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Biomechanics of Cervical Spinal Cord Injury in Rollover Crashes

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Abstract

A series of physical models of the human head and neck were constructed to study the biomechanics of the cervical spinal cord during a simulated rollover crash. These models were dynamically loaded in axial compression and the spatial and temporal patterns of strain in the spinal cord were calculated. The largest strains were seen in the regions near subluxations from simulated ligament disruptions. The strain rates were calculated, and were sufficiently high in some regions that functional failure of the axons in the spinal cord would be expected in neural tissues undergoing these loads. The location and magnitude of strain and strain rate correlate with regions of cord damage in pathology data. The critical factors which predict the type and severity of spinal cord injury are the initial position of the head, the amount of vertical crush, and the rate of loading. In order to minimise these injuries in rollover accidents, the occupant imust be sufficiently restrained to prevent axial compression of the neck if the head contacts the roof, and that the roof crush is minimised so that the occupant space is not compromised

Keywords

spinal cord injury, biomechanics, neural injury, modelling

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Biomechanics of Cervical Spinal Cord Injury in Rollover Crashes

Report to the Federal Office of Road Safety

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CONTENTS

EXECUTIVE SUMMARY

A series of physical models of the human head and neck were constructed to study the biomechanics of the cervical spinal cord during a simulated rollover crash. These models incorporate anatomically similar surrogates for the skull, vertebrae, spinal cord and brain. The spinal cord and brain surrogates have embedded within them a grid of dots to allow visualisation of deformation patterns within the tissues during the experiment. The mechanical properties of each element of the physical models were matched to those of the human. The kinematics of the head and spine were validated carefully against the cadaver experiments of Pintar et al (1989) and the human volunteer experiments of Margulies et al (1992).

This model was dynamically loaded in axial compression while high speed film was taken. Individual frames from this film were acquired into a personal computer for analysis. The spatial and temporal patterns of strain in the spinal cord were calculated by digitising the grid points in the spinal cord on each frame and comparing their coordinates with the previous frames. The largest strains were seen in the regions near subluxations from simulated ligament disruptions. The strain rates were calculated, and were sufficiently high in some regions that functional failure of the axons in the spinal cord would be expected in neural tissues undergoing these loads. The location and magnitude of strain and strain rate correlate with regions of cord damage in pathology data from these types of injuries.

From these experiments, it may be concluded that the critical factors which predict the type and severity of spinal cord injury in axial compression loadings such as those seen in rollover crashes are the initial position of the head, the amount of total vertical crush, and the rate of loading. In order to minimise these injuries in rollover accidents, the primary concern is to ensure that the occupant is sufficiently restrained (eg by a seatbelt) so that the weight of the torso does not result in axial compression of the neck of more than 25mm if the head contacts the intruding roof structure, and that the amount of roof crush is minimised so that the occupant space is not compromised.

CHAPTER 1 - INTRODUCTION AND BACKGROUND

Although much work has been done in the biomechanics of spinal injuries, the majority of this work has been focused on the vertebral and ligamentous injury rather than the neural injury. Little has been done to investigate the effects of the injury to the vertebral column on the spinal cord itself. Recent work Bilston et al (1993) and Pintar et al (1993; 1995) has begun to address these problems by the incorporation of a spinal cord surrogate model in simulations of neck injury to measure strain and pressure changes, respectively. In the latter studies, a gelatine cord surrogate with embedded pressure sensors is placed in a cadaver neck during a simulation to measure dynamics pressure changes in the cervical canal. The largest pressures were associated with the more severe vertebral and soft tissue injuries. This study continues the work on flexion and extension injuries done by Bilston (1993).

Tolerance criteria for individual neck structural elements have been suggested by other investigators. Investigators from Duke University, Myers et al (1989), McElhaney et al (1988), and Doherty (1990) have characterised the fracture and deformation behaviour of isolated cervical vertebrae and the neck as a whole, under a variety of loading conditions. Kirby et al (1989), among others, have looked at mechanical properties and failure strength of the ligaments of the cervical and lumbar spine.

Studies using human volunteers and cadavers on accelerated sleds, have characterised the kinematics and dynamics of the head and neck during simulated frontal and lateral automotive impacts. Early work with human volunteers, by Ewing and Thomas (1972) has been added to by the group at the University of Heidelberg, using cadavers (Wismans et al 1987). More recent work at Chalmers University in Sweden has continued the human volunteer work (Davidsson 1996) in low velocity rear impact. These experiments give details on the linear and angular displacements and accelerations of the head and T1 vertebra, and, in Wismans et al study, gross pathology resulting from the loading.

In previous studies of traumatic brain injury, anatomically and mechanically accurate physical models have been used to quantify the spatial and temporal strain patterns experienced by a surrogate brain material under rotational and translational acceleration. These studies were instrumental in determining the intracranial deformation and the influence of acceleration and loading direction on axonal and vascular damage in diffuse axonal injury (DAI) and acute subdural haematoma (ASDH) (Margulies et al 1990; Meaney & Thibault 1990). They also provided an important link between clinical pathology and microscopic and cellular experimental studies, through the correlation of strain and strain rates with axonal and vascular damage. In the current study, these techniques have been extended to a model of spinal cord injury.

The need for a better understanding of the detailed mechanics of spinal cord injuries motivates this study. We investigate the mechanics of the spinal cord in the most common injury mode leading to neural damage from motor vehicle accidents, namely axial compression of the neck which occurs during vehicle rollovers.

CHAPTER 2 - AIMS

The primary aim of this study was to examine the relationship between the detailed mechanism of cervical spinal injury and the resulting spinal cord deformation patterns for injury mechanisms common to rollover crashes. The specific aims were:

1. To construct and validate a series of physical models of the head and neck which were capable of simulating the biomechanics of the cervical spine and spinal cord during an axial compression injury of the neck similar to those seen in rollover accidents.

2. To model both flexion-extension and flexion-compression injuries and to simulate clinically relevant injury mechanisms seen in vehicle accidents, such as vertebral subluxations and jumped facet joints.

3. To examine the relationship between detailed injury mechanisms to the cervical spine and the resulting loading placed on the spinal cord.

4. To predict neurological injury based on the spinal cord deformation patterns

5. To compare pathology data from similar real world injuries to these predictions.

6. To formulate injury tolerance criteria based on the results of these experiments.

CHAPTER 3 - METHODS

3.1 MODEL CONSTRUCTION

A series of inanimate physical models of the adult skull and cervical spine, which are anatomically similar to the human were constructed. Physical modelling techniques have previously been used to study the mechanisms of traumatic brain and spinal cord injury (Holbourn 1943; Margulies et al 1990; Meaney & Thibault 1990; Bilston 1994). The model construction has been described in more detail by Bilston et al (1993). In brief, these models incorporate a plastic reproduction skull and vertebrae, simulated spinal ligaments and musculature made of elastic, and a silicone gel brain and spinal cord whose mechanical properties are matched to those of the human brain and spinal cord respectively. Prior to assembly, the skull was sectioned approximately 3cm left of the midsagittal plane, and 0.5cm sections of the left lamina of each vertebra were removed to allow visualisation and measurement of the deformation of an embedded grid within the surrogate brain and spinal cord in the models during the simulated injury. The removal of these sections was done in such a way that the articulations of the intervertebral joints and the atlanto-occipital joints were not affected. Black circular markers were placed on the surface of the vertebral elements to allow measurement of the individual vertebral motions during each experiment. A schematic of the model is shown below (Figure 1).

