FINITE ELEMENT MODELLING OF BLUNT OR NON-CONTACT HEAD INJURIES

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ABSTRACT

At present the injury that the human head is subjected to is predicted by the Head Injury Criterion (HIC). This criterion is inadequate as it is not based upon a thorough understanding of the underlying head injury mechanisms.

The important blunt or non-contact head injury mechanisms are diffuse axonal injury, bridging vein disruption and surface contact contusions. They are the result of the relative motion of the brain and skull.

Tissue failure criteria are developed for these injury mechanisms so that head injury tolerance curves can be developed.

Validated finite element models are used to determine the biomechanics of head injury and develop head injury tolerance curves.

The severity of head injury is related to the magnitude, direction and pulse duration of both translational and rotational head acceleration.

This paper represents a summary of research conducted as part of the authors Phd.

INTRODUCTION

Head injuries not only represent a serious trauma for those involved but are also responsible for a significant societal burden. These have been the driving forces behind continued research. This attempts to develop a better understanding of the biomechanics of head injury and develop criteria which can be used to develop improved safety devices and systems.

At present the injury that the human head is subjected to is predicted by the Head Injury Criterion (HIC). This is a single function which takes into account resultant translational head accelerations (a) and pulse duration (t_2-t_1), see equation 1.

It is a quantitative index used in the prediction of the severity of head injury in automotive

$$HIC = \left[\frac{1}{(t_2 - t_1)} \int_{t_1}^{t_2} a\,dt \right]^{2.5} (t_2 - t_1) \text{ ----- (1)}$$

and other instances where human injury is important in design considerations. A value of 1000 is considered to represent an acceptable level of risk for head injury. It is estimated that a HIC of 1000 represents an 8.5% risk of death from head injury. This equation developed from earlier attempts to use mathematical functions to approximate the Wayne State Tolerance Curve (WSTC) [32]. The WSTC is a head injury tolerance curve which is based upon the assumption that linear skull fracture is linked to brain damage. Cadaver experiments were used to evaluate the acceleration tolerance for pulse durations up to 6ms. Other information from cadaver and animal experiments and human volunteer studies were used to evaluate the tolerance data for longer pulse durations.

The assumption that skull fracture and brain injury are directly linked has been challenged for a number of years. Recent pathological studies have found that brain damage is not necessarily linked to skull fracture [30]. The criterion neglects the effect of rotational acceleration on the severity of brain injury although rotational acceleration of the head has been found to cause brain injuries. Studies have also demonstrated that HIC deviates from the WSTC at pulse durations above 15ms.

The Head Injury Criterion has been effective in reducing the risk of head injuries by reducing the resulting levels of translational accelerations experienced by the head. To reduce the risk even further there is a need for ever more sophisticated safety systems and products. This requires a better understanding of the biomechanics of head injury and the use of improved head injury criteria. Validated head injury finite element models are valuable tools in investigating the injuries that result from linear and rotational head accelerations.

To predict the severity of head injuries it is essential to understand the underlying head injury mechanisms. This research project will examine blunt or non-contact closed head injuries. These injuries are the least understood and are becoming increasingly problematic in modern vehicular accidents with improved safety systems. The injury mechanisms examined are diffuse axonal injury, bridging vein disruption and surface contact contusions.

The economic cost of motor vehicle crashes is approximately $150 billion per year in the US and European Communities. The direct and indirect cost of head injuries in the US is estimated at $25 billion per year [24]. The 40000 fatalities and 5.2 million non fatally injured patients that occur due to vehicle crashes each year is a significant contributor to this figure.

Injuries to the brain and spinal cord are the major causes of serious disability and fatality. Traumatic brain injuries account for 68% of all trauma-related fatalities, 34% of all injuries and 50% of all injuries in vehicular crashes. Diffuse axonal

injury is associated with mechanical disruption of axons in the central nervous system. Diffuse axonal injury is responsible for more than one-third of all head injury deaths, and is the greatest cause of severely disabled and vegetative survivors. 55% mortality, 3% vegetative survival and 9% severe deficit is the experience of patients 1 month after injury. Bridging vein disruption is responsible for some of the intracranial haemorrhages that produce haematomas. Subdural haematoma is one of these and has a 30% incidence with 60% mortality rate in the severely injured. Surface contact contusions are caused by the brain impacting the inner surface of the skull. They have an incidence of 89% in brains examined at post-mortem [5]. A computerised tomography (CT) scan study showed that they have a 31% rate of incidence in head injuries admitted to hospital [32]. The clinical outcome associated with contusions is difficult to assess although the affected region dictates the function that will be impaired. Mortality rates are thought to range from 25-60% [5].

The societal cost of injuries, the relative importance of head injuries, the limitations of the Head Injury Criterion and the increased demand for safer motor vehicles explains the extensive attention that head injury research has received from medical and biomechanical researchers over recent years. The relatively high incidence and mortality of diffuse axonal injury, bridging vein disruption and surface contact contusions in head injuries justifies the scope of this research project. Biomechanics of human injury is a difficult activity as it relies on a series of hypotheses which are sometimes difficult to categorically prove or disprove.

HEAD INJURY MECHANISMS

Three major factors influence the intracranial injuries sustained when the head is subjected to a rapid change in motion. They are related to the relative motion of the brain and the skull [6] and have been demonstrated through experimentation [10]. The degree of relative motion is limited by various anatomical entities. These are the meningeal partitions, bridging veins, subarachnoid (pacchionian) granulations and the brain skull interface which contains a interstitial fluid layer [6].

The meningeal partitions include the falx cerebri and tentorium cerebelli, see Figure 1. The falx cerebri is situated between the cerebral hemispheres and the tentorium cerebelli is situated between the cerebral hemispheres and the cerebellum. The falx cerebri mostly restricts cerebral hemisphere motion when the head is accelerated in the coronal plane. The tentorium cerebelli mostly restricts cerebral hemisphere motion when the head is accelerated in the sagittal plane.

