A Theoretical Model for and a Pilot Study regarding Transient Pressure Changes in the Spinal Canal under Whip-lash Motion

M.Y. Svensson, B. Aldman, P. Lövsund
Department of Injury Prevention
Chalmers University of Technology
Göteborg, Sweden

T. Örtengren, T. Seeman
Department of Surgery
East Hospital, University of Gothenburg
Göteborg, Sweden

R 005

Göteborg, Sweden, 1989

CONTENTS

Page

1 INTRODUCTION

Injuries to the neck have been an increasing problem in recent years in Sweden. The largest contribution to this increase comes from road traffic accidents and rear end car collisions in particular, which give rise to the so called whip-lash injuries. Even at low impact velocities[1] in rear end collisions with passenger cars, neck injuries appear and, according to Nygren (1984), some 10% of the cases give permanent problems with at least 10% disability. In other types of car accidents 4% of the car occupants are permanently disabled with at least 10% disability (Nygren,1984). The injury risk for a car occupant in a rear end colllision is some 50% less in the rear seat than in a front seat (Lövsund et al.,1988). Head restraints in modern passenger cars give poor protection against whip-lash injuries (Nygren et al.,1985; Olney and Marsden,1986).

Whip-lash injuries to the neck and cervical spine consists of conditions on a scale from paralysis (tetraplegia) to unaffected mobility but with symptoms like dizziness, headaches, hyperestesia, pricking fingers and pain in neck, shoulders and arms (States,1979; Nygren et al.,1985). Even at low velocity rear end collisions the car occupants often loose consciousness immediately after the collision even when no signs of impact to the head can be found.

These symptoms are probably caused by damage to nervous tissue. The symptoms often occur even though no signs of skeletal injury or injury to the vertebral disks or ligaments can be diagnosed (Maimaris et al.,1988). The mechanisms causing damage to the nervous tissue but leaving the surrounding tissues virtually unaffected have not yet been given a satisfactory explanation.

In earlier efforts to explain whip-lash injuries, for instance by Mertz and Patrick (1971), the main interest has been directed towards the vertebra, discs and ligaments and their response and tolerance to injury. The predominant theory of the injury mechanisms for the nervous tissue of the central nervous system (CNS) has been mechanical stretching and compression of cervical nerve roots (McMillan and Silver, 1987).

A new model for explaining these injuries was presented by Aldman (1986). He claimed that the pressure gradients, caused by fluid motion under the shortening of the spinal canal at extension movement, give rise to damage to the nervous tissue of the CNS.

The aim of this project is to develop a theoretical model and to determine the pressure phenomena in the CNS under whip-lash motion and their effects on the

---

[1] In this paper rear end collisions, involving two passenger cars, where the initial velocity of the impacting vehicle does not exceed *30 km/h* and the impacted vehicle initially is standing still are considered *low velocity* collisions.

nervous system according to the hypothesis presented by Aldman.

2 A THEORETICAL MODEL

2.1 The Whip-lash Motion

In a rear end car collision the struck car is accelerated. This means that the car occupant is pushed forward by the seat back. If the seat is equipped with a head-rest there is usually space between the skull and the head-rest. This means that the skull, due to its inertia, tends to lag behind when the trunk is accelerated forward (Martinez and Garcia, 1968; White and Panjabi, 1978). An extension motion of the neck will follow. This motion is abruptly interrupted either when the head is reached by the head-restraint or when the head and neck reach the maximum extension angle (*Fig. 1*). The motional scenario described above is in the litterature called *whip-lash motion.*

In one example of a low velocity (13 km/h) impact with a human subject, the head and shoulder accelerations had durations of 130 ms and 200ms respectively (Severy et al.,1955; McKenzie and Williams,1971).

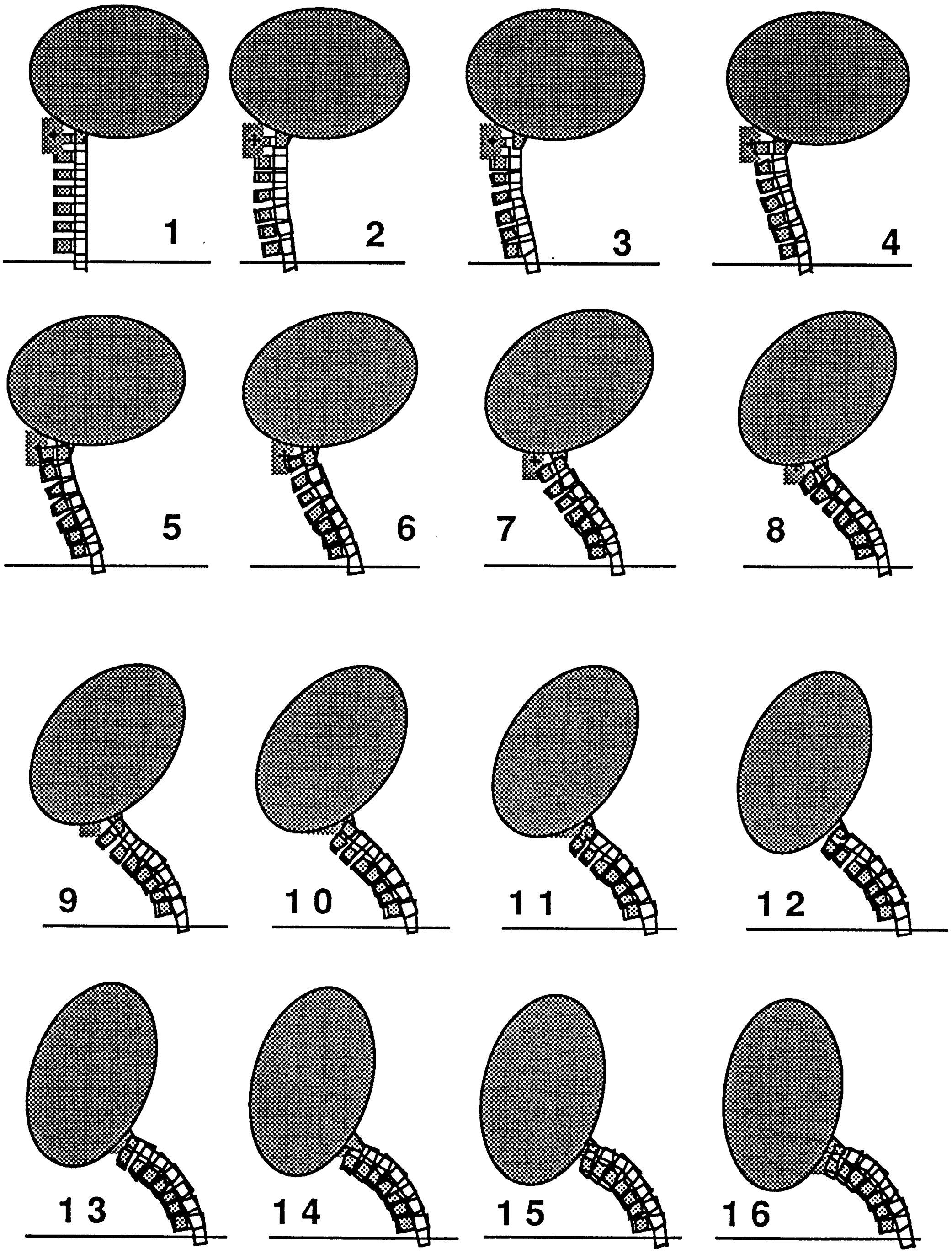


Figure 1 The whip-lash motion

2.2 The CNS from a Hydro Mechanical Point of View

The CNS, i.e. the brain and the spinal cord, is situated in a container consisting of the cranial cavity and the spinal canal. The CNS is floating in the cerebro spinal fluid (CSF) inside this container (*Fig. 2*).