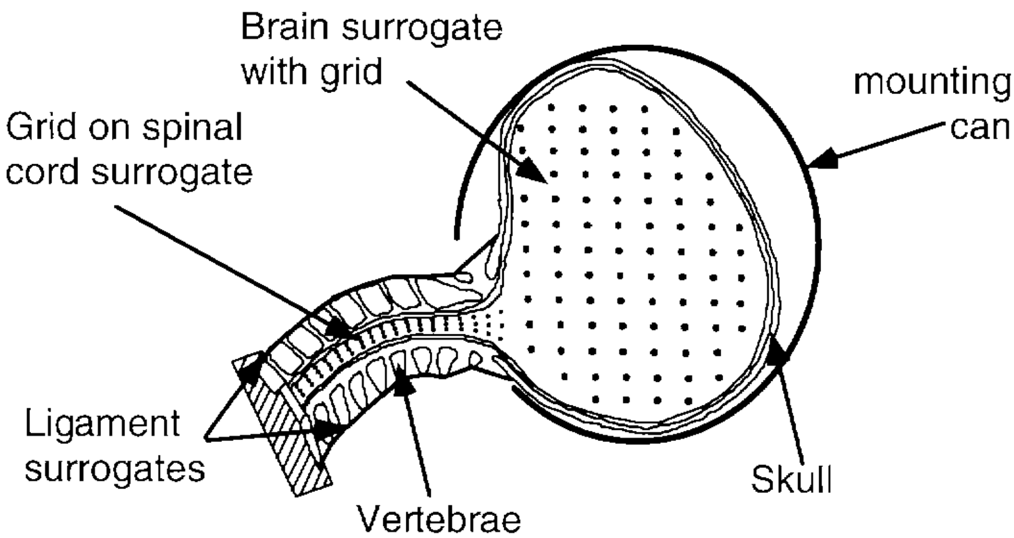


Figure 1. Schematic of the physical model showing the grid embedded in the spinal cord surrogate.

In order to evaluate the effects of a flexion-compression and extension-compression mode of buckling, one model was mounted so that the impact simulated an occipital contact, and the other a crown of the head impact.

3.2 MODEL VALIDATION

It was then necessary to validate the motion of each element of the model against human motions in order to ensure that the model was an accurate rendition of the human head and neck. Two types of validation were performed. The first compared the motions of the individual model vertebra and the axial motions of the spinal cord within the canal to those for normal human volunteers in flexion and extension motions as measured by Margulies et al (1992) and further analysed by Bilston (1994). The model was placed on a flat surface, and manually flexed and extended quasistatically. The motion was recorded using a video camera and video cassette recorder. Individual frames were digitised from the videotape using a frame grabber (DT55, Data Translation) in a personal computer. The position of markers on the vertebrae were located, their coordinates digitised on screen and saved to a file using an image analysis program (NIH Image, NIH, Bethesda, MD). The displacement and rotation of each vertebra was calculated relative to the T1 vertebra throughout the motion. The position of grid dots in the spinal cord adjacent to the vertebrae were also digitised, and the relative axial motion calculated.

The second validation process compared the vertebral motions from the model to those from a cineradiographic study using cadaver necks by Pintar et al (1989), and Yoganandan et al (1991). For this validation, a series of frames were grabbed from the film of the model motions during the injury simulation (see below) and stored as digital images on a personal computer. Similar to the quasistatic validation, the position of markers on the vertebrae were located, their coordinates digitised on screen and saved to a file. The displacement of each vertebra was calculated relative to the T1 vertebra throughout the motion.

3.3 INJURY SIMULATIONS

The models were mounted in an impact testing device (Shimadzu, Japan). They were dynamically compressed by 25-35mm at a rate of $1.5ms^{-1}$. Force and displacement data were recorded and stored on a digital oscilloscope (Thurlby DSA254, Thurlby Instruments, UK) at a sampling rate of 10kHz. This data was then downloaded to a personal computer for analysis. The deformations of the surrogate brain and spinal cord throughout the experiments were recorded by high speed photography (Stalex WS-3 camera & Kodak Ektachrome 7297 film) at 1000 frames per second. The films were then processed and transferred to high resolution (SVHS) videotape for further analysis. After the experiments were concluded, the testing device's load cell and LVDT (used to measure force and displacement) were calibrated using standard weights and vernier calipers respectively.

3.4 DATA ANALYSIS

Individual frames from the videotape were digitised using a frame grabber board in a personal computer, and saved as digital images. Custom image processing routines were used, within the MATLAB programming environment (The Mathworks, MA. USA), to digitise the coordinates of the gridpoints embedded in the model spinal cord from the images. The coordinates of the vertebral markers were also digitised from each frame. From these coordinates, the axial strain and average strain rate in the surrogate spinal cord were calculated. This method gives a digitising accuracy of approximately 0.5 pixels in regions of high contrast, which corresponds to a strain error of approximately ±5%. The strain and strain rate was compared with existing functional tolerance criteria for neural and vascular tissues to tensile loading to determine the likely location and degree of cervical cord injury. The force and displacement data were read into a spreadsheet, and plotted.

CHAPTER 4 - RESULTS

4.1 MODEL VALIDATION

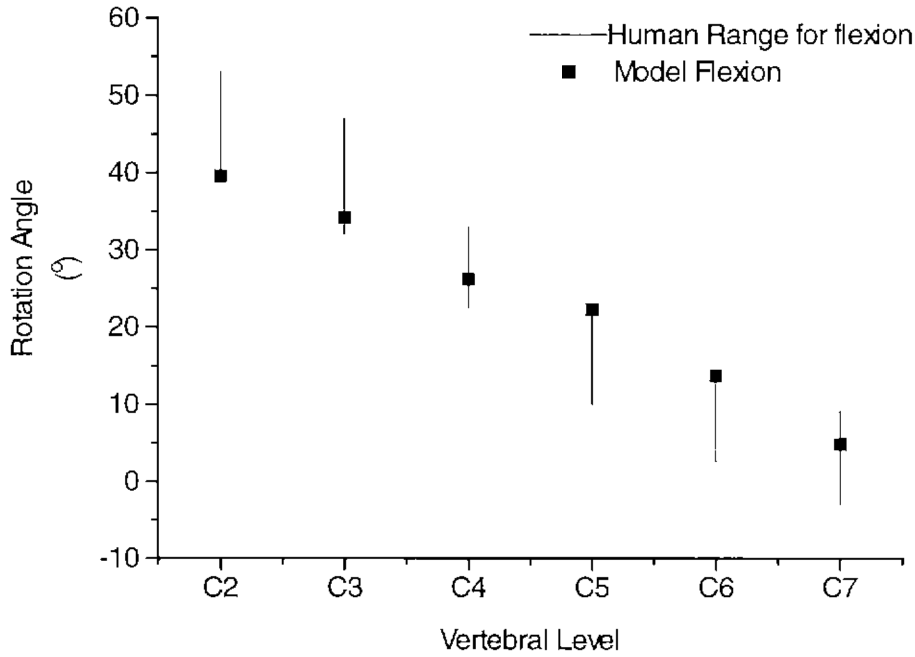
The rotations of the vertebrae relative to T1 during both flexion and extension motions were found to increase along the length of the cervical spine, as shown in Table 1 for flexion and extension motions. The rotations were very similar to the motions of normal humans, as measured by Bilston (1994), being within the reported range at all times, except that in the extension motion, the rotations for C5-C7 were slightly (1.6-4 degrees) above the human range. This was deemed to be unlikely to significantly affect the response of the model during axial compression, unless large rotations of the lower cervical spine were seen. The angles also increased with increasing head angle, as shown in Figure 2.