The bridging veins drain blood from the brain into the dural sinus, see Figure 2. The majority of the bridging veins are located on the saggital midline. The arachnoid granulations drain cerebrospinal fluid from the subarachnoid space into the dural sinus, also shown in Figure 2. The subarachnoid granulations are located on the cerebral vertex.

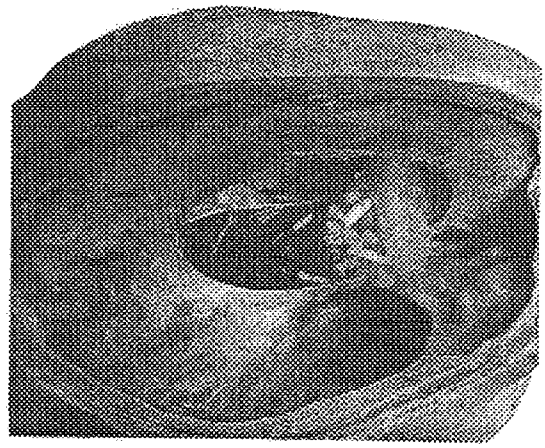

Figure 1 : Meningeal Partitions - Cranial Cavity with the Brain Removed.

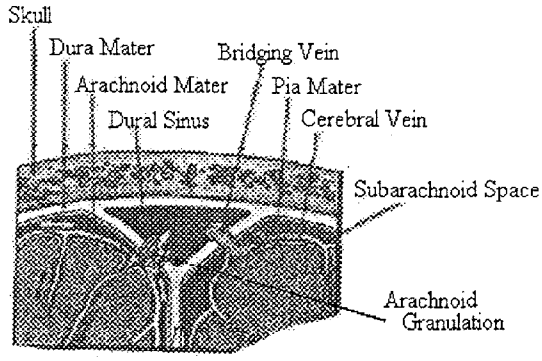

Figure 2 : Drainage of Blood and Cerebrospinal Fluid into Dural Sinus.

The cerebrospinal fluid system involves the production of cerebrospinal fluid in the ventricles which flow into the subarachnoid space. This is then drained into the dural sinus via the subarachnoid granulations once a certain pressure is reached. This maintains a certain pressure as well as continuously renewing the fluid. The fluid within the ventricles acts as a strain release. The fluid contained in the subarachnoid space of the brain/skull interface, which can decouple, damps the brain's response [6].

The first major factor that influences intracranial injuries is the distribution of shear waves throughout the central nervous system; the second is the disruption of bridging veins at the brain skull interface, and the third is the contact of the brain surface with the irregular skull.

Brain tissue has a relatively low shear modulus and therefore has a low resistance to shear waves and high strains result. Axon tracts, as well as other elements of the neurone, see Figure 3, are sensitive to strain. When the central nervous system is subjected to strain, diffuse axonal injury occurs. This is the widespread microscopic damage of the axons due to the resulting brain tissue shear strain.

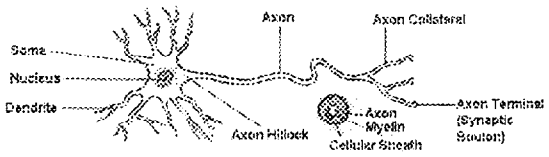

Figure 3 : Typical Neurone.

Observations of diffuse axonal injury were pioneered by Strich during the 1960's [19]. They were found to be prevalent in long duration, gradual onset accelerations as found in many modern vehicle accidents. The strain is severe at changes in geometry and material properties [19]; this includes the white/grey matter and brain tissue/ventricle interfaces, brain periphery, areas around the dural partitions, cerebral hemispheres below the slender stalk of the midbrain [19], the spinal cord, and its interaction with the foramen magnum. The levels of strain are also heavily dependent upon the applied axis of rotation. Coronal rotation produces the most severe strain levels, mainly due to the influence of the falx.

Axon damage is a combination of structural alteration and dysfunction. Much of the pioneering work was carried out by Thibault over the last few decades [9]. Total structural failure occurs at strains above 25% and dysfunction by calcium absorption at strains above 20%. It has been discovered that up to 5% of the applied elongation can be used in taking up the slack of the axons' natural orientation.

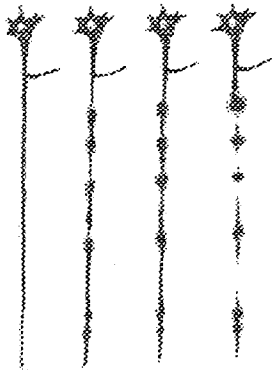

Figure 4 : Axon Damage Sequel.

The mechanism of axon damage is not fully understood. The current thoughts are that when the axons are subjected to shearing and tearing, they form retraction balls produced by the extrusion of axoplasm, forming large retraction swellings, see Figure 4. The common understanding of retraction ball formation is due to the work of Ramon and Cajal in 1928 who investigated sections of damaged central nervous system elements [22]. The continuum of axon damage is time dependent and the exact mechanisms are not that important unless they can be reversed or prevented by treatment.

Another possible consequence of the brain tissue strain is that the intracerebral blood vessels of the brain's blood supply system could be damaged. This would lead to intracerebral haemorrhages.

The relative motion of the brain in the cranial vault causes the sagittal bridging veins that pass blood from the brain into the venous system to be subjected to strain [9]. The positions of the sagittal bridging veins have been investigated, see Figure 5, and their damage is a sign of serious closed head injury.