To get an understanding of the hydromechanical behaviour of the CNS and its ambient tissues, when involved in dynamic processes with short duration (tenths of a second), the following approach is made:

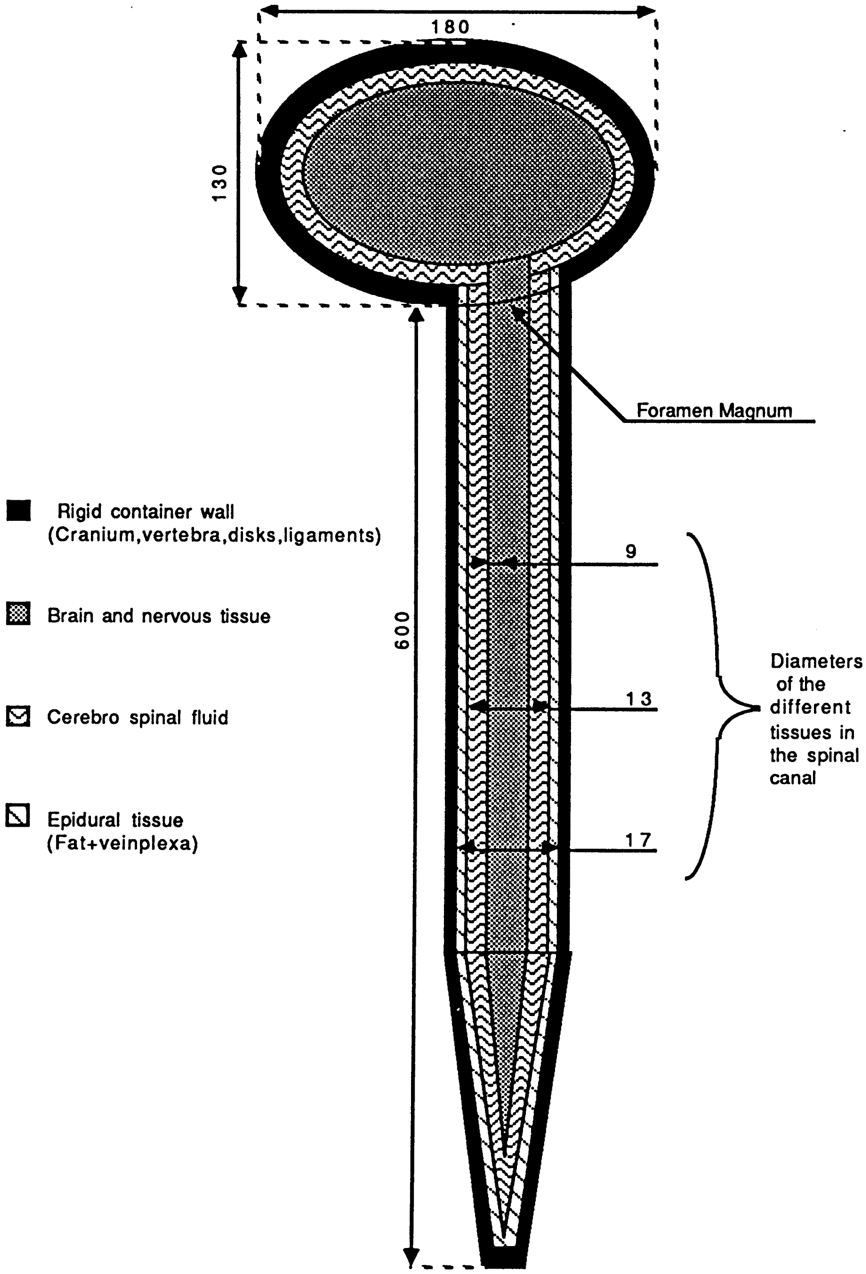


Figure 2 Schematic picture of the CNS (mm).

A *simplified* model of the cranial cavity and its contents can be described as follows:

The cranial cavity is considered a rigid and impermeable container with one large opening, the foramen magnum. All other openings in the cranial vault are relatively much smaller than the foramen magnum and almost no fluid is expected to penetrate these small openings under the very short duration of the whip-lash motion.

The brain and CSF are of almost identical density (Margaria,1953) so they will act as one homogenous medium. We consider this medium to be incompressible (Estes and McElhaney,1971), thus no net fluid flow will take place through the foramen magnum.

Pressure gradients inside the container only occur due to acceleration forces since we consider velocities far below the velocity of sound in bodily tissues. Unless the head is submitted to rotational acceleration there will be no fluid flow inside the cranial cavity. It must be emphasized that the simplified assumptions above only consider processes taking place under the short duration of a whip-lash motion.

(Emphasis must be put on the fact that this is a model with considerable simplifications. Its purpose is to make the theoretical analysis of the flow-pressure phenomena of the cervical spinal canal less complex. The model is unlikely to be fully applicable in an analysis of the injury mechanisms inside the skull when, for instance, impact to the skull is studied.)

A *simplified* model of the spinal canal and its contents can be described as follows:

The spinal canal consists of the vertebral foramina with intervertebral disks and ligaments filling the interspaces forming a tube which is radially rigid but axially flexible. The inside of the canal is filled with different coaxially oriented tissues (*Figs 2 and 3*).

The spinal cord in the center of the spinal canal mainly consists of nervous tissue. It is surrounded by the CSF. The epidural tissue is situated peripherally. Between the epidural tissue and the CSF lies a flexible impermeable wall, the dura mater.

The epidural tissue consists of fat with a lattice of inter connected veins, the veinplexa (Parke,1982). The epidural tissue can easily change its volume within a certain range by changing the volume of blood in the vein vessels (Löfgren,1973). The veinplexa have cross connections with veins outside the spinal canal in the interspace between every pair of vertebra (Crosby et al.,1962). Blood can also easily move along the spinal canal in the veinplexa.

Nerve roots spring out from the spinal cord and pass through each interspace between the vertebrae. The dura mater is forming a meningeal tube around every nerve root. Outside the spinal canal the meningeal tube is attached to the epidermium of the nerve root forming an impermeable sack around the nerve root at its intervertebral passage. The nerve root is floating in CSF inside this sack (*Fig 3*).

It is possible that the volume of CSF inside the spinal canal can be altered if the CSF flows through the meningeal tubes and alters the size of the tube at its end outside the intervertebral passage.

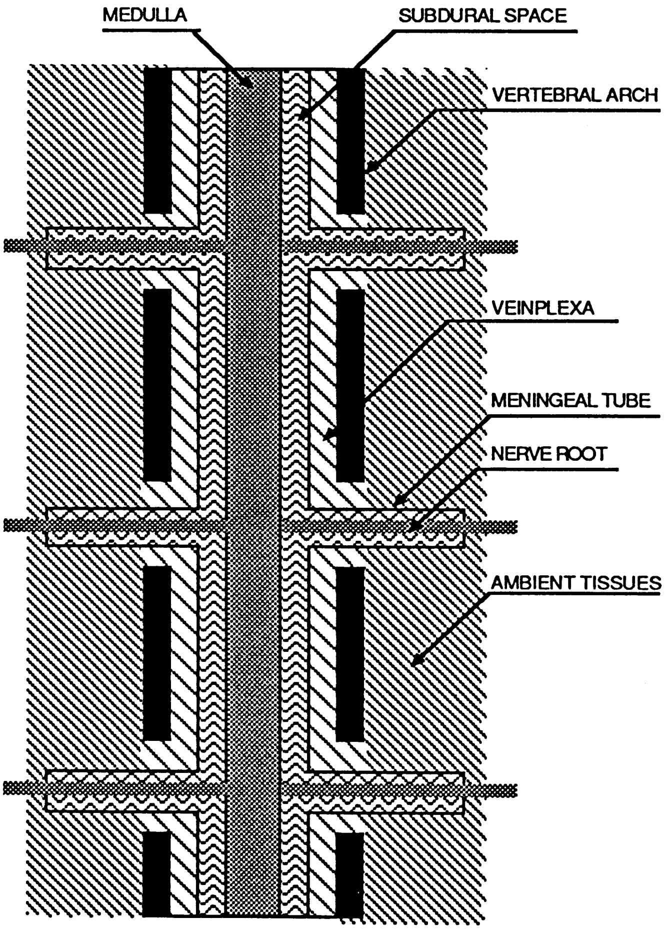


Figure 3 Schematic view of the tissues of the spinal canal.