Axial motion of the model spinal cord within the canal was measured during the same flexion and extension experiments. In flexion both the patterns and magnitudes of displacement were very similar to human motions, with increasing displacement with head angle. In the physical model, the cord moved down relative to C3 6mm, near C4 the amount was 4mm, near C5 and C6 the cord moved about 3.5mm. In the Margulies et al study, these amounts were the same as the first subject's motions within 1mm, and very similar to the second subject's motions. In extension, the model cord at C3 level moved caudally approximately 5mm and the C5 region moved caudally approximately 2mm. The C7 region did not move. This is similar to the motions seen for the first subject in the Margulies et al study. (C3=3-8mm, C5=1.5-3.5 mm, C7= 0.5-2.5mm) although another subject had different patterns with each extension motion, some with larger caudal displacements at the lower vertebral levels. This is summarised in Table 2.

Table 1. Comparison Between the Vertebral Motions in Flexion and Extension for the Current Model and Adult Humans, from Bilston (Bilston 1994)

	Current Model		Human Volunteer	
Vertebral Level	Flexion (°)	Extension (°)	Flexion (°)	Extension (°)
C2	39.6	48.1	40-53	26-48
C3	34.2	30.9	32-47	20-33
C4	26.2	28.4	22.5-33	18-30
C5	22.3	21.6	10-23	12-20
C6	13.6	23.1	2.5-14	3-19
C7	4.8	11.4	-3-+9	-4-+7

(a)



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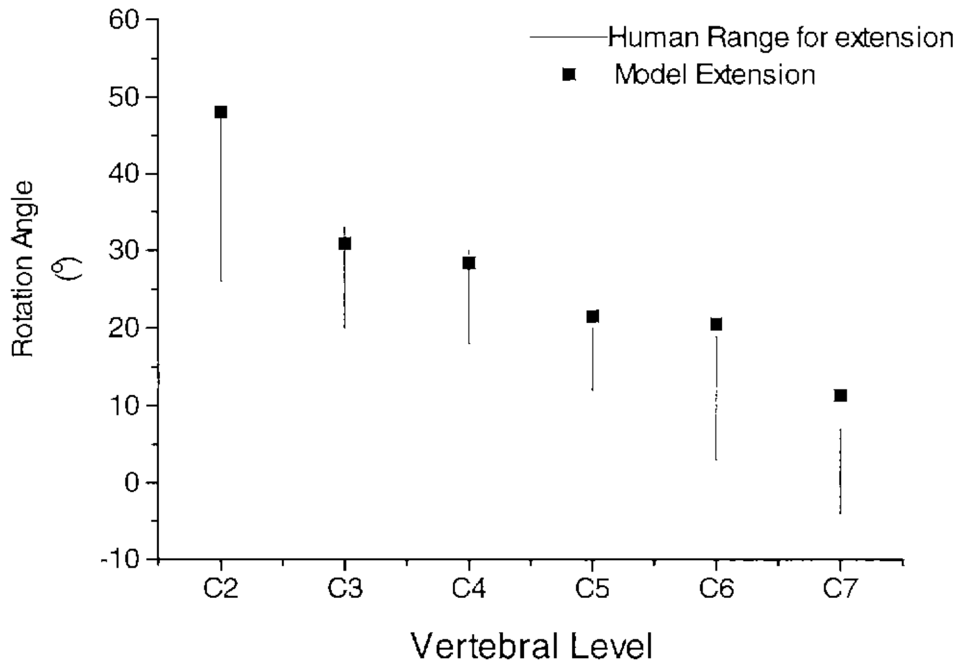


Figure 2. Variation of the vertebral angles with head angle during (a) flexion and (b) extension motions.

Table 2. Comparison of the Axial Motion of the Spinal Cord during Flexion and Extension for the Current Model and Adult Humans, from Margulies et al (1992)

	Current Model		Human Volunteer	
Vertebral Level	Flexion (mm)	Extension (mm)	Flexion (mm)	Extension (mm)
C2/C3	-5.5	-6	-6.3 - +4	-3.6 - -8.7
C3/C4	-5	-5	-5.9 - +2	-4.6 - +3
C4/C5	-3	-3	-4 - +3	-4 - +1
C5/C6	-2.5	-5	-3 - 3.6	-5 - +0.6
C6/C7	-3.5	0	-2.5 - +4	-5 - -1.3
C7/T1	-	-	1.6 -6.6	-3 - -2

Validation of the vertebral motions during axial compression was also performed. The displacement of each vertebra during the injury simulation was calculated and compared to those measured by Pintar et al (1989) in their cadaver neck study. The results are summarised in Table 3. Two specific neck injury modes seen in their study were simulated in the current study, namely their first specimen, which had a compression-flexion failure, with a C1/C2 subluxation, and their second specimen, which had a compression-extension failure, with a subluxation of C4 on C5. The pattern and magnitudes of the displacements were within 5 millimetres for these two modes of injury for these vertebral motions, and most were within 2mm. Thus, the vertebral motions of our model appear to be very similar to those experienced by cadaver necks during axial compression with similar injury patterns.

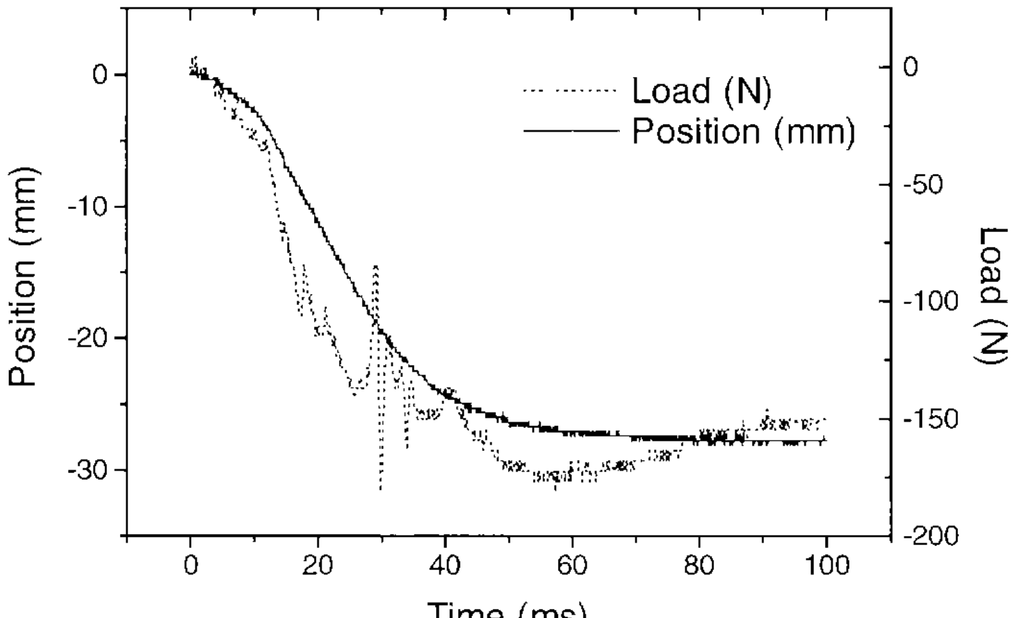
Table 3. Comparison of the Motion of the Model Cervical Spine in Axial Compression with Data from Yoganandan et al (1991) F-C is flexion - compression, and E-C is extension-compression

	Model		Cadaver	
Level	F-C (mm)	E-C (mm)	F-C (mm)	E-C (mm)
C2	11.4	10.4	9.4	12
C3	13.1	7.6	6.4	6.3
C4	3.7	17.6	2.9	12.3
C5	4.5	3.0	6.0	2.5
C6	2.9	2.0	1.4	2.5
C7	2.3	2.6	-	-