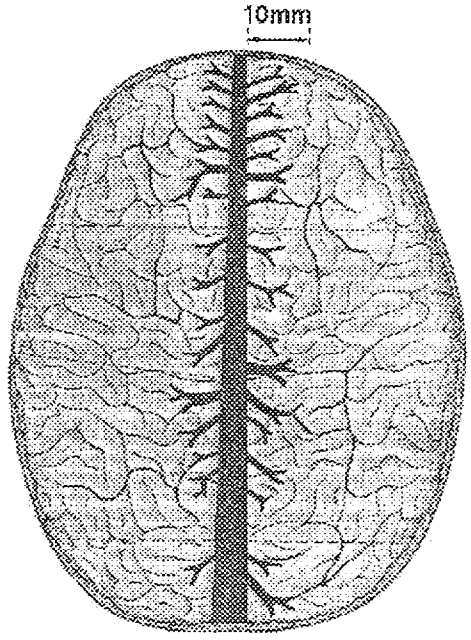

Figure 5 : The Location of Bridging Veins.

Sagittal plane rotations are the most injurious to the sagittal bridging veins. This is due to the majority of the bridging veins being situated in the parasagittal region. Dorsal bridging veins are the most easily injured, according to experiments conducted by Lindenberg [15]. This is due to the lack of brain tethering and the orientation of the bridging veins in this region.

Bridging vein strain results in a reduction in diameter and so hypoxic and ischaemic problems arise; strains above 15% result in significant effects. Their breaking strength varies from 1.5 - 14 Mpa and they can withstand an ultimate strain in excess of 100% [9]. Experiments conducted by Lee [14] showed

that bridging veins have a characteristic load-stretch curve, see Figure 6. It suggests that the bridging veins start to neck at a stretch ratio of 1.5 and total structural failure occurs at a stretch ratio of 2 [14].

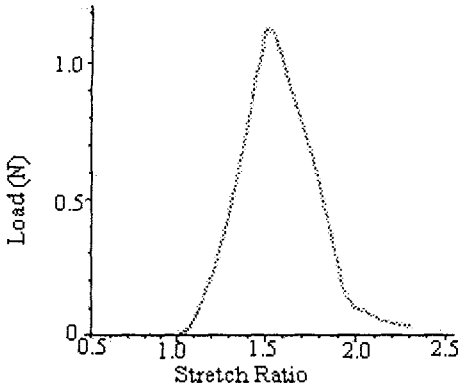

Figure 6 : Load v Stretch Characteristic of Bridging Veins.

The failure of the bridging veins results in a variety of haemorrhages including subdural and subarachnoid. It is important to realise that the clinical signs may take some time to manifest themselves. This is particularly the case when the venous system is concerned, where the blood pressure is relatively low and haematomas, for example, take time to form.

The relative motion of the brain within the cranial vault causes the brain to impact with irregularities of the inner surface [11]. These cause contusions at the impact site due to damage of blood vessels in the superficial brain layers or direct deformation of brain tissue [3]. Strains of between 20 and 40% cause the brain tissue to retain the effects of indentation [3].

The contusions found at sites remote from the impact site are most likely the result of the same injury mechanism as the contusions found at the impact site. The relative movement of the brain causes contact with skull irregularities and dural partitions, and is most likely due to the complex head motions that occur in real accidents [22]. This is in agreement with a novel theory that complex modal responses cause contusions of the brain on the irregularities of the skull [34]. The fact that contrecoup injuries occur in similar frontotemporal regions regardless of impact site strengthens this theory.

TISSUE FAILURE CRITERIA

It is important to have a way of linking the parameters determined in the head injury finite element models to the severity of injury. Tissue failure criteria have been developed for this purpose. The parameter used to predict the severity of injury is dependent upon the injury mechanism.

The severity of diffuse axonal injury is linked to the level of strain to which the axons are subjected [17]. Animal studies, isolated tissue tests, physical models and numerical techniques [17, 35], have been used to determine the most suitable measure of the strain tolerance of axons. The research conducted in these studies determined that shear strain is the best indicator of the severity of diffuse axonal injury. Shear strains under 5% represent no injury, 5 - 10% represent mild injury, 10 - 15% represent moderate injury and strains above 15% represent severe injury.

The brainstem and corpus callosum regions are common areas of diffuse axonal injury [7]. If diffuse axonal injury occurs in these regions, the clinical outcome is usually serious. The shear strain in the corpus callosum and brainstem regions of the head injury finite element models will be used to predict the severity of diffuse axonal injury. This is directly linked to the subsequent clinical outcome. This information is also used to develop the head injury tolerance curves for diffuse axonal injury.

Bridging vein disruption can cause a continuum of injuries ranging from hypoxic and ischaemic problems, starting at relatively low elongations through severe necking, to structural failure leading to severe haemorrhaging. An elongation of 50% produces necking and an elongation of 100% produces structural failure [14]. The mean length of bridging veins is 6mm and therefore 50% and 100% elongations are equivalent to 3mm and 6mm respectively [14]. The mean length of the bridging veins is used to develop the tissue failure criteria.

Surface contact contusions result from the brain impacting skull irregularities and dural partitions. The clinical outcome of this type of injury is dependent upon the area that is involved.

A compressive strain of above 20% will result in a contusion [3]. The site of the contusions is used in the validation process, where predicted injury sites are compared to the pathological findings. Tolerance curves are not developed for surface contact contusions as they have a limited link with the degree of clinical outcome and it would be difficult to grade the predicted contusion injury.

MODEL DEVELOPMENT

The directional dependence of the injury mechanisms justifies the use of two dimensional models. The mechanisms of injury are dependent upon the applied acceleration direction. Sagittal and coronal models are developed as these directions of acceleration are critical in producing diffuse axonal injury and bridging vein disruption. A horizontal model would only be useful in predicting superficial

contusions. It would require increased anatomical detail and would be of little benefit in terms of determining the biomechanics of head injury. A fine mesh is required when the models are used to analyse severe head trauma. Coarse meshes would be unable to handle the high brain and membrane deformations. The finer brain mesh will enable a better representation of the ventricular cerebrospinal fluid which has been found to affect the developed strain field. It will also better represent the various contacts and subsequent deformation patterns of the brain and membranes. These affect the motion of the brain with respect to the skull and the developed strain fields. The relatively stiff behaviour of the free edges of the partitioning membrane structure enables the use of stabilising springs in the two dimensional analysis models to be used to achieve the same behaviour.