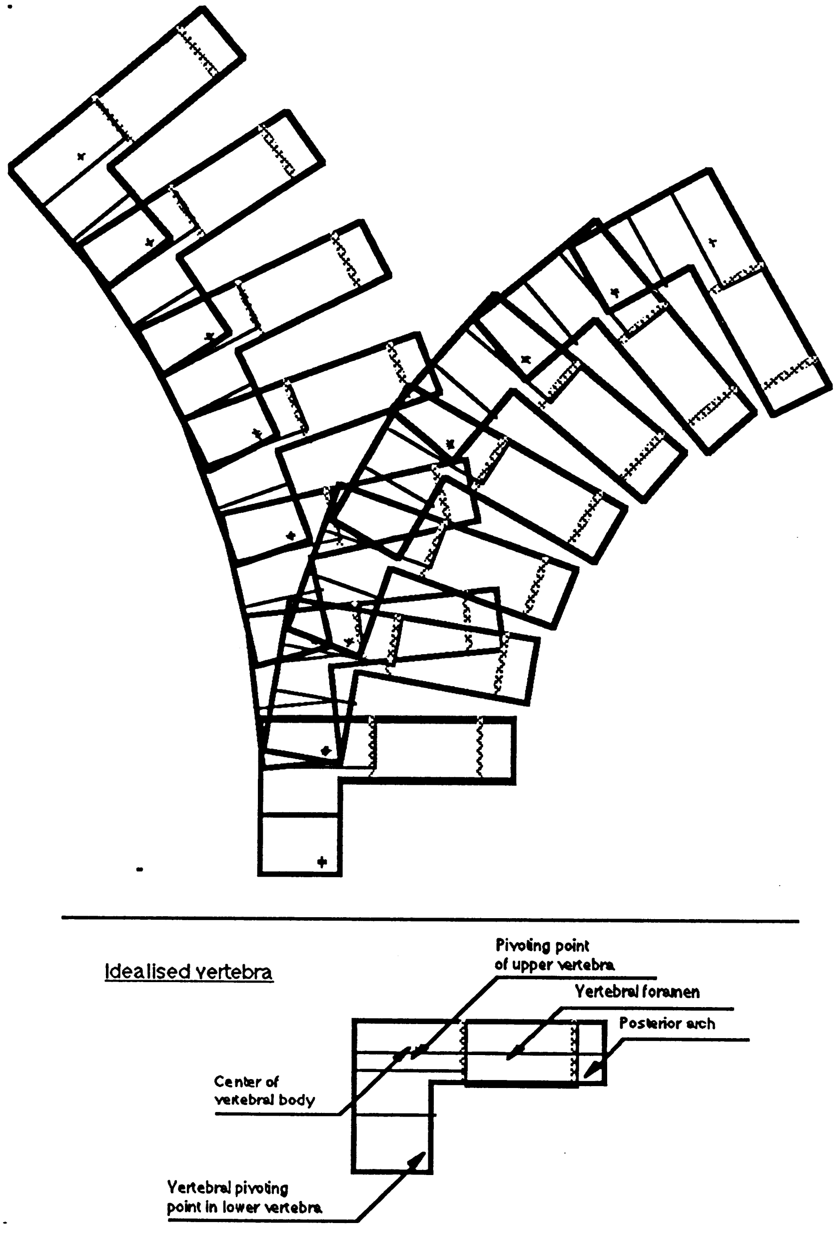


Figure 4 Schematic view of the cervical spine in full flexion and full extension respectively.

2.3 Volume Change Inside the Spinal Canal

The length of the spinal canal will increase at flexion of the neck and decrease at extension. This occurs since every vertebra is pivoting around a virtual axis in the vertebral body of the closest inferiorly laying vertebra (Kapandji,1974) and the spinal canal passes through the vertebral foramina posteriorly to the column of vertebral bodies (*Fig.4*). Breig (1978) describes one case where the cervical spine of a human cadaver was moved from maximal flexion to maximal extension, resulting in a change of the length of the canal of about 30mm. The cross sectional area of the vertebral foramen in the lower neck can be estimated to 200-250mm^2. Thus the maximal volume change inside the canal in this case would be about 7ml. Since the spine consists only of tissues and fluids that can be considered incompressible, the change of volume in the spinal canal ought to correspond to a fluid exchange between the inside and outside of the canal. This means a change of the volume of either the CSF or of the blood in the veinplexa, or both.

2.4 Pressure Mechanisms

The spinal canal: The whip-lash motion is swift compared to the maximal physiological speed of motion. We anticipate that both flow speeds and flow accelerations in the spinal canal must be higher under the whip-lash motion than under physiological conditions. We expect that this induces markedly higher pressure gradients in the spinal canal under whip-lash motion compared to physiological conditions.

To make a simple analysis of the possible flow-pressure mechanisms in the spinal canal the following *simplified approach* is made:

> No distinction between blood and CSF is made. The bending of the canal is omitted and the cross sectional area is considered to be circular. The influence of gravitational forces is omitted.
>
> With the model of the cranial cavity and its contents as described above, the net fluid flow through the cavity is zero. Thus, at the cranial end of the spinal canal the foramen magnum can be considered to be a rigid wall.
>
> In other words, we consider the spinal canal to be a straight circular pipe, radially rigid but axially flexible. Both ends of the pipe have rigid walls. Fluid exchange between the inside and outside of the pipe can take place through holes in the pipe wall at levels along the pipe that correspond to the intervertebral vein connections and meningeal tubes.

In the following approach we consider the first part of the whip-lash motion where the neck is moved from an uppright position to full extension. The motion corresponds to a caudal movement of the wall at the cranial end of the pipe. Pressure phenomena according to the following three points are possible:

1) Consider a short moment of the whip-lash motion under which the cranial end moves caudally with *constant velocity* so that the inner volume of the pipe

decreases. When fluid is forced to flow out through the holes in the pipe wall a relative pressure increase inside the pipe arises due to flow resistance in the holes.

2) Consider, as in 1), a short moment of the whip-lash motion during which the cranial end moves caudally with *constant velocity*. A fluid flow in caudal direction is induced with caudally decreasing flow velocity due to successive outflows along the pipe through the holes. Due to flow resistance, a flow velocity dependent pressure gradient along the pipe is induced. Thus, this gradient decreases successively caudally.

3) When the cranial end of the pipe is *accelerated* caudally the fluid column in the pipe will be accelerated caudally. Due to the successive outflows along the pipe the acceleration will decrease caudally and at the caudal end it will be zero. The acceleration will induce a pressure gradient along the pipe that is decreasing caudally. In the later part of the whip-lash motion the cranial end of the pipe will be decelerated, which means that this pressure gradient will change sign.

In a real rear end car collision these three pressure phenomena will be added the initial natural pressure inside the spinal canal of a sitting car occupant. A pressure gradient due to the horizontal acceleration of the whole body will also be added. When considering the pressure gradient from the whole body acceleration, the bending of the spine should not be omitted.

The skull: According to the previously presented simplified model of the skull, the pressure inside the cranial vault will be constant and equal to the pressure in the spinal canal at the foramen magnum, when the body is at rest and if gravity is omitted. This pressure will be added a pressure gradient inside the skull due to gravitation and acceleration

2.5 Injury Mechanisms

On the basis of the model presented, the following possible injury mechanisms can be deduced:

a) The pressure gradient directly influences the spinal cord and the nerve roots in the spinal canal.

b) The pressure gradient influences on the nerve roots in the meningeal tubes caused by swift pressure changes at expedited cerebro spinal fluid flow through the tubes.

c) Ruptures at the end of the meningeal tube, where the dura mater connects to the epidermium. Spinal fluid is forced to flow through the tube due to the increased pressure in the spinal canal during the whip-lash motion and the outer end of the meningeal tube is filled up. The dura mater as well as the epidermium and the nerve itself might rupture.

d) The unconsciousness that often follows immediately after a rear end collision might be induced by the rapid pressure change at foramen magnum that momentarily spreads through the hole of the skull cavity and might affect the function of the brain.

3 MATERIALS AND METHODS

3.1 Pressure Measurement In a Mechanical Model

A mechanical model of the spinal canal has been built. When more data on the qualities of the soft tissues in the human spinal canal is attained and materials that can simulate these qualities well are found a more refined version of the model will be designed. The model is aimed to give more refined measurement where single parameters can be studied isolated from others and with good repeatability. The results could be used when planning for animal tests.

The present mechanical model is based on a circular metal pipe with the same inner cross sectional area (227 mm^2) as a typical human spinal canal. Both ends of the pipe are closed with rigid plugs. The cranial plug is movable so that the length of the canal can be altered. In the pipe wall, holes resembling cross connecting veins and meningeal tubes, have been drilled. The distances between the holes and the length of the pipe are similar to those of a typical human. The pipe is filled with water. The model is sunk into a water bath which simulates the surrounding soft tissues.

The depth in the bath gives a realistic pressure in the canal. No distinction between blood and CSF is made in this first approach. A rapid movement of the movable cranial plug gives a rough qualitative simulation of the pressure buildup taking place when the head is swiftly moved in the sagittal plane.

3.2 Pressure Measurements In Animal Models

In order to be able to measure the pressure phenomena in the spinal canal under whip-lash motion we have used pigs as animal models. They are easily available at a relatively low cost and the different dimensions of the spine are fairly similar to those of the human.