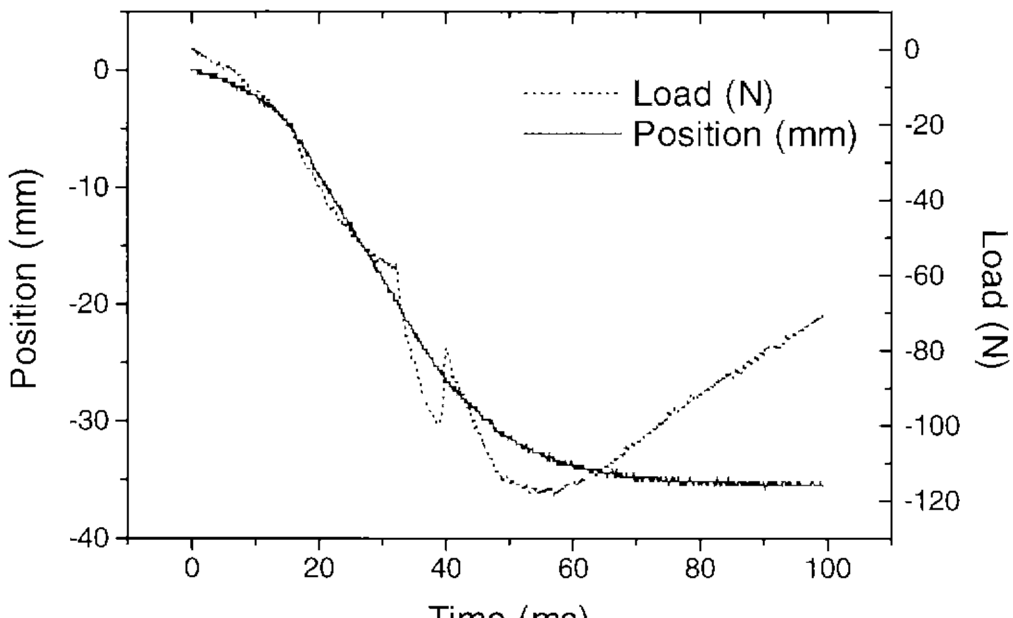
4.2 INJURY SIMULATIONS

4.2.1 Force and Displacement Data

The force and displacement data were recorded and plotted. This data showed that the 25-35mm compression occurred over a period of approximately 35-40ms. A sample set of data for a flexion-compression mode and an extension-compression mode are shown in Figure 3. In some experiments, a sudden decrease (or notch) in the measured force was seen part way through the run. This corresponded to the time when dislocation of C4 on C5 happened in the extension-compression mode simulation, or when the C2 dislocation occurred in the flexion-compression simulation. This was seen from the high speed film.



(a)



(b)

Figure 3. Force and displacement responses of the models in dynamic axial compression. (a) Extension-compression mode, (b) Flexion-compression mode. Force notches in both figures correspond to vertebral dislocations seen on the high speed film.

4.2.2 Spinal Cord Deformation

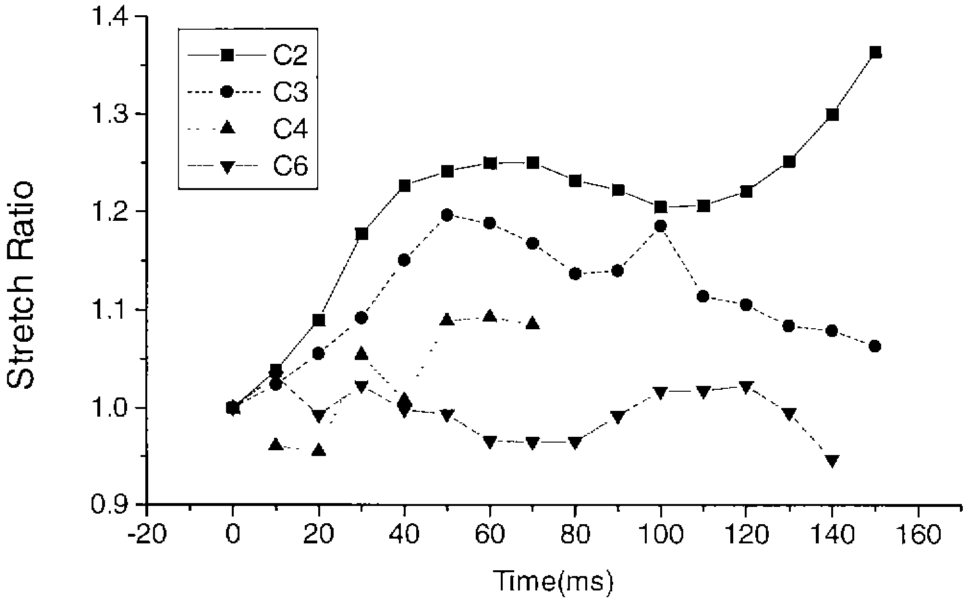
The model spinal cord showed significant amounts of deformation during the injury simulations. The axial strains and strain rates were calculated throughout the spinal cord during a simulated injury from the high speed film images. The largest strains (up to 40%) and strain rates (up to $6.5s^{-1}$) were, as expected, found in the regions immediately adjacent to vertebral dislocations, when these occurred. That is, in the flexion-compression simulations, the largest strains were adjacent to the C2 dislocation, and in the extension-compression, they are adjacent to the C5 dislocation. The regions of high strain extend for 1-2 vertebral levels above and below the level of the dislocation, however, as seen in Figure 4. The peak values of the strain and maximum tensile strain rates are summarised in Table 4. The data is from run 4 is missing, as the image quality was very poor (due to bad lighting), and the error in the strain data made the results unreliable.

Table 4. Peak Strain and Strain Rate in the Model Spinal Cord During Injury Simulations

	Mode	Run #	C2/3	C4/5	C6/7
Strain	Flexion	1	40%	23%	7%
Strain Rate	Flexion	1	$6.5s^{-1}$	$4s^{-1}$	$0.5s^{-1}$
Strain	Flexion	2	40%	5%	-4%
Strain Rate	Flexion	2	$5s^{-1}$	$4s^{-1}$	-
Strain	Flexion	3	35%	11%	1%
Strain Rate	Flexion	3	$2.9s^{-1}$	$6s^{-1}$	-
Strain	Extension	4	-[1]	-	-
Strain Rate	Extension	4	-	-	-
Strain	Extension	5	-5%	17%	2.5%
Strain Rate	Extension	5	$-1.6s^{-1}$	$6s^{-1}$	$5s^{-1}$
Strain	Extension	6	8%	35%	14%
Strain Rate	Extension	6	$2s^{-1}$	$13s^{-1}$	$4s^{-1}$

[1] Note: The image quality in Run 4 was poor due to insufficient lighting, and the strain data is unreliable.

(a)



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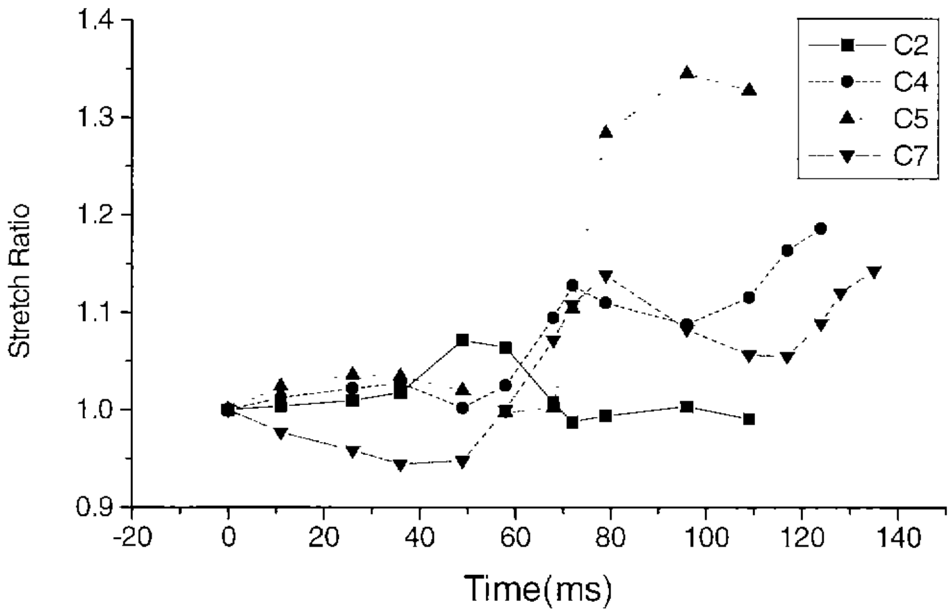

Figure 4. Stretch ratios in the model spinal cord during two injury simulations. (a) Flexion-compression mode, (b) Extension-compression mode.

