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Simulation of the development of frontal head impact injury

M. D. Gilchrist, D. O'Donoghue

Abstract This paper describes the results of computational simulations of a frontal impact to the head and the predicted development of coup and contrecoup contusion (i.e., at and opposite the site of impact, respectively) within brain tissue. Three separate two-dimensional plane strain finite element models of the head, each of which incorporated the skull, the cerebrospinal fluid and the brain, were constructed. Two of these models represented the coronal plane of the head as being elliptic whilst the third model was geometrically representative of an actual human head. This third model was taken in an off-centre mid-sagittal plane in an anterior–posterior direction and all three models were used to investigate the dynamic response of the human head when subject to direct translational impact events. The physiological consequences of modelling the human brain as being elastic were established. Compressive and tensile strains were predicted at the coup and contrecoup sites for a simulated frontal impact event by means of a simple elastic analysis. These distributions of most severe strain correspond directly to the occurrence of coup and contrecoup contusion such as are witnessed in clinical studies which arise under the action of translational acceleration.

1 Introduction

Injury to the head constitutes one of the major causes of death in road traffic accidents. Despite increased use of protective devices such as seat belts, airbags, safety restraints and safety helmets, brain injury disables or kills someone in the United States every two and a half minutes. The annual cost of hospitalisation and rehabilitation within the United States has been estimated to be some twenty five billion dollars (Meaney et al., 1994).

Due to its complex nature, functional and traumatic damage to the brain is difficult to quantify and it is because of this complexity that a variety of alternative mechanisms have been proposed for explaining the development of brain injury. All theories, however, agree that injury is related to acceleration or deceleration of the head regardless of whether the impact is applied directly or indirectly. The brain is loosely coupled to the skull and consequently its motion will lag that of the skull, resulting in 'bruising' of the brain as it impacts against the interior surface of the skull. Stretching of the tethering blood vessels, which arises as a result of this lag in motion, can cause them to strain excessively, rupture, fail and bleed. The brain tissue itself may be damaged by normal and shear forces that develop during translational and rotational accelerations or decelerations.

Biomechanical research attempts to understand the development of injury and thereby help to avoid or alleviate the damage that can occur from various impacts. This can only be achieved by first understanding the mechanics of impact and the biomechanical response of the head (i.e., the skull-brain system) to a variety of loading conditions. Brain injury mechanisms are generally described in terms of the mechanical and physiological changes that result in anatomical and functional damage (Viano et al., 1989).

The constitutive properties of both the skull and brain influence the system response to mechanical loads and must be known if the physical response of the skull-brain system is to be predicted accurately (Dassios et al., 1998). This has posed significant difficulties for researchers in recent decades following the development of more powerful computational resources (Nisitani & Chen, 1997), since the properties of living human tissue deviate from those of cadavers and primates and no harmless non-invasive procedures for establishing such properties in vivo have been established.

Finite element modelling offers significant potential for understanding and predicting the mechanical and physical response of a brain to impact loadings. The present paper is a description of preliminary investigations that have been carried out into the analysis of translational impact events by means of various two-dimensional finite element models.

2 Mechanics and mechanisms of head injury

A dynamic force applied to the head induces a complex series of mechanical and physical reactions involving local bending of the skull, volume changes to the intracranial contents, shock waves propagating throughout the brain and inertial effects, all of which induce tissue strains and stresses which may give rise to damage of the scalp, cranium, blood vessels or brain matter (O'Donoghue and Gilchrist, 1998a, b; O'Donoghue, 1999). It is clear that the mechanics that arise during a given head impact event are difficult to define (Jones et al., 1998). This is not surprising

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as the consequence of the mechanics, i.e. tissue damage, seldom can be detected macroscopically and normally only can be observed by neurophysiological examination after the impact. Moreover, brain damage associated with non-penetrating or closed head injuries tends to be spread over a large area. The type of injury is determined by the location and severity of the mechanical distortion of the skull bone, blood vessels and brain tissue. There has been much consequent speculation and several independent and conflicting theories proposed to explain the different head injury mechanisms.