Pressure in the spinal canal and inside the skull of anesthetized pigs has been measured. When the measurements were made, heads and necks of the pigs were subjected to simulated whip-lash motion. The major purpose of these tests was to gain experiences in order to develop more refined test equipment as well as test procedures.

An initial test series of four anesthetized pigs has been made. The body mass of the animals where in the range of 35-70 kg.

Experimental equipment:

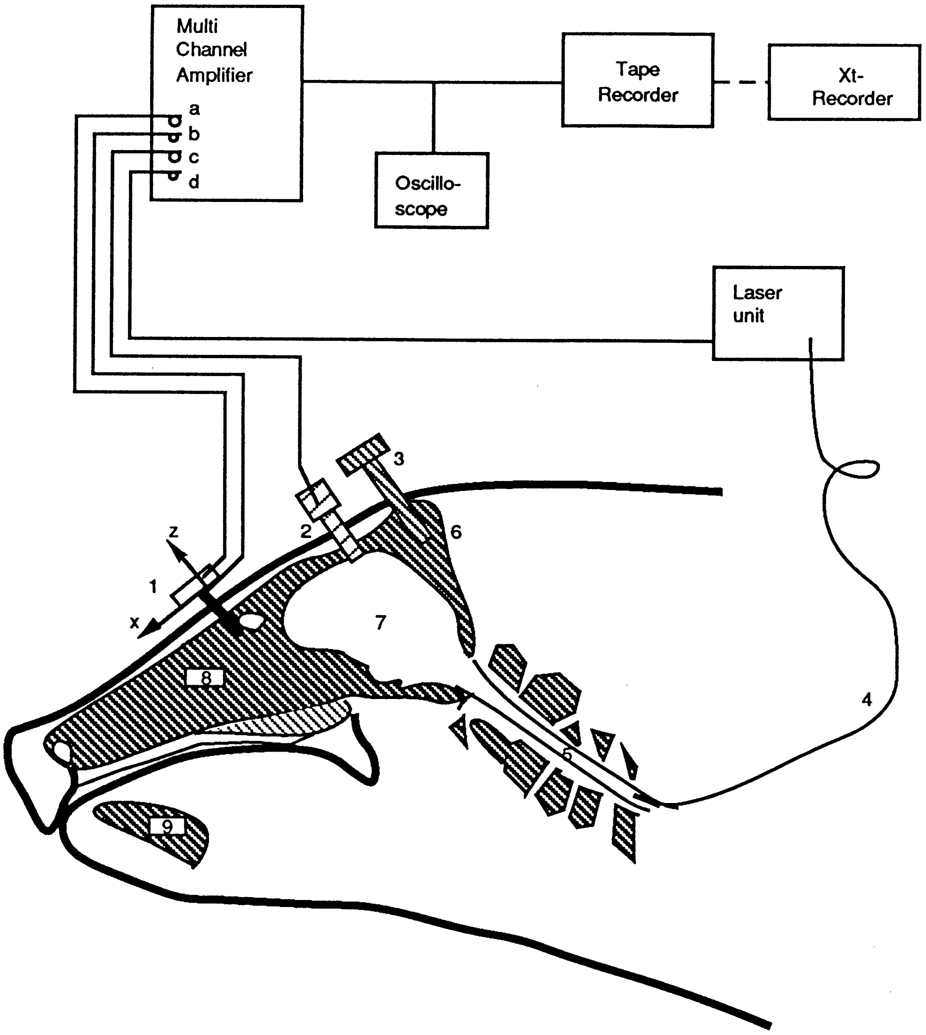
An optical pressure sensor was placed in the subdural space in the spinal canal at the level of T1. The sensor which consists of an optical fiber with a sensing probe at its peripheral end (Tenerz and Hök,1988) was introduced into he subdural space at the level of T12 and guided to the level of T1. The probe and its fiber have a diameter of 0.46mm.They where placed inside a catheter (18G catheter made for epidural injections) with an outer diameter of about 1mm. The catheter made it easier to guide the sensor through the subdural space. The free space inside the catheter was filled with saline to ensure that the surroundings of the probe did not contain air.

A hole was drilled through the cranial vault and a piezo resistive pressure transducer (Endevco 8510-100) was screwed into the hole (Fig. 5). The transducer measured the intracranial pressure. Efforts were made to avoid air entering the cranial cavity.

Two accelerometers (Entran EGA 125-100 DSC) were attached to the skull. They were mounted on a small metal plate which was screwed to the nasal bone. The measuring directions of the accelerometers where parallel to the sagittal plane. One accelerometer parallel and the other perpendicular to the nasal bone.

A wire was screwed to the nuchal crest of the skull. The wire was connected to a specially designed "pulling device". A torsional spring was used as power source for this device.

As an alternative to the pulling device, the head was pulled by hand which gave lower acceleration pulses with longer duration. When pulling by hand we where also able to expose the neck to several consecutive whip-lash movements in a short time interval and also to make fast flexion motions.



Fifure 5 The pigs head and neck with instrumentaion and the signal acquisition equipment.

1. Accelerometers (Entran EGA 125-100 DSC).
2. Pressure transducer (Endevco 8510-100).
3. Screw for attachment of pulling device.
4. Optical fiber with pressure sensing probe.
5. Spinal canal.
6. Nuchal crest.
7. Cranial cavity.
8. Skull.
9. Mandible.

Multi Channel Amplifier	- Johne+Reilhofer 8 MV 1
Oscilloscope	- ITT OX750 digital oscilloscope.
Tape Recorder	- Brüel & Kjær 7003.
Xt Recorder	- PHILIPS PM 8272-XYt recorder.
Laser unit	- RADIsensor

4 RESULTS

In animals no. 2 and no.4 all measurements were successful. The body mass of the animals in these two tests were 50kg and 70kg respectively. Apart from the simulated whip-lash motions with the pulling device on anesthetized animals we tried a few modified test procedures. For the 2:nd animal the pulling device was disconnected and motions in both extension and flexion were made manually. Immediately after the animal had been put to death these procedures where tested again.

Figures 9-12 contain curves from animal no. 2, that were recorded less than five minutes after the animal had been sacrificed and where the head was pulled manually.

Cyclic pressure changes were observed both at spinal and cranial level when the animal was at rest (fig.6). The figure also shows an acceleration pulse and the superimposed pressure peaks which almost instantly follows in the CSF.

The same acceleration pulse and pressure peaks extended in time scale are shown in figure 7. The X-acceleration is parallel to the nasal bone and the Z-acceleration is in the normal direction. The dashed lines in the pressure curves show the undisturbed contour of a previous period of the cyclic pressure changes in the CNS. The two acceleration curves have almost identical information content concerning the timing of different events in the whiplash motion. This is typical for all the tests. Thus we have chosen to display only the x-acceleration curve in the following figures.

Figure 8 shows the same parameters as Figure 7 but from a test with another pig with larger body mass. Note that the shape of the acceleration curve is rather different to the one in Figure 7 and so for the pressure pulses.

Figures 9-12 contain curves which were recorded after the animal had been sacrificed. The head was pulled manually.

Figure 9 shows three consecutive extensions.

Figure 10 shows three consecutive flexions and Figures 11 and 12 shows the first of the three pulses of Figures 9 and 10 respectively but in extended time scale.