CHAPTER 5 - DISCUSSION

5.1 GENERAL OBSERVATIONS

In this physical modelling study, several things were observed. First, the largest values of strain and strain rate occurred in the model spinal cord in the region surrounding the simulated dislocations. These strains and strain rates are not confined only to the region immediately next to the dislocation, but tend to be spread over one or two vertebral levels above and below the injury. The values of the strains and strain rates measured in this study can be examined in the light of available data on the mechanical tolerances of axons to dynamic stretch. Work by Galbraith et al (1993) on the giant squid axons showed that strains in excess of approximately 15-20% at strain rates greater than 1-5s^{-1} can cause reversible or irreversible injury to the axon, compromising its calcium ion homeostasis, and possibly leading to depolymerisation of its internal structures. From this information, we would expect that the regions immediately surrounding vertebral dislocations would experience some axonal loss from an injury similar to those simulated in this study. This correlates well with the gross cord pathology data reported by Kakulas' group (Davis et al 1971; Kakulas, et al 1973; Kakulas 1984), which indicates that cord damage is usually centred around a vertebral subluxation when it occurs, with damage spread across two or three vertebral levels around the bony lesion. This also suggests that spinal cord strain and strain rate is a reasonable method of predicting axonal damage in cervical spinal cord injuries.

The methods used in this study are, at best, an approximation of the complex structure and mechanics of the human cervical spine. The model reproduces the kinematics and dynamics of the cervical spine, as demonstrated by the validation studies, but does not reproduce the detail of all the fine ligamentous and meningeal structures. The effects of the ligamenta flava, nerve roots, and dentate ligaments on the cord deformations are not included in this model. It would be expected, however, that the ligamenta flava would tend to increase the local cord strains on contact, rather than ameliorating the deformation. The role of the dentate ligaments and exiting nerve roots, while not simulated by this model, are accounted for in the mechanical properties of the model spinal cord, and its kinematics within the spinal canal, respectively.

The use of physical models to study neural injury is not new, dating to Holbourn's work of the 1940's (Holbourn 1943). They allow the investigation of mechanisms of injury in a direct fashion, provided that the models are suitably validated, kinematically, anatomically, and mechanically. They also provide useful information about mechanical deformation patterns in the central nervous system, as caused by known applied forces and motions. These data can then be used in the construction and validation of finite element simulations, which may be used to further investigate the type of injury under study. The data from this study is currently being used to construct and validate a finite element model of axial compression in the cervical spine to further investigate these disabling injuries. In these models, the effects of such detailed structures as the ligamenta flava, dentate ligaments and nerve roots can also be studied.

Finally, the main aim of research such as this into the mechanics of spinal injury is to sufficiently understand the injury to be able to design preventative measures to avoid the occurrence of the types of neck loading which cause long term disability. Such devices might include stiffeners for the vehicle frame to limit intrusion into the passenger space during rollovers, along with better restraint design to prevent the occupant from sliding out of the restraint. It can be seen from this study, that axial neck compressions of as little as 25-35mm can cause severe spinal cord injury, if the forces are sufficiently large to rupture ligamentous structures, allowing subluxations and/or fractures to occur.

5.2 CONCLUSIONS

A physical model was constructed to estimate the deformations in the cervical spinal cord under axial compression loading. Two different injury modes were simulated, flexion-compression and extension-compression. Three simulations of each type of failure gave repeatable results from the models. In the flexion-compression models, a dislocation of the atlanto-axial joint was observed, with very large local strains and strain rates in the cord adjacent to that area. In two of the three extension-compression models, a dislocation of the C4-C5 joint was observed, again with local cord deformations.

A comparison of the measured strains and strain rates with isolated tissue data for axons under tensile loading (Galbraith et al. 1993), shows that in both cases, irreversible damage to some of the axons in the regions adjacent to the simulated dislocations might be expected in a live tissue experiencing these loadings. Moreover, the locations of large strain at high strain rates in the model cord correlate well with pathology from human spinal cord injuries, where the regions of worst spinal cord damage are centred around the area adjacent to a vertebral displacement, when this occurs (Davis et al. 1971; Kakulas 1987).

The results from this study suggest that protective devices or vehicle modifications designed to reduce serious neck injury in rollover crashes must include the following concepts as design requirements:

- The occupant space in the head region *must* be maintained to ensure that significant axial compressions (>25mm) of the cervical spine do not occur. That is, allowable vehicle crush must ensure that clearance for the head remains during rollover crashes.

- Adequate passenger restraint to ensure that the occupant is prevented from making head contact with the upper interior structure in such a way as to load the cervical spine with the body weight. Existing seatbelts may not adequately prevent this, although this needs to be investigated.

- If contact is unavoidable with the vehicle interior, the loading rate of the cervical spine must be kept below 0.25 m/s to reduce the likelihood of high strain rate loading of the cervical spine and spinal cord, and thus reduce injury.

Further Investigations:

- It is recommended that further investigation into the "real world" loading of the cervical spine be carried out in rollover crashes. This would enable more detailed tolerance criteria to be developed, linking vehicle and restraint characteristics to the serious neck injury risk. Currently, little real world information about occupant kinematics in rollover crashes is available, and even less information about the detailed loading of the neck during such a crash, although rollovers are one of the more common types of crash resulting in cervical spinal cord injury.

- The effectiveness of current and future seatbelt designs in limiting the occupant's vertical motion during rollover crashes needs to be investigated more thoroughly. Many serious neck injuries could be avoided if the occupant cannot "slip out" of the seatbelt during rollover crashes.