The skull is assumed rigid and the bridging veins are assumed to have a negligible effect on the response of the brain. The ventricular cerebrospinal fluid is considered an elastic compressible fluid. This is related to the fact that the fluid system allows flow between ventricles, which causes the fluid to act in a compressible manner. The partitioning membranes affect the motion of the brain during trauma and therefore elastic representations of the falx and tentorium are included. They are restrained using stabilising springs, which ensure that they deform in the same way as the three dimensional structure. The brain is modelled as a viscoelastic incompressible material, see equation 2.

$$G(t) = Gx + (Go - Gx)e^{-\beta t} \quad \text{---------- (2)}$$

The brain mesh does not include the sulci, as they would be difficult to deal with and they do not have a significant effect on the brain's global movements. The general shape of the brain is maintained and the major longitudinal fissure, lateral sulcus and cerebellum are included as these significantly affect brain motion and developed strain field of the brain. The coronal slice passes through the corpus callosum region of the brain, which is a major indicator of the severity of diffuse axonal injury. It also includes the temporal lobes which have a significant effect on the brain's response. The sagittal slice passes through the midline plane and includes the brainstem, spinal cord, cerebellum and bridging veins. The brainstem is a major indicator of the severity of diffuse axonal injury and the bridging vein response is a major indicator of bridging vein disruption and associated haemorrhages. The spinal cord is included, as its response affects the strain field in the brainstem. The cerebellum is included, as it has a significant effect on the brain's response.

The brain mesh includes a representation of the white and grey matters. This is done because they have different material properties and this will affect the developed strain field. The white and grey matter material property parameters are developed by modifying existing data [25] to include established material differences [29]. The brain/skull interface is made up of membrane layers with an interstitial fluid layer, which is assumed to decouple under certain levels of applied acceleration, see Figure 7.

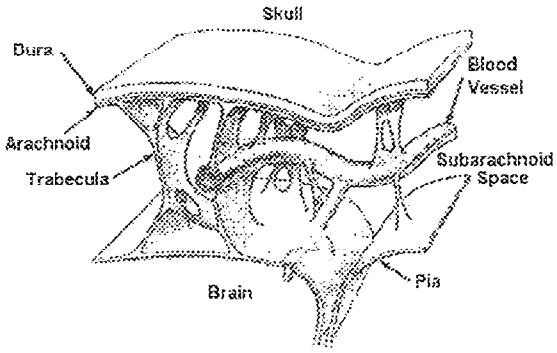

Figure 7 : Meningeal Layers.

The dura is attached to the skull and the pia is attached to the brain. The arachnoid lies between these two membranes and is held to the pia by a variety of trabecula. The gap which is formed is termed the subarachnoid space and is filled with cerebrospinal fluid. This fluid protects the brain during trauma by acting as a structural damper. The arachnoid and dura are free to slide past each other. The models have to take these interfaces into account as they have a significant effect on the behaviour of the brain during trauma. The arachnoid/dura interface can decouple if sufficient mechanical input is applied to the head during trauma. The models assume that the brain/skull interface decouples under any level of loading. Both coupled and decoupled interfaces cannot be included in the same model and as the injuries are caused due to decoupling, the interface is modelled in that way. This does mean that the models will predict more severe injuries in minor head trauma. It will also produce unrealistic field parameter values in some regions of the brain where the coupling effect is significant. However, this will have a negligible effect on the critical regions of the brain such as the corpus callosum and brainstem.

In the models, the damping produced by the subarachnoid cerebrospinal fluid is included by having a representation of the fluid layer using LS-Dyna3D elastic fluid elements, which have zero shear modulus, between the brain and skull elements, see figure 8.

The brain contacts the fluid elements (contact 1) before contacting the skull elements (contact 2), therefore reducing the relative velocity and motion of the brain with respect to the skull resulting in less severe injuries. The contact force is related to the penetration that is calculated between

time-steps. The fluid elements are attached to the skull to stabilise their behaviour and prevent numerical instabilities.

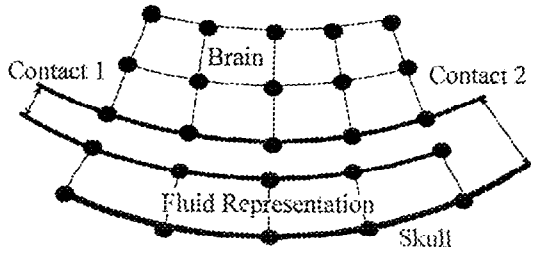

Figure 8 : Brain/Skull Interface

As the brain moves relative to the skull, membrane contacts on membrane as the arachnoid slides on the dura. The contact between the brain and the skull in the models is considered to be that of arachnoid and dura. The elements that represent the subarachnoid cerebrospinal fluid are stabilised using spring elements to prevent instabilities occurring during the simulations. These have a negligible effect on the response of the brain and are valuable tools in ensuring that the simulations reach the termination time successfully.

The cerebrospinal fluid circulates around the central nervous system. The cerebrospinal fluid is produced in the ventricles of the brain and passes through other ventricles into the subarachnoid space. Cerebrospinal fluid is produced all the time and as the pressure builds, older cerebrospinal fluid is removed. It is passed into the venous sinuses via subarachnoid granulations which act as one way pressure relief valves. These are located at the top of the parasagittal region. Through post-mortem examinations [28], it was found that these granulations restrict the relative motion of the brain and the skull. This is inagreement with forensic neuropathological findings [6]. A spring representation of these is included in the models. The stiffness is evaluated by considering them to be dura matter. The models have these granulations attaching the brain to the skull. In reality they attach the arachnoid matter to the dura matter of the superior sagittal sinus. During the analysis higher local strains can occur in this region due to this simplification.