Rotational injuries may be produced by direct or indirect impacts to the head. Translational (linear) injuries involve direct linear acceleration of the head and lead to contusions while diffuse axonal injuries are commonly associated with rotational acceleration impacts. Concussion and haematomas may result from either translational or rotational acceleration impacts.

Shear strains that are induced within the brain as a result of the action of rotational accelerations can damage the bridging veins, thus leading to haemorrhaging (bleeding) and haematomas. As rotation continues over time, the shear strains would extend towards the inner area of the brain resulting in diffuse injuries. There is significant clinical evidence to support the belief that diffuse injuries are produced as a consequence of shearing of the brain tissues by rotational acceleration forces. However, the mechanics that govern rotational forces fails to explain the occurrence of coup and contrecoup injuries (i.e., at and opposite the site of impact, respectively) and it is currently accepted that translational (linear) acceleration is also responsible for causing severe injury.

A number of theories have been proposed to explain how the brain is injured as a result of translational acceleration of the head (eg. Gross, 1958; Lindenberg and Freytag, 1960; Gurdjian, 1972). These theories include pressure waves passing through the brain, deformation of the skull, brain deformation at the skull and the formation of pressure gradients. These theories all aim to explain the phenomenon of contrecoup injury where, in addition to injury at the site of impact, injury is often witnessed at the opposite site, namely the contrecoup site.

A centripetal theory was proposed by Ommaya and Gennarelli (1974) to account for both rotational and translational forms of injury. This theory was formulated as a result of their laboratory experiments investigating the response of the primate head to acceleration. Rotation of the head appeared to be necessary to produce certain types of damage, namely those involving loss of consciousness. The centripetal theory states that the distribution of diffuse damage resulting from inertial loading would decrease in magnitude from a maximum at the surface to a minimum at the centre of the intracranial contents. Hence, for low levels of impact severity, injury would occur only at the surface and more severe impacts would be required to cause injury towards the central regions of the brain. The severity and location of injury that will be induced depends on the constitutive properties of the brain matter and the immediate shape of the bony environment of the interior surface of the skull. However, this same centripetal theory also acknowledges that translation of the head in the horizontal plane produces another type of injury, namely bruising of the brain's surface, and that the site of impact is important when considering the severity of this injury outcome.

After studying a number of post mortal cases and the blows that caused each death, Willinger et al. (1992) deduced that impacts against hard surfaces lead to contusions and subdural haematomas, while impacts against soft surfaces give rise to diffuse axonal injury. An impact against a hard structure takes place over a shorter period while an impact with a soft structure results in a longer impact duration. A longer duration is required for the development of serious damage resulting from rotational acceleration than is required from translational acceleration. Hence, the dominant injuries for shorter impact durations are those that can be attributed to translation while those due to rotation are associated with longer impact durations. Soft structures act to absorb impact energy and decrease the conditions which cause focal injuries. However, they result in lower acceleration rates and the consequent longer duration of the impact pulses may aid the development of diffuse injuries.

The two-dimensional models of the present investigation have only been used to simulate the effects of translational accelerations to identify the occurrence of coup and contrecoup contusion due to frontal impact events.

3
Constitutive and geometric properties of head models

The constitutive properties of human brain have been the subject of much research within recent decades. Since these properties cannot be obtained by means of in vivo tests on humans, alternative methods have been used such as in vivo tests on primates with an associated scaling law or an in vitro examination of human brains. The main concern with the former method is the lack of an adequate scaling law (Ljung, 1978), whilst it is uncertain how the properties obtained from the latter method are altered as the brain dries out once blood supply has ceased (Viano et al., 1989).

The tissues of the brain are quite heterogeneous on both the macroscale and the microscale. The mechanical properties of the brain and cerebrospinal fluid will affect the pressure response of the brain. Thus any model of the intracranial system that is excessively stiff or compliant will not accurately simulate a physical head response. The intracranial contents have, on the whole, been treated as incompressible comparable with water. However, the brain tissues are more similar to a gel-like material containing approximately 77–78% water. The opening at the base of the skull, the foramen magnum, and the presence of the lateral ventricles allow the pressure of the intracranial contents to change. These effects can be simulated by assuming a certain degree of compressibility for the intracranial contents, (Ruan et al., 1994) with a Poisson's ratio for the brain material in the range of 0.48–0.499.