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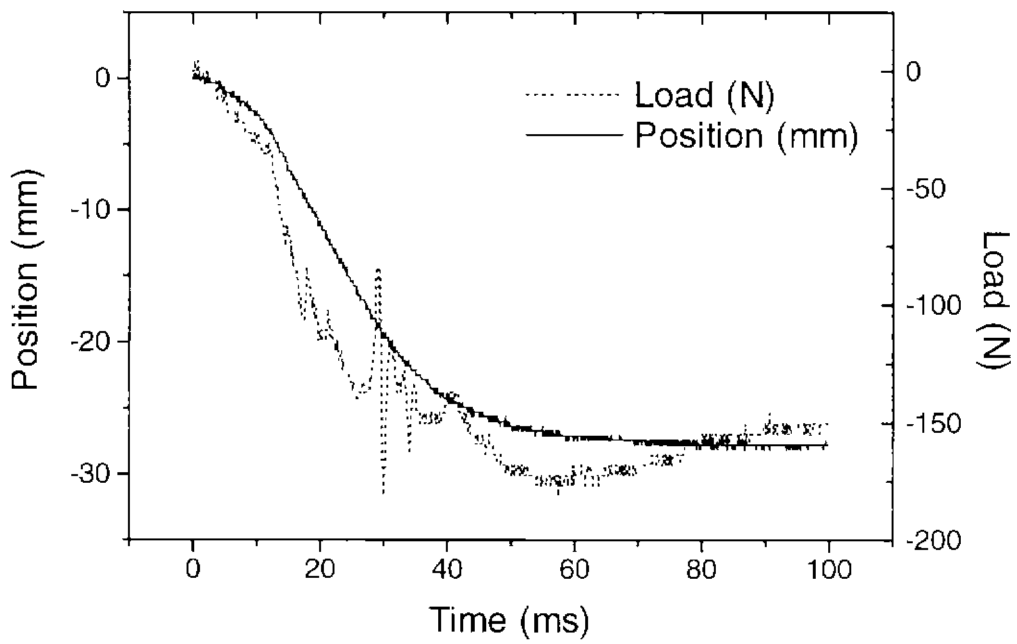
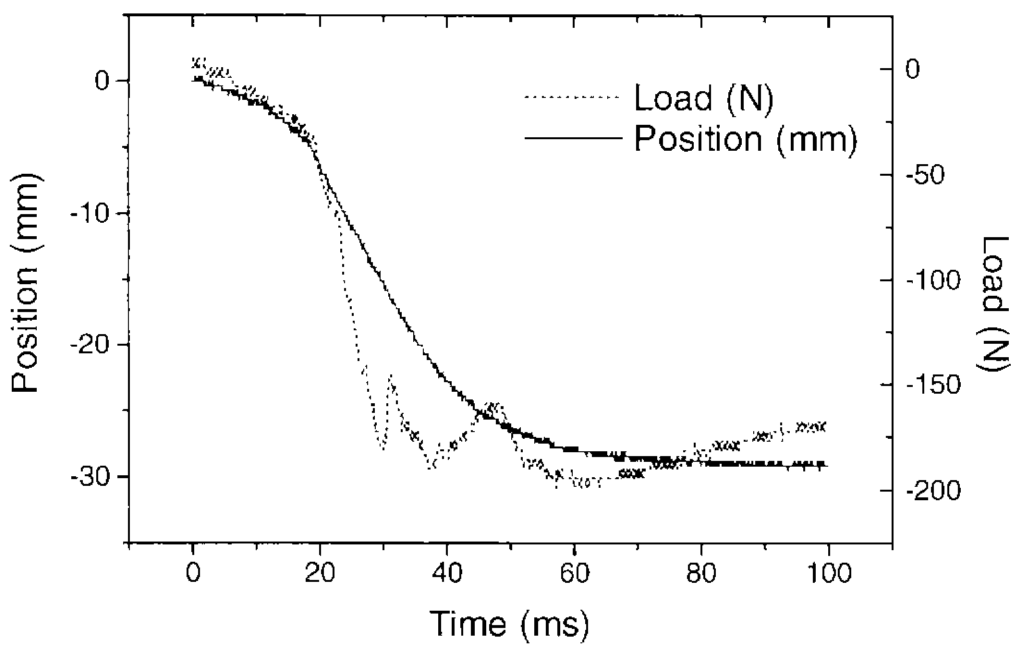
CHAPTER 7 - APPENDICES

APPENDIX 1 - FORCE AND DISPLACEMENT DATA

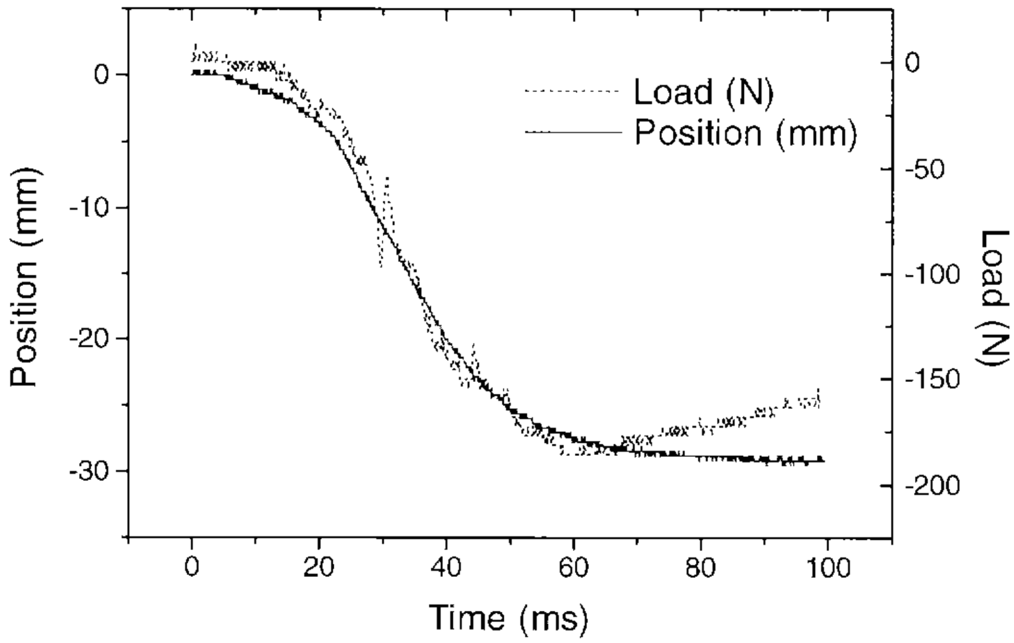
Run 1 (Flexion-Compression):



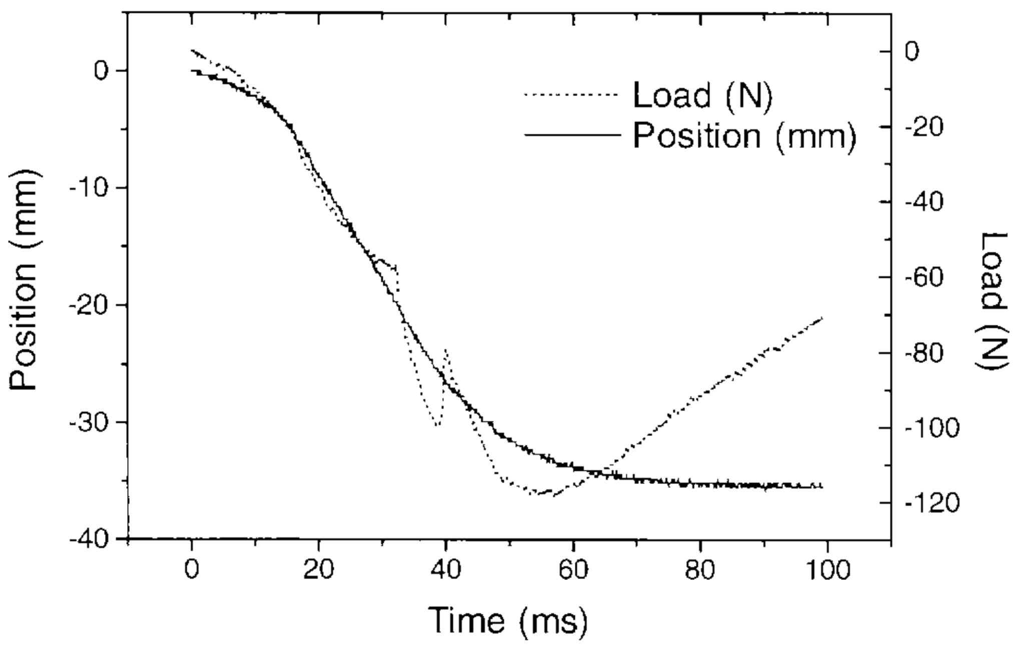
Run 2 (Flexion-Compression):