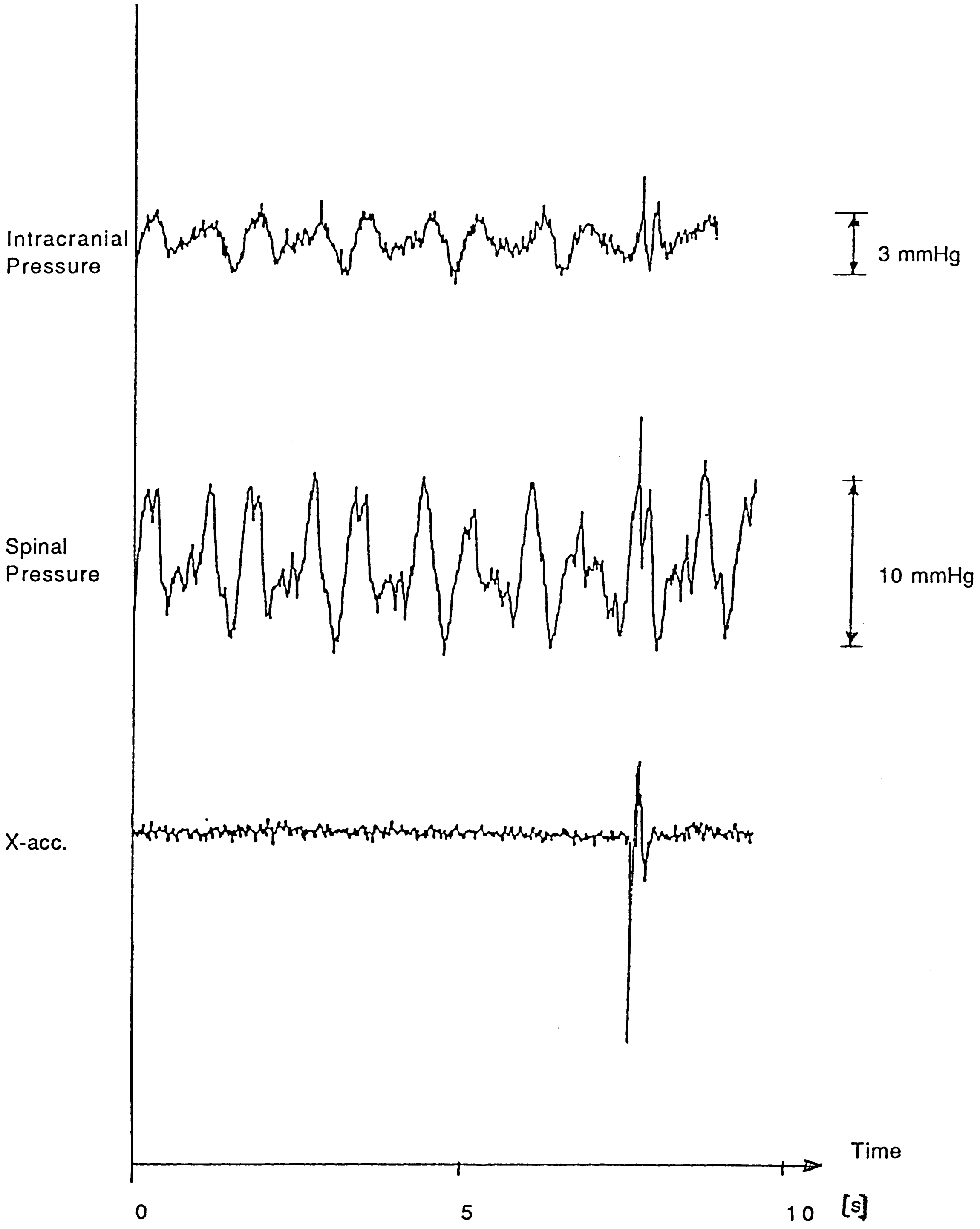


Figure 6 The cyclic pressure curve in the CNS and one superimposed pressure pulse from a whip-lash motion.

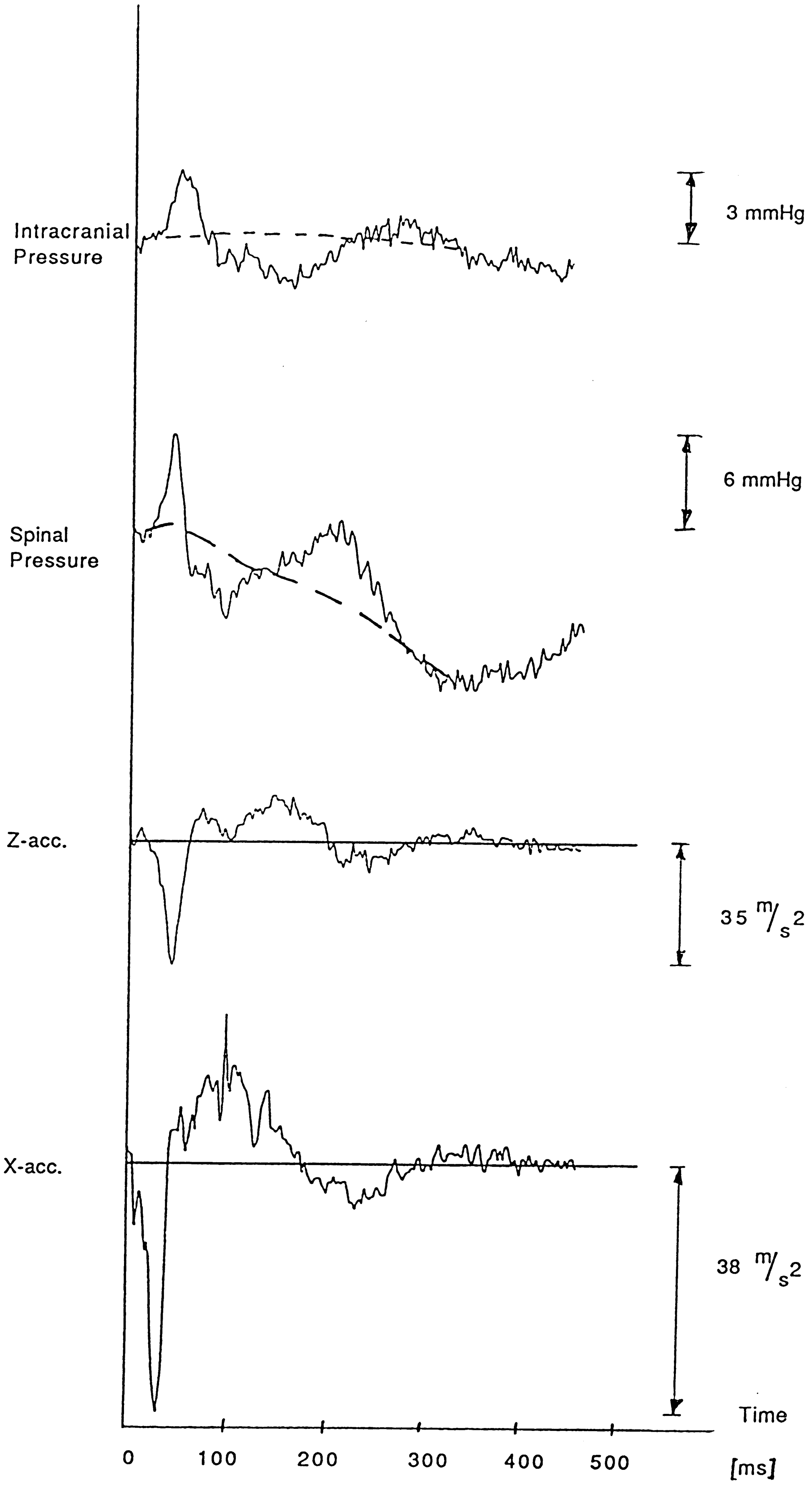


Figure 7 The superimposed pulse of Figure 6, extended in time scale.

