Prevention, Chalmers Univ. of Techn., S-412 96 Göteborg, Sweden, ISBN 91-7032-878-1;

Svensson, M. Y.; Aldman, B.; Lövsund, P.; Hansson, H. A.; Seeman, T.; Suneson, A.; Örtengren, T. (1993): Pressure Effects in the Spinal Canal during Whiplash Extension Motion - A Possible Cause of Injury to the Cervical Spinal Ganglia. SAE paper no. 1993-13-0013, Proc. 1993 Int. IRCOBI, Conf. on the Biomech. of Impacts, pp. 189-200, Eindhoven, The Netherlands

Tarriere, C.; Sapin, C. (1969): Biokinetic Study of the Head to Thorax Linkage. Proc. 13th Stapp Car Crash Conf., pp. 365-380, SAE, New York, LC 67-22372

Thomson, R. W. et al (1993): Dynamic requirements of automobile seat backs, SAE SP-963

Warner, C. Y. et al. (1991): Occupant protection in rear-end collisions: II. The role of seat back deformation in injury reduction, Proc. 35th Stapp Car Crash Conf., pp. 379-390, SAE/P-91/251, SAE paper no 912914, ISBN 1-56091-190-5

Watkinson, A.; Gargan, M.F.; Bannister, G.C. (1991): Prognostic Factors in Soft Tissue Injuries of the Cervical Spine. Injury, 22, (4), pp. 307-309