The fact that the ventricles are filled with cerebrospinal fluid and many blood vessels are present in the brain indicate that the bulk modulus of the brain should be somewhat lower than that of water. Since brain motion can lag behind that of the skull during an impact the fluid in the subarachnoid space would be expected to have a lower shear modulus and bulk modulus than that of the brain in order to represent the fact that it can move within the skull.

The isotropic homogeneous constitutive mechanical properties that were used within the three models of this investigation are given below in Table 1 and were prescribed for a three layered skull (model 1 only), an isotropic skull (models 2 and 3 only), a subarachnoid space filled with cerebrospinal fluid and an isotropic brain.

The cerebrospinal fluid acts as an important damping mechanism within the skull-brain system. Higher contrecoup injuries have been witnessed where high accelerations are involved prior to impact, such as during a fall. This is attributed to the theory that the cerebrospinal fluid moves towards the impact site during the fall, leaving the contrecoup site relatively deficient of cerebrospinal fluid which has been confirmed experimentally by Edberg et al. (1963). Consequently, this implies that any computational model must consider the influence of the cerebrospinal fluid when attempting to predict the physical response of the brain to a mechanical impact.

The subarachnoid space, within which most of the cerebrospinal fluid is contained, is non-existent over the surface of the gyri (where the irregular surface of the brain rises according to the folds of the cerebral cortices), is relatively small where the arachnoid bridges over small sulci (where the cerebral surfaces falls), and is much larger in certain locations where it bridges over larger surface irregularities. The cerebrospinal fluid has, to date, been modelled as a uniform layer whereas in reality it is a dispersed collection of discrete pockets along the longitudinal fissure. The manner of representing the cerebrospinal fluid is addressed with increasing accuracy in each of the three models that are described in Sect. 4 below.

4
Development of computational models

Three two-dimensional models of increasing degrees of complexity were developed and used to investigate the effects of impact load conditions upon the skull-brain system. The constituent components of these models were assumed to be homogeneous, isotropic and linearly elastic. These are shown successively below in Figs. 1–3.

Table 1. Isotropic constitutive properties used in elastic analysis of head impact for models 1, 2 and 3. (After Ruan et al., 1994 and Ruan & Prasad, 1996)

Material	Young's modulus (MPa)	Density (kg/m^3)	Poisson's ratio
Outer table (model 1 only)	5465	3000	0.22
Dipole (model 1 only)	2684	1750	0.22
Inner table (model 1 only)	5465	3000	0.22
Cerebrospinal fluid	0.1485	1040	0.499
Brain	0.558	1040	0.485
Skull (models 2 and 3)	6650	1410	0.22

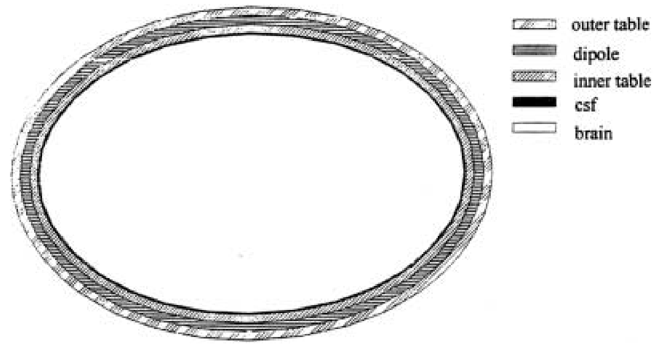


Fig. 1. Two-dimensional model (Model 1) of coronal plane of head which includes a three layered skull, cerebrospinal fluid and brain tissue