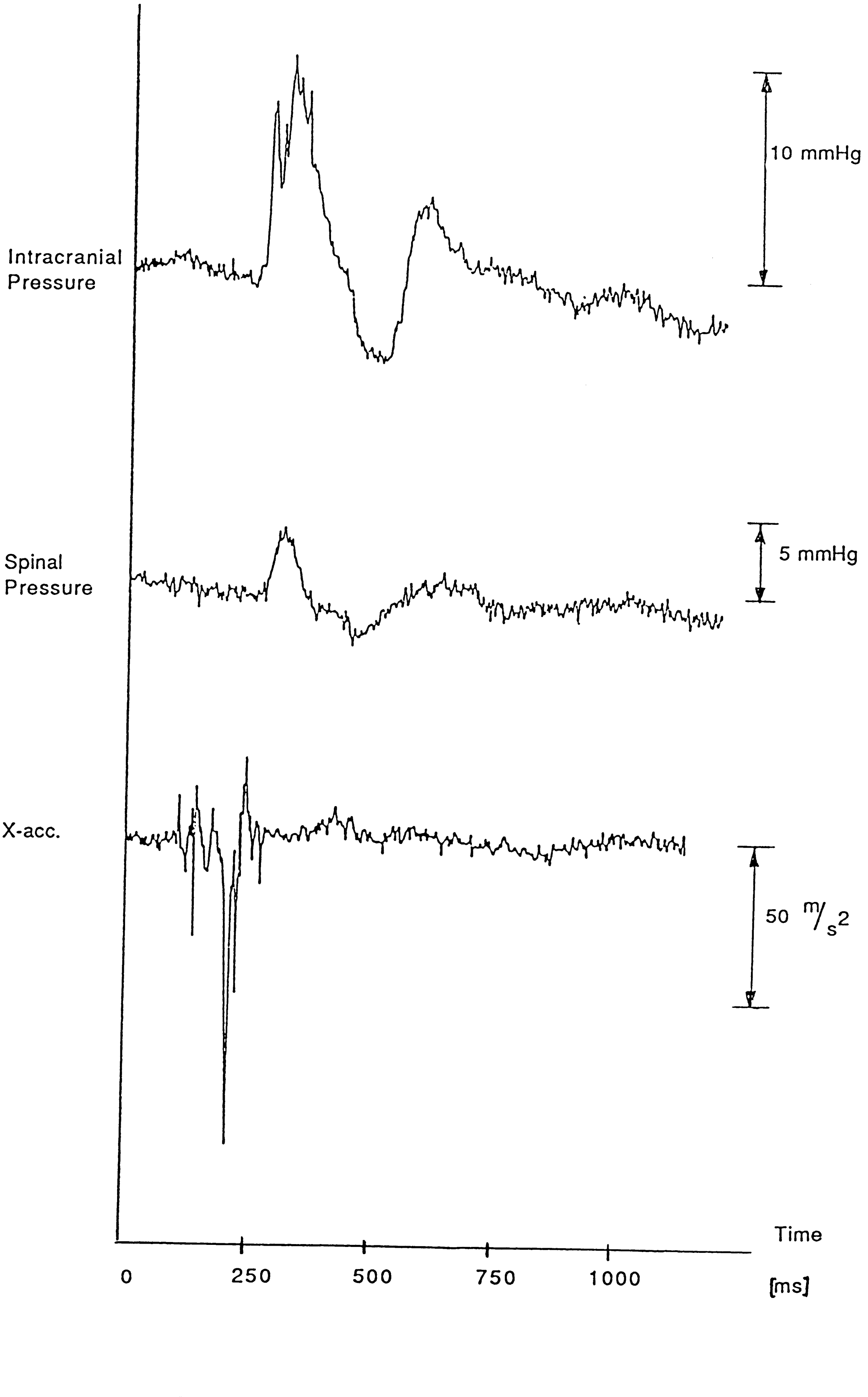


Figure 8 The same measurements as in Figure 6 but taken from a larger animal and with a different acceleration pulse

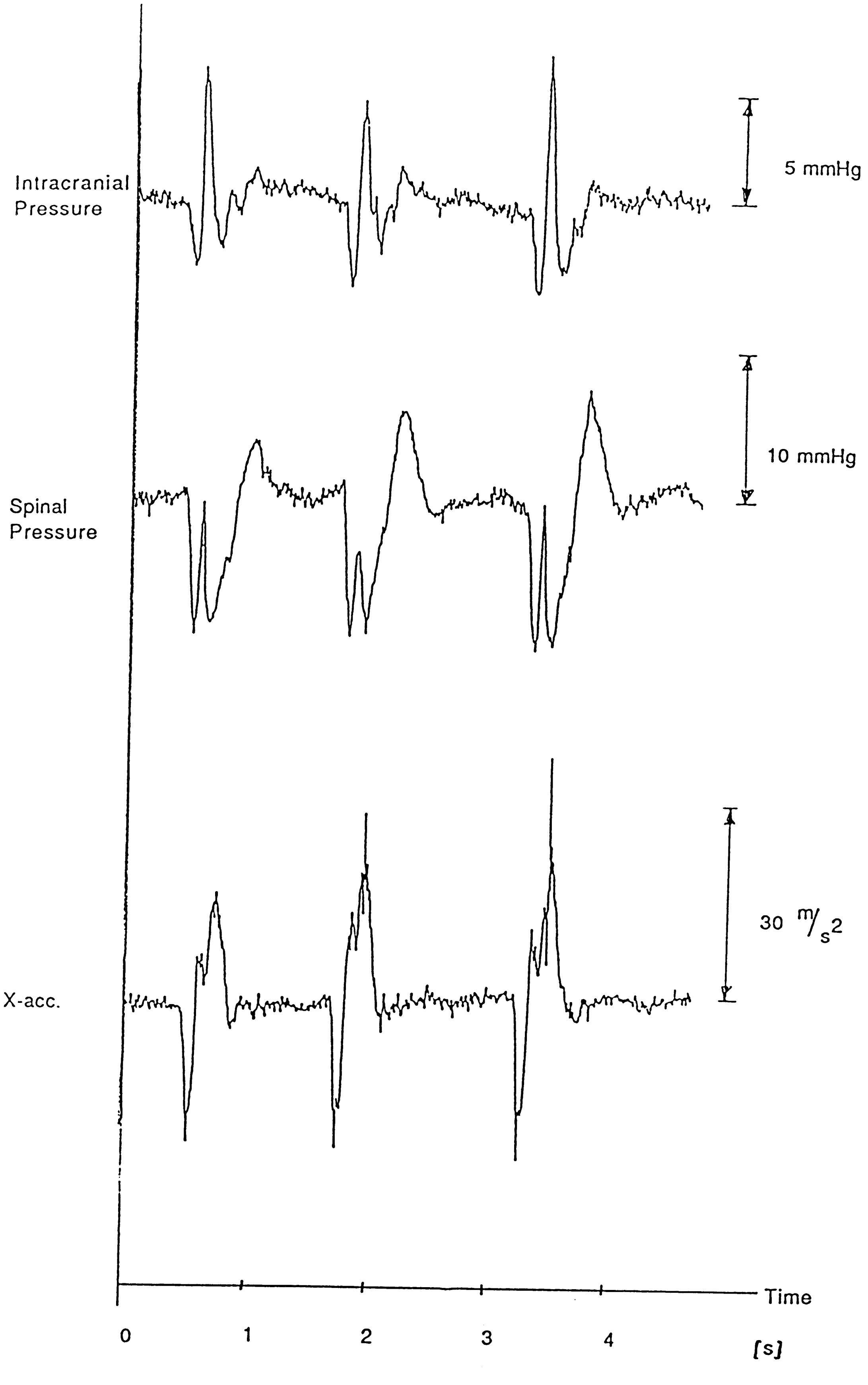


Figure 9 Three consecutive extensions immediately after the animal was sacrificed. Pulling is done manually.