The subarachnoid granulations and the brain/skull interface are the important details that limit motion of the brain with respect to the skull.

The falx and tentorium membranes that are included in the models are restrained using stabilising springs. These are used to ensure that the two dimensional representations behave in the same way as the three dimensional structure when they are loaded. The springs are attached to the membranes at one end and to an artificial structure at the other. The artificial structure is used to anchor the stabilising springs, so that only movements of the membranes cause changes in length of the spring elements.

Dentate ligaments restrain the spinal cord from moving along its length [27]. This restraint limits the relative motion of the brain and also significantly affects the strain field that develops in the brainstem. The sagittal model includes a rigid spring approximation of the dentate ligaments at the position of the first dentate ligament. The springs are fixed to the elements which represent the spinal cord at one end and to an artificial structure at the other. This artificial structure is used to represent the denticular ligament's rigid anchorage points.

The pia matter that covers the brain restricts the motion of the temporal lobes with respect to the cerebral hemispheres. A spring element representation of this pia matter is applied between the cerebral hemispheres and the temporal lobes. This ensures that the coronal model accurately predicts the brain's response.

The two dimensional coronal and sagittal models are shown in Figures 9&10. The figures clearly show the various entities modelled. The material property parameters are obtained and adapted from published literature, see Table 1.

The head complex input either comes through the neck or by direct impact, depending upon the type of trauma. The skull then transfers this input into the intracranial contents. Brain motion is caused by the movement of the skull and membranes. The rigid body skull is given prescribed velocity pulses about the centre of mass of the head to achieve both the desired acceleration and realistic skull kinematics.

The field parameters in particular elements which are critical to the prediction of the severity of the injury mechanisms are chosen for analysis. These are used in the validation cases and also to determine the biomechanics of head injury and develop improved head injury tolerance curves. The maximum shear strain of the central elements of the corpus callosum and brainstem regions are used to predict the severity of diffuse axonal injury. The elongation of the spring elements is used to indicate the severity of bridging vein disruption in various regions of the brain/skull interface. The maximum principal strain of the surface elements of the brain are monitored to determine the severity of surface contact contusions.

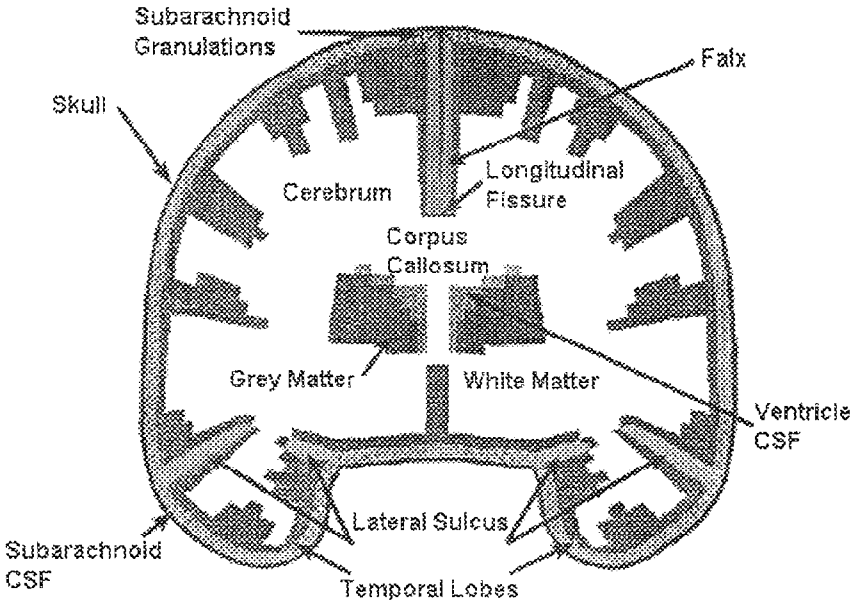

Figure 9 : Two Dimensional Coronal Model.

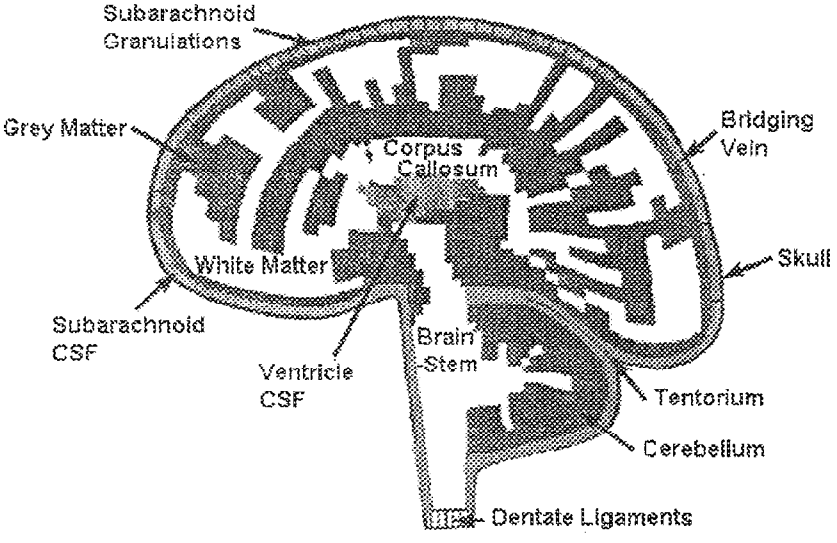

Figure 10 : Two Dimensional Sagittal Model.