The first of these models, Fig. 1, treats the cerebrospinal fluid as a discrete layer surrounding a uniformly isotropic brain whilst the second model, Fig. 2, is more sophisticated since it represents the cerebrospinal fluid as a discrete collection of pockets distributed along the surface of the brain. The same volume of cerebrospinal fluid was used in these two models. Both of these models account for the cranium as a three layered material consisting of the outer table, the dipole and the inner table. Both models 1 and 2 assume the skull to be perfectly elliptic and uniformly 11.1 mm thick (each sublayer being 3.7 mm thick). Whilst this two-dimensional representation of the coronal plane of the head is an approximation to a three-dimensional ellipsoid, it cannot accurately provide quantitative estimates of the distribution of stresses or strains within either the skull or the intercranial contents. This is solely due to the fact that such a two-dimensional representation of the skull cannot represent the lateral constraints that would be provided by a three-dimensional ellipsoid. However, despite this limitation on these two-dimensional models, they can usefully provide an indication of the instantaneous distribution of stresses and strains within a midsection slice of the brain, cerebrospinal fluid and skull, following an impact event.

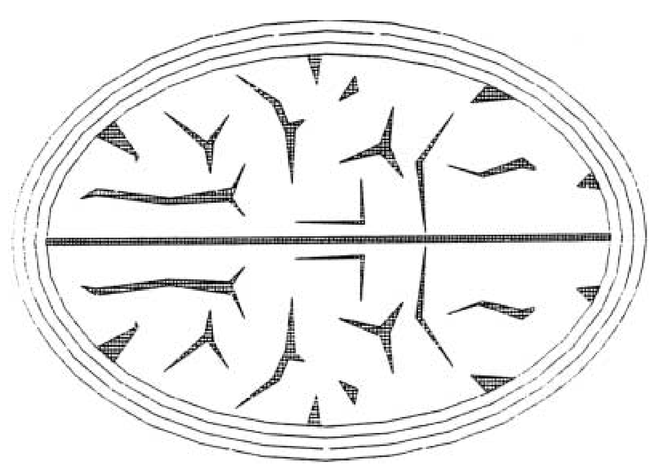


Fig. 2. Two-dimensional model (Model 2) of coronal plane of head which includes the skull, discrete pockets of cerebrospinal fluid and brain tissue

Models 1 and 2 were subjected to an identical short duration impact force that was collinear with the major axis of the models; this represented a frontal impact. Both models predicted the generation of compressive strains at the coup site, i.e., under the site of impact, and tensile strains at the contrecoup site, i.e., opposite the site of impact. Whilst these results are not presented here (results are presented only for model 3, which is more sophisticated), model 2 indicates that more localised strain gradients develop within the brain tissue than in model 1. This suggests that correct estimates of the levels of neurological trauma can only be predicted by including the effect and localised distribution of cerebrospinal fluid surrounding the brain matter. Furthermore, these greater strain gradients and strain concentrations were predominantly located around regions of brain matter that was deficient of cerebrospinal fluid. This suggests that some of the impact energy arising from an impact event is absorbed by the cerebrospinal fluid, which can be considered as acting partially to protect the brain tissue.

Model 3 is not based upon any regular mathematical geometry but rather is generated by taking a planar section from a topologically accurate three-dimensional model of a brain. A two-dimensional plane strain finite element model was constructed in the mid-sagittal plane of the head in an anterior–posterior direction; the outline of this model is shown in Fig. 3. The geometry of the model has been carefully defined to incorporate the uneven surface of the brain. The model is dimensionally accurate and accounts for the irregular topology of the sulci and gyri of the cortex and the varying thickness of the cranium. Consequently, the location of all cerebrospinal fluid is also geometrically correct. A single layer skull of varying thickness was modelled encompassing the brain. The two-dimensional slice was generated slightly off-centre in order to avoid the falx cerebri and corpus callosum. The length of the model measures 192 mm from anterior to posterior and, as such, represents an average male human head.

The mesh for the finite element model was generated in MSC/Patran (1998) using four noded isoparametric quadrilateral elements, all of which were of unit thickness. The finite element model, which is shown in Fig. 4, contained 12081 elements and 25766 degrees of freedom. The material properties of the model were assumed to be homogeneous, isotropic and linearly elastic and are as given previously in Table 1. Boundary conditions were chosen to simulate restraint against large rigid body motions at the base of the cranium by constraining the relevant nodes in all six degrees of freedom.