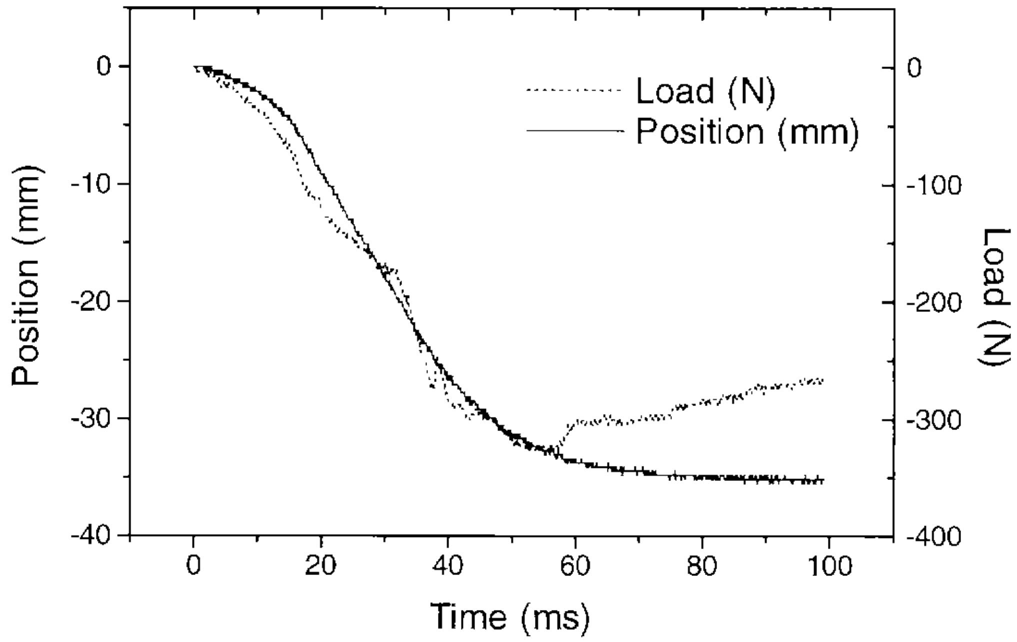
Run 3 (Flexion-Compression):



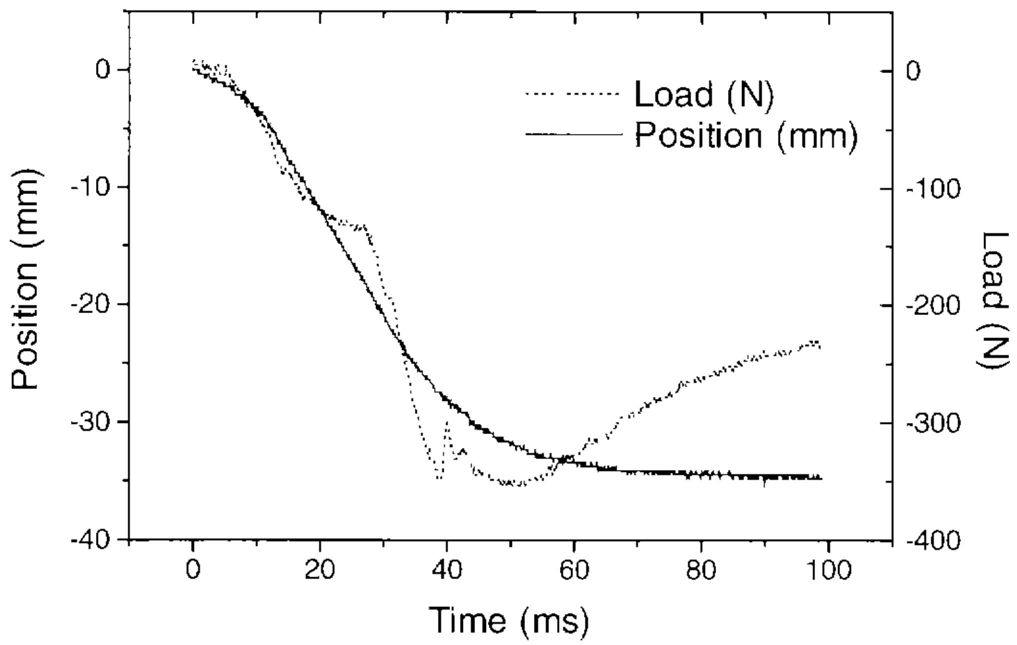
Run 4 (Extension-Compression):



Run 5 (Extension-compression):

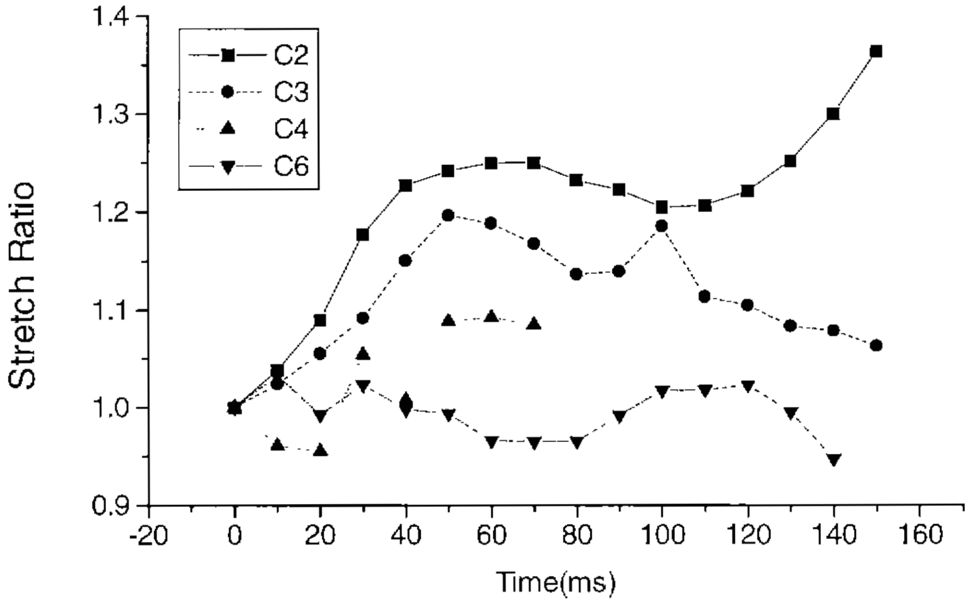


Run 6 (Extension-compression):