Table 1 : Material Property Parameters.

Reference	Structure	Density (kg/m^3)	E (N/m^2)	Poisson Ratio	Go (N/m^2)	Gx (N/m^2)	B (/s)	K (N/m^2)
25	Dura	1133	31.5E06	0.45				
23	Brain							
&	White Matter	1040			5.48E05	1.74E05	35	2.19E09
29	Gray Matter	1040			5.08E05	1.62E05	35	2.19E09
23	CSF							
&	Ventricular	1000						1.11E07
14	Subarachnoid	1000						4.45E05
12	Skull	1410	4.46E09	0.21				
35	Pia Matter	1133	11.5E06	0.45				

VALIDATION

Validation of the head injury finite element models is critical if they are to be used as engineering and medical tools. The validation process involves the use of pathological observations, cadaver experiments, accident reconstructions and volunteer data. Each of these sources of validation data has its limitations. This paper includes the qualitative post mortem validation. The complete validation process can be found in the authors Phd [13]. Cadaver experiments demonstrate brain/skull interface decoupling. Accident reconstructions show that the models can predict real world injury data and volunteer studies show that the models can predict no or minor injury.

There have been many studies regarding the observations made at post-mortems. These studies discuss the patterns of injuries that occur in real life head trauma. This information is used to qualitatively validate the injuries predicted by the head injury finite element models.

The saggital and coronal head injury finite element models demonstrate the degree of relative motion of the brain and the skull that occurs, see Figures 11 and 12. The coronal model also shows the motion of the temporal lobes that is induced, see Figure 12. This motion would produce the superficial contusions seen at post-mortem in the frontotemporal region. These occur regardless of the impact site and are due to the skull irregularities that exist in this region [6,7].

The relative motion of the brain would not produce contusions in the occipital lobes where the skull is smooth. This is in agreement with post-mortem observations where the occipital lobes are rarely contused in frontal impacts [6]. The degree of relative motion would also explain the occurrence of contusions at the impact site [6] where the brain impacts the inner surface of the skull. The motion of the temporal lobes causes high strains to be induced in the temporal lobe at the impact site as well as the other temporal lobe. This is in agreement with post-mortem observations that indicate contusions in both temporal lobes after lateral impacts [6]. The relative motion of the brain produces high strains in the corpus callosum, brainstem and parasaggital regions of the brain as well as regions adjacent to the ventricles. These regions are areas of diffuse axonal injury and lesions that are found at post-mortem [7]. The relative motion of the brain causes the bridging veins to be elongated, see Figure 11. This explains the subarachnoid haemorrhages that are often present on one or both sides of the brain [7]. The subarachnoid haemorrhage can be caused by distant bridging vein disruptions and can be confused for coup and contrecoup lesions at post-mortem [7]. This can lead to confusion when determining the mechanism of injury particularly with respect to coup and contrecoup.

The relative motion of the brain and the skull, the complex motion of the temporal lobes and the pattern of strain induced in the brain predicted by the models, explain the injuries found at post-mortem. They explain the classic triad of

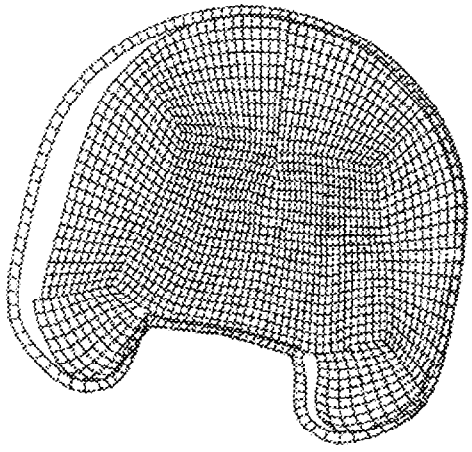

Figure 11 : Coronal Model.

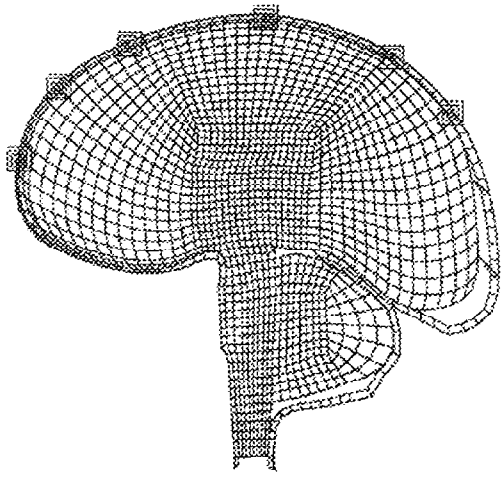

Figure 12 : Sagittal Model.

diffuse axonal injury involving focal lesions in the corpus callosum and brainstem and microscopical evidence of diffuse damage to axons [1]. They also explains the contusion patterns that are seen in the frontotemporal and parasagittal regions of the brain plus the disruption of bridging veins.

BIOMECHANICS AND HEAD INJURY TOLERANCE CURVES

The validated head injury finite element models are used to determine the biomechanics of head injury and develop head injury tolerance curves. Simulations are carried out to determine whether the biomechanics of head injury due to applied accelerations are as hypothesised. Head injury tolerance curves have been developed for diffuse axonal injury and bridging vein disruption [13]. They are developed using the tissue failure criteria discussed earlier.

The head injury finite element models show that the motion of the brain with respect to the skull is responsible for the head injury mechanisms as hypothesised. The three head injury mechanisms are produced by different consequences of the motion of the brain relative to the skull.

In the coronal model, the relative motion of the two cerebral hemispheres induces relatively high shear strains in the corpus callosum. In the sagittal model the motion of the cerebral hemispheres is resisted by the brainstem, causing relatively high shear strains to be induced. The level of shear strain is dependent upon the severity and direction of the applied acceleration and the applied pulse duration. This dependency is illustrated in the head injury tolerance curves [13]. The shear strain pattern then spreads throughout the brain tissue as the events progress.