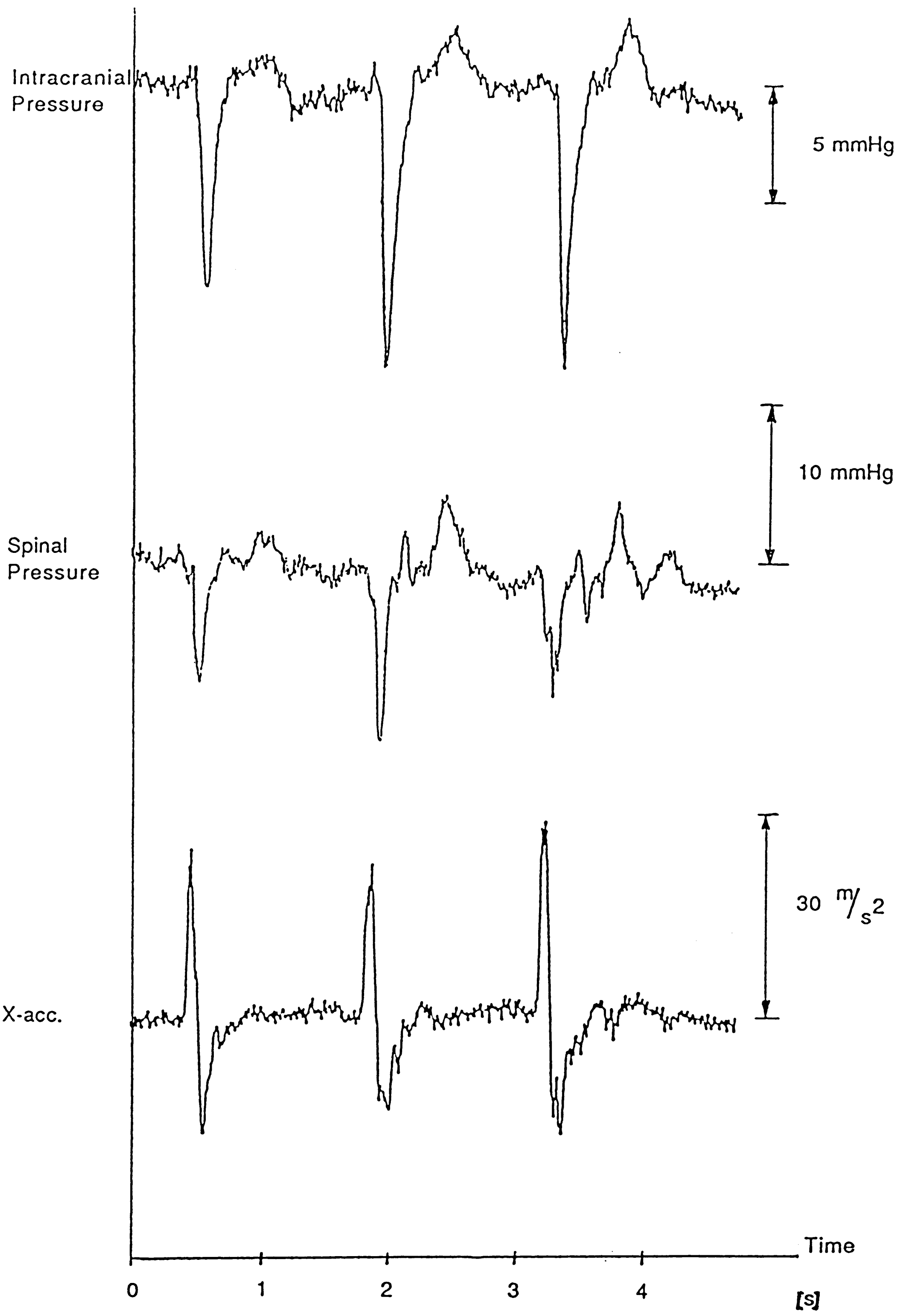


Figure 10 Three consecutive flexions immediately after the animal was sacrificed. Pulling is done manually.

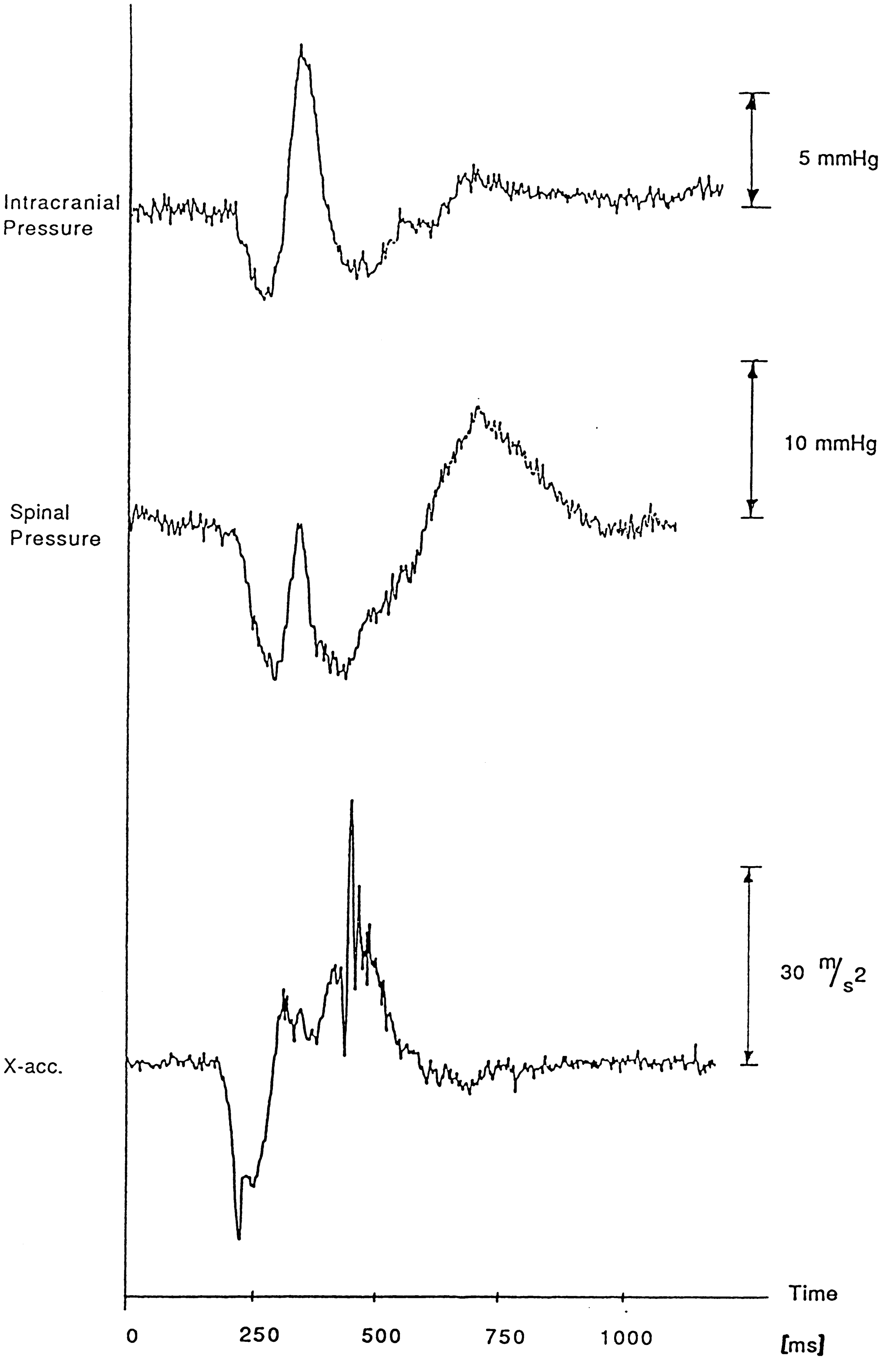


Figure 11 The first of the pulses in Figure 8 extended in time scale.

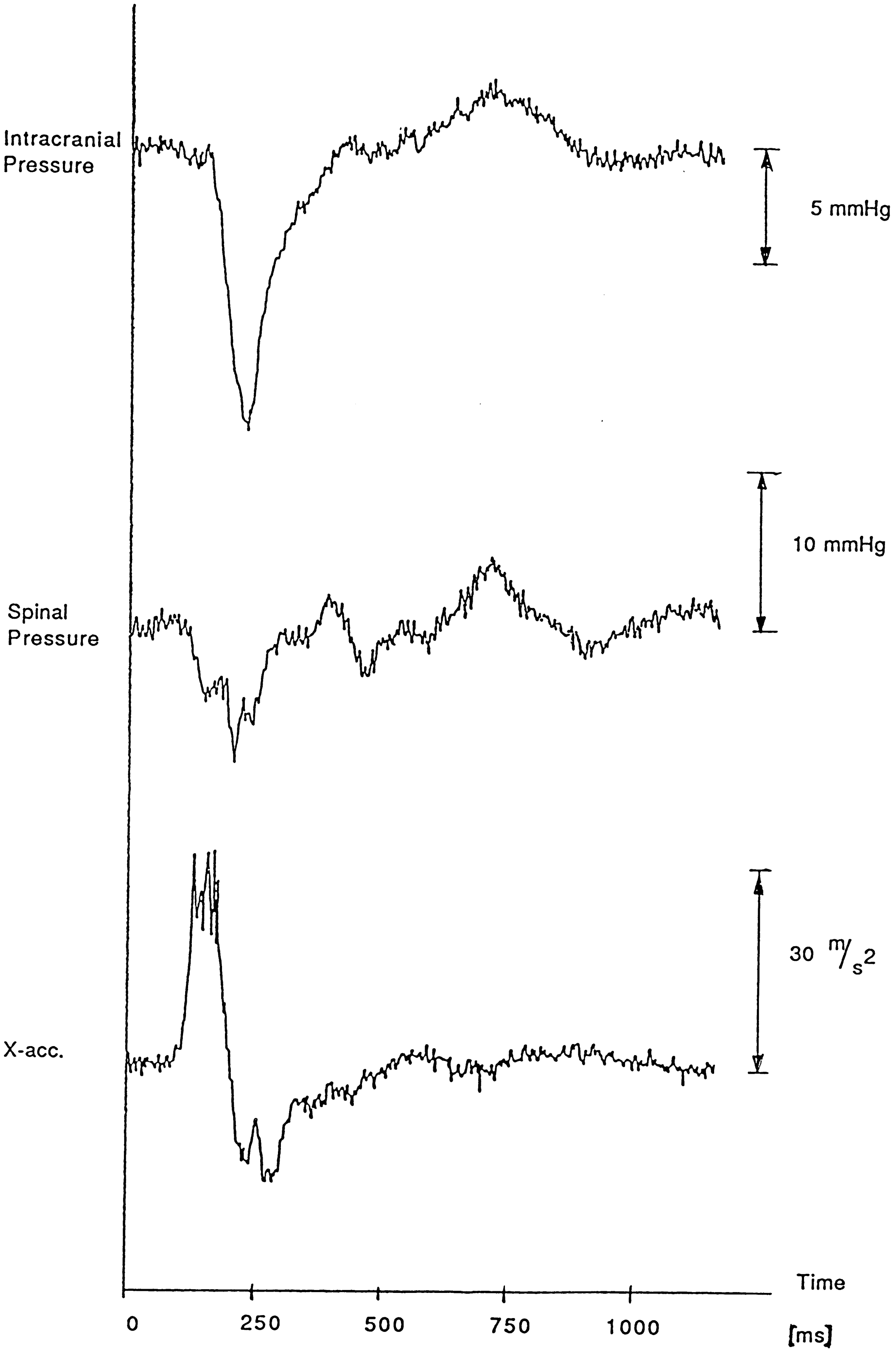


Figure 12 The first of the pulses in Figure 9 extended in time scale.