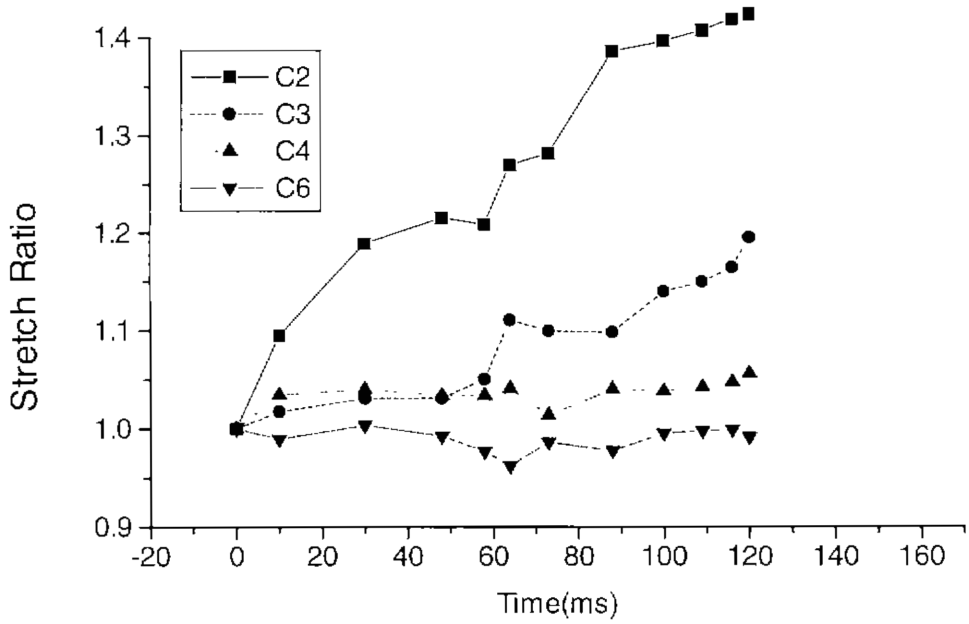


APPENDIX 2 - STRAIN AND STRAIN RATE DATA

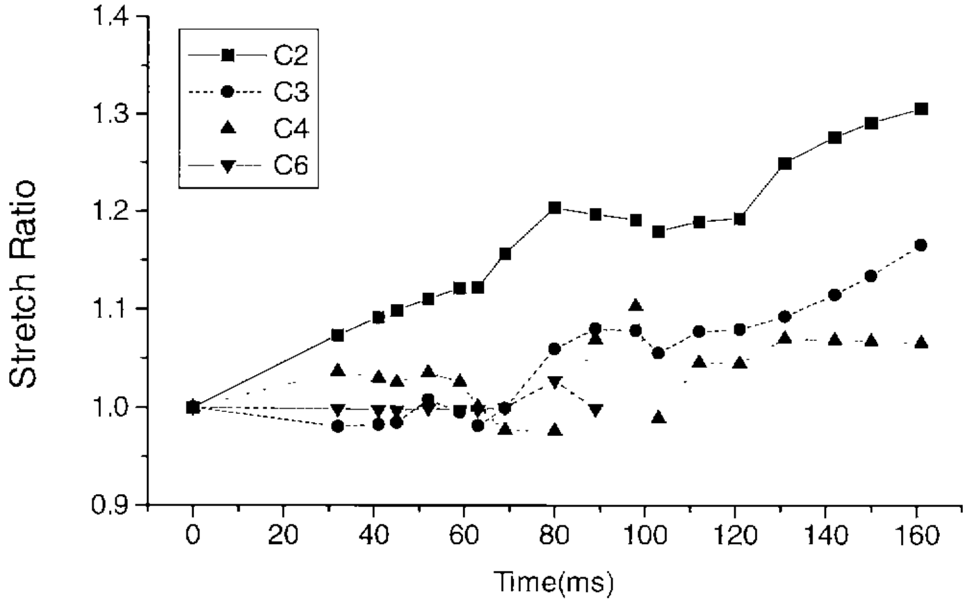
Run 1:



Run 2:



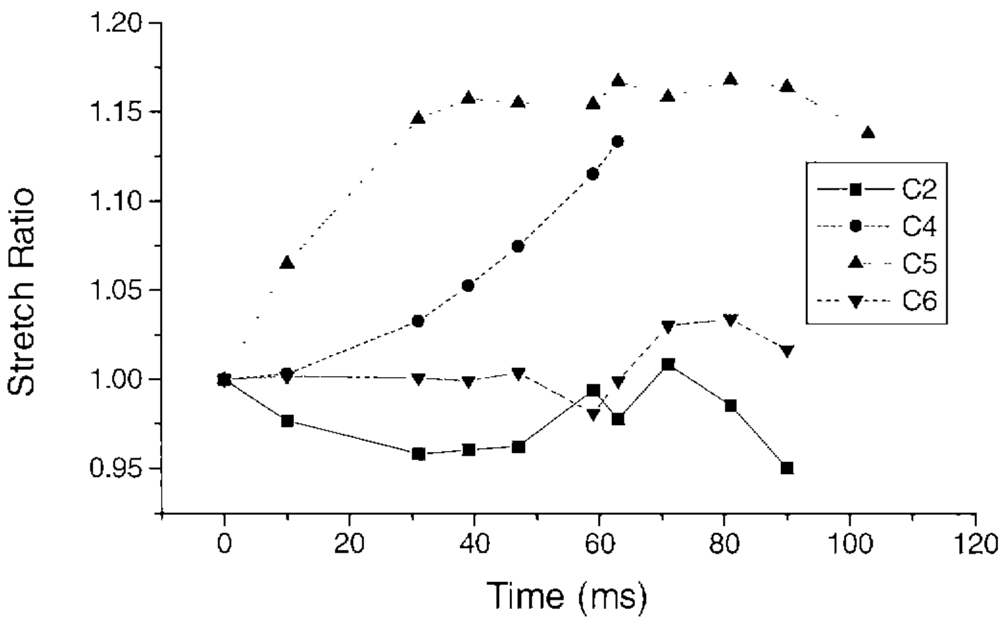
Run 3:



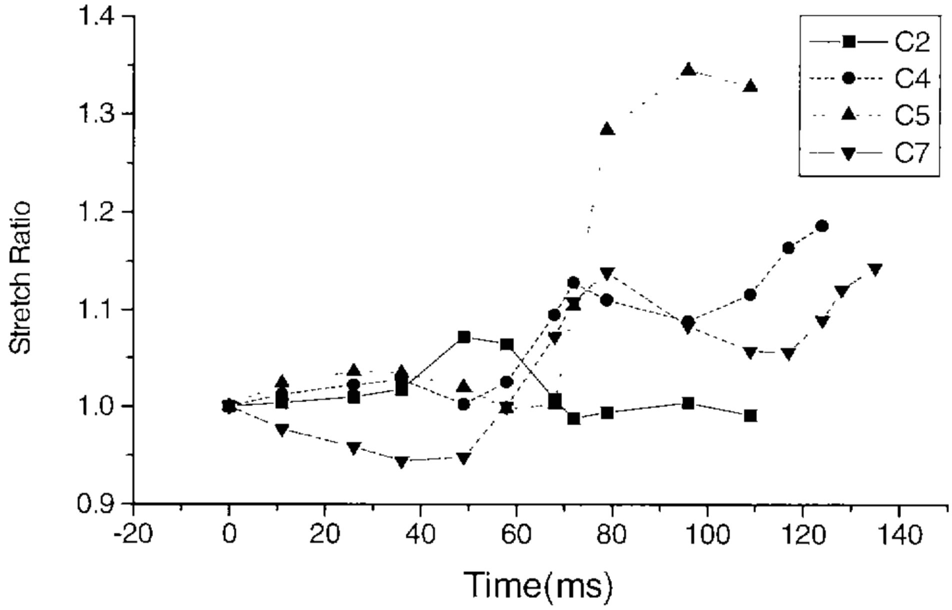
Run 4:

Image quality in run 4 was poor, due to mispositioning of the lights, and thus the data was unreliable.

Run 5:



Run 6:



1.4
1.3
1.2
1.1
1.0
0.9
Stretch Ratio
-20
0
20
40
60
80
100
120
140
Time(ms)
C2
C4
C5
C7

APPENDIX 3 - SAMPLE IMAGES

These images come from Run 1, a flexion-compression injury simulation.

Time t=-60ms

Initial position, before impact. Note the initial lordosis of the spine, and that the head is positioned slightly flexed. This results in a flexion of the upper cervical spine during the simulation.

Time t=72msec

Note that the C2 vertebra is beginning to displace laterally, due to the local flexion in the C1-C3 region. Large deformations of the cord region adjacent are occurring.

Time t=119msec

The dislocation is more severe now. Note also that the lower cervical spine is locally in extension, while the upper region is flexing.

Time t=169msec

The simulation has finished now. This shows the final position of the model.