The models provide viable hypotheses of the mechanisms behind the classic triad of diffuse axonal injury. The relatively high shear strains are responsible for the focal lesions found in the corpus callosum and brainstem regions. The lower level shear strains which spread throughout the brain are responsible for the microscopical evidence found in the axons of the white matter.

The sagittal model shows that when the head is accelerated, the brain moves relative to the skull. This relative motion elongates the spring elements which represent the bridging veins passing between the brain and dural sinus, which is attached to the skull. The level of elongation is dependent upon the severity and direction of the applied acceleration and the applied pulse duration. This dependency is illustrated in the head injury tolerance curves.

The sagittal model provides a viable hypothesis of the mechanism of bridging vein disruption found at post-mortem.

The relative motion of the brain predicted by the coronal model produces relatively high compressive strains in the temporal region of the brain. The relative motion predicted by the sagittal model would result in high compressive strains in the frontal region of the brain, if the model included the irregular features of the skull in that region. The model does not include such irregular features, as the resulting element deformations would have caused numerical instabilities.

The models provide a viable hypothesis of the mechanisms behind the frontotemporal contusions that are found at post-mortem. The relative motion of the brain and the skull also explains contusions found at other sites, including the impact region.

Figures 13 and 14 contain a summary of the head injury tolerance curves, which represent moderate injury, determined during the study [13] and the tolerance curves contained within the literature.

The Head Injury Criterion (HIC) is the accepted means of determining the severity of head injuries due to acceleration. Figure 13 contains the translational head injury tolerance curves determined during this study and the tolerance curve which represents a HIC of 1000. This is the value used by the automotive industry as representing an acceptable level of risk of head injury. It makes no allowance for the severity of rotational acceleration. The comparison shows that the HIC equals 1000 curve underestimates the risk of injury at shorter pulse durations and overestimates the risk of injury at longer pulse durations. This has implications when HIC is used to influence the design of motor vehicles in relation to head injuries.

Figure 14 contains the rotational head injury tolerance curves determined in this study and the tolerance curves contained within the literature.

Lowenhielm et al [16] developed this tolerance curve using cadaver experiments and mathematical modelling. Ryan et al [26] used the reconstruction of pedestrian accidents and mathematical modelling to determine the tolerance curve. Both these tolerance curves ignored the important influence of translational acceleration that was present. Both Thibault et al [33] and Ommaya et al [20] used animal experiments as the basis for the tolerance curves. The anatomy of the human head is different from that of even the closest primate. The injury mechanisms are related to the brain's response and the differences in anatomy would produce different responses. These differences are not overcome with the use of simple scaling laws. Bycroft et al [4] used a simplified mathematical model to examine angular accelerations. Stalnaker et al [31] used head injury finite element models to determine a rotational acceleration limit for recoverable diffuse axonal injury. These models were incapable of simulating