5 DISCUSSION

Initial tests have been made with the present version of a mechanical model of the spinal canal. They indicate that the acceleration of the cranial end (i.e pressure phenomenon 3) (section 2.4)) gives the greatest contribution to pressure buildup in the spinal canal. Since the tube does not contain any substitutes for the soft tissues this result might be misleading. The model will be improved when better knowledge about the qualities and dimensions of the tissues involved are available.

At the initial tests on anesthetized pigs no heart rate recordings were made, but we assume that the cyclic pressure pulses recorded with the animal at rest where due to the cyclic pulsations of the blood pressure. Heart rate recordings will be made in the next series of tests to check this.

The CSF pressure returns to normal between repeated extensions (fig. 8). One can also see a clear similarity in the pressure "pattern" between consecutive pulses. The same things could be seen in a similar test which was made before the animal was sacrificed.
From this, one can conclude that the mean pressure in the CNS is rather well stabilized under physiological motions of the neck.

The magnitude of the pressure pulses recorded are not likely to cause any damage to the nervous tissue since they are of the same order of magnitude as the normal cyclic pressure changes in the CSF. It has not yet been possible to measure pressure at C3 to C4 level, which is expected to be the center of pressure generation. The animals have not yet been exposed to levels of violence which exceed the levels used in tests with human volunteers.

The pressure transducer which was used in the spinal canal proved to be sensitive to bending. It gave a false pressure signal when the radius of bending of the optic fiber was small. Particularly if it was bent close to the the tip where the pressure sensing probe is situated. This artifact can easily be detected when the sensor is put into position by making slow flexion extension motions. If the artifact is present, the pressure signal will have an offset difference between full flexion and full extension when the head is held steady in these two positions.
To avoid artifacts due to this phenomenon we were confined to make our measurements in the upper thorachial region where the spine is stiffer and bends very little. In the next test series we will use another transducer which can be used in the middle of the cervical region of the spinal canal without these artifacts.
The complexity of the pressure curves is a major problem. The contour indicates that several pressure phenomena occur simultaneously. As suggested in section 2.4, we believe the phenomena to be pressure gradients due to flow resistance, flow acceleration, and the acceleration of the skull.

The analysis of the curves is even more difficult since the pulse shape and the direction of the acceleration varies between each test. The initial posture of the animals head and neck also varies between each test.
The relative motion between adjacent vertebra in the cervical spine can not be fully controlled. Thus the s-shape of the spine in the initial part of the whip-lash motion (fig. 4) develops in an arbitrary way. This will contribute to differences in results between two tests on the same animal even if they have identical acceleration pulses and initial postures.

In further tests the pulling device will be improved to give adequate repeatability of the acceleration pulse and repeatability of the path of travel for the head. The device will also give the possibility to increase the energy of the motion to the limit where ruptures and fractures of the spine occurs.
In order to attain knowledge of how different parts of the whip-lash motion contributes to the pressure pulse, a rig that can guide the head in a desired path of motion will be designed. The rig could for instance be made to constrain the head to a pure translational motion. One could then study what influence the development of the s-form of the cervical spine has on the pressure pulse. Another idea is to expose the head to a pure rotational moment and let the spine guide the translational motion. In this case the development of the s-form probably would not occur.

6 CONCLUSIONS

* The recorded pressure pulses give support to the theoretical model presented in chapter 2.

* Valuable experiences have been made from the methods and the experimental equipment used. With a modified equipment it will be possible to acquire more detailed information about the pressure phenomena and to distinguish the effects of single parameters.

* The pressure levels recorded are not likely to be injurious but with a modified equipment it will be possible to measure pressure in the cervical region of the spinal canal where the damage to the nervous tissue is expected to occur and where the pressure levels are expected to be higher according to chapter 2. It will also be possible to expose the head and neck to higher motional energy.

7 ACKNOWLEDGEMENTS

This project is sponsored by the Swedish Transport Research Board.(TFB 3/87-52) We would like to thank Assoc Prof Sten Holm and his staff at the Sahlgren Hospital in Göteborg for most valuable help in the lab.
We would also like to thank Research Engineer Lennart Thorngren at the Department of Injury Prevention for important assistance.

8 REFERENCES

Aldman,B. (1986): An Analytical Approach to the Impact Biomechanics of Head and Neck. 30th Annual Proceedings American Association for Automotive Medicine, pp. 439-454, LC 64-1965

Breig,A. (1978): Adverse Mechanical Tension in the Central Nervous System. Almqvist & Wiksell International, Stockholm,Sweden, ISBN 91-2200126-3

Crosby,E.C., T.Humphrey, E.W.Lauer (1962): Correlative Anatomy of the Nervous System. The Macmillan Company, New York, LC 62-7511

Estes,M.S., J.H. McElhaney (1971): Response of Brain Tissue of Compressive Loading. American Society for Mechanical Engineers Paper

Kapandji,I.A.(1974): The Physiology of the Joints, Volume Three. Churchhill Livingstone, Edinburgh, ISBN 0 443 01209 1

Löfgren,J. (1973): Pressure-Volume Relationships of the Cerebrospinal Fluid System. Diss., University of Gothenburg, ONR 99-0614854-3, Sweden

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

Maimaris,C., M.R.Barnes, M.J.Allen (1988): Whiplash injuries´ of the neck: a retrospective study. Injury 19, pp. 393-396

Martinez,J.L., D.J. Garcia (1968): A Model for Whiplash. J. Biomech., Vol. 1,pp. 23-32

Margaria,R.(1953): On the Functions of Cerebrospinal Fluid. Exp. Med. Surg. 11, pp. 297-301

McKenzie,J.A., J.F.Williams (1971): The Dynamic Behaviour of the Head and Cervical Spine during Whiplash. J. Biomech.,4:477

McMillan,B.S., J.R. Silver (1987): Extension Injuries to the Cervical Spine Resulting in Tetraplegia. Injury 18,224-233

Mertz,H.J., L.M. Patrick (1971): Strength and Response of the Human Neck. Proc. of Fifteenth 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. Almqvist & Wiksell,Stockholm,Sweden, ISSN 0365-5237

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

Olney,D.B., A.K.Marsden (1986): The Effect of Head Restraints and Seat Belts on the Incidence of Neck Injury in Car Accidents. Injury 17, pp. 365-367

Parke,W.W. (1982): Applied Anatomy of the Spine; in The Spine,vol.1. ed.Simeon and Rothman, W.B.Saunders Co., Philadelphia, ISBN 0-7216-7719-3

Severy,D.M., J.H. Mathewson and C.O. Bechtol (1955): Controlled Automobile Rear-End Collisions, an Investigation of Related Engineering and Medical Phenomena. Canadian Services Medical Journal, pp. 727-759

States,J.D. (1979): Soft Tissue Injuries of the Neck. in The Human Neck-Anatomy,Injury Mechanisms and Biomechanics,pp.37-43, Society of Automotive Engineers, SAE/SPSP-79/438,

Tenerz,L., B. Hök (1988): Guidewire-mounted sensor for biomedical pressure meassurements. Proceedings of the World Congress on Medical Physics and Biomedical Engineering, San Antonio,Texas, ISSN 0589-1019, LC 61-24788

White,A.A, M.M.Panjabi (1978): Clinical Biomechanics of the Spine. J.B.Lippincott Company, Philadelphia, USA, ISBN 0 397 50388 1, LC 78-15708