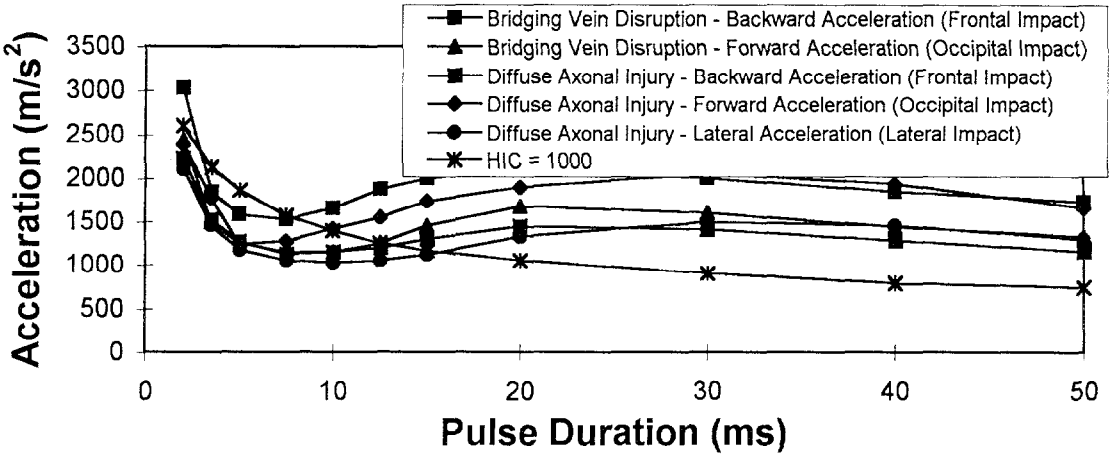

Figure 13 : Translational Acceleration Head Injury Tolerance Curves

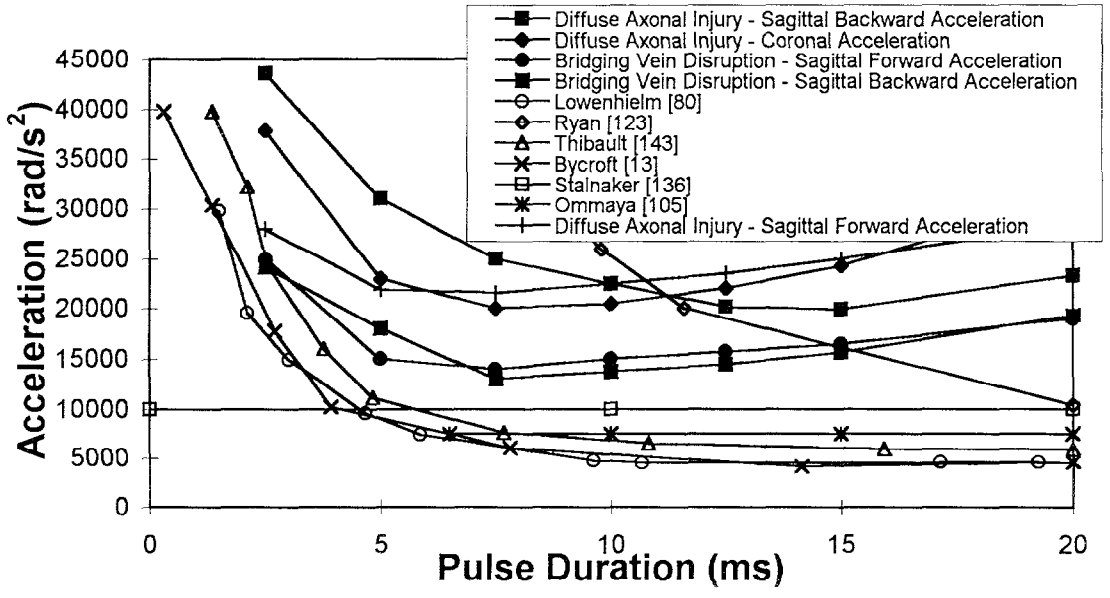

Figure 14 : Rotational Acceleration Head Injury Tolerance Curves.

the complex brain kinematics that occur during rotational accelerations of the head.

All the tolerance curves except that of Ryan et al [26] have limits for concussion or irrecoverable injury that are significantly lower than the acceleration pulses experienced by boxers. These are regularly in excess of 5000 rad/s^2 and can reach 13600 rad/s^2 [21]. These tolerance curves are obviously far too conservative. The tolerance curve of Ryan et al [26] ignored the important influence of translational acceleration. The tolerance curve cannot be relied upon because the translational acceleration is an unknown input which has a significant effect on injury severity.

The head injury models have been successful in determining the biomechanics of head injury and developing improved head injury tolerance curves. The severity of the three head injury mechanisms is related to the consequences of the relative motion of the brain and the skull. The head injury tolerance curves provide a valuable tool for engineering safety system design.

Other head injury finite element models [18,24,35] have concluded that translational acceleration has a minimal effect on the severity of injury. This is due to the modelling assumption used for the brain/skull interface, such that models did not predict realistic motion of the brain with respect to the skull. The models in this study showed that translational and rotational accelerations are key in determining the severity of head injury. This was achieved because of the unique method of modelling the brain/skull interface, allowing the more realistic prediction of levels of relative motion. The different combinations of translation and rotation in terms

of applied direction are important in determining the severity of injury. It is the complex head kinematics that must be considered when trying to predict the severity of head injury. Consider the combination of two tolerable acceleration pulses applied to the coronal model. A translational pulse of 1025 m/s^2 with a 10ms pulse duration produces a corpus callosum maximum shear strain of 10%. A rotational acceleration pulse of 20500 rad/s^2 with a 10ms pulse duration also produces a corpus callosum maximum shear strain of 10%. Combining these two acceleration pulses produces a range of corpus callosum maximum shear strains of 6.0% to 13.0% dependent upon the combination of directions used.

The head injury tolerance curves developed in this study show that HIC is not an accurate predictor of the severity of head injury. It doesn't consider the injury mechanism, direction of applied acceleration pulse or rotational acceleration. These have been shown to have an influence on the severity of head injury. The models also suggest that HIC underestimates the severity of injury at short pulse durations and overestimates the severity of injury at long pulse durations.

DISCUSSION AND CONCLUSIONS

The coronal and sagittal models provide a viable hypothesis behind the classic triad of diffuse axonal injury, bridging vein disruption and frontotemporal contusions found at post-mortem. The relatively high shear strains that are produced in the corpus callosum and brainstem regions are responsible for the corresponding lesions. The high shear strains in the corpus callosum region are caused by the relative motion of the two cerebral hemispheres as a result of the relative motion of the brain and the skull. The high shear strains in the brainstem region are caused by the motion of the cerebral hemispheres being resisted by the brainstem, during the relative motion of the brain and the skull. The lower level shear strains, which spread throughout the brain, are responsible for the microscopical evidence found in the axons of the white matter. The elongation of the spring elements which represent the bridging veins are responsible for the bridging vein disruption. The high compressive strains in the temporal regions of the brain are responsible for the corresponding contusions. The relative motion of the frontal region of the brain is responsible for the corresponding contusions.

Diffuse axonal injury and bridging vein disruption tolerance curves have been developed for translational and rotational acceleration in the coronal and sagittal planes [13]. These tie in with existing head tolerance data and represent an improvement on existing head injury tolerance curves. They demonstrate the directional and injury mechanism dependency of head injuries. The use of a single simple function such as the Head Injury Criterion (HIC) is not sufficient in predicting the severity of head injury. It doesn't consider the injury mechanism, direction of applied acceleration pulse or rotational acceleration. These have been shown to have an influence on the severity of head injury. The models also suggest that HIC underestimates the severity of injury at short pulse durations and overestimates the severity of injury at long pulse durations.

Translational and rotational accelerations both have a role to play in the severity of the relative motion of the brain and the skull and therefore the resulting injury severity. These are the first head injury finite element models that have shown that both translational and rotational accelerations are responsible for the severity of injury. The use of HIC has shown that translational acceleration is responsible for injuries. Reducing the severity of translational acceleration through HIC has led to a reduction in injuries. The many combinations of translational and rotational accelerations that occur in head trauma are critical to the resulting head injury severity. It is therefore necessary to determine the complex kinematics of the head during head trauma if a reliable estimate is to be made of the severity of injury.

Further research will aim to develop a three dimensional model with an improved representation of the brain/skull interface. This interface representation will aim to explicitly represent the fluid and decoupling process.

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