5
Results and discussion

An elastic analysis of a frontal impact, which corresponded to cadaveric experiments of Nahum et al. (1977), was performed using model 3 (cf. Figs. 3 and 4). These experiments had examined the consequences of a blow to the head of a seated human cadaver. The impact was directed to the frontal bone in the mid-sagittal plane in an anterior–posterior direction. The inter-cranial pressure-time histories were recorded by accelerometers in the frontal bone adjacent to the impact area and in the occipital bone. These pressure locations had been termed coup and contrecoup respectively by Nahum et al.

The impact from the experiment was simulated by imposing an approximately half sine wave force with a peak value of 8000 N on the frontal bone as shown in Fig. 5. A transient dynamic analysis was performed in ABAQUS for a 10 ms period with a time step of 0.5 ms.

The computed strain levels in the cerebrospinal fluid were identified and the manner in which they varied during the impulse was compared against the results of Nahum et al. which had been recorded in terms of subdural pressures in the cerebrospinal fluid. These are compared below in Figs. 6 and 7.

The trend of the computational results agree well with the experimental data for the duration of the pulse: negative strains, indicating compression, were predicted at the coup site for the duration of the impact as shown in Fig. 7.

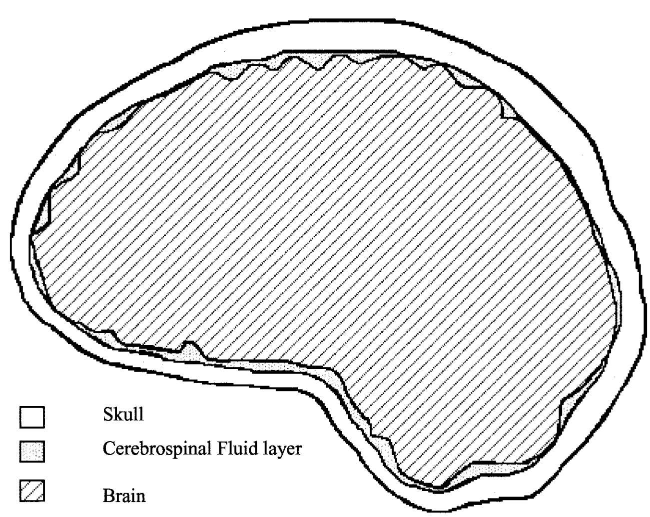


Fig. 3. Two-dimensional head model (Model 3) taken in the off-centre mid-sagittal plane which includes brain tissue, cerebrospinal fluid and skull. This geometrically accurate model is 192 mm long and corresponds to an adult male head

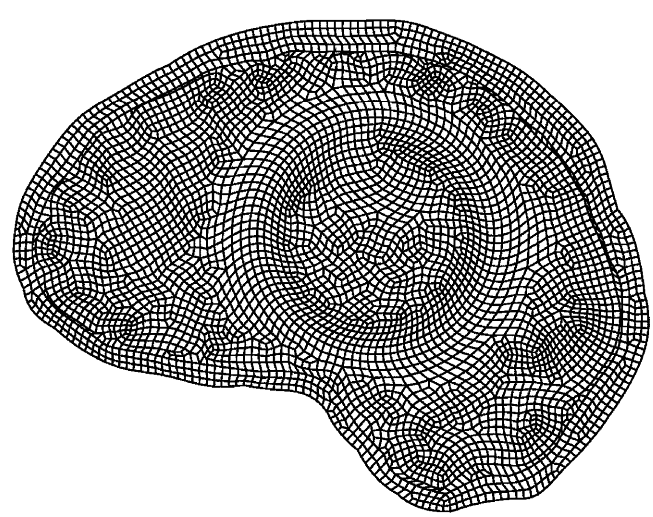


Fig. 4. Two-dimensional finite element mesh (Model 3) of the head (cf. Fig. 3) containing 12081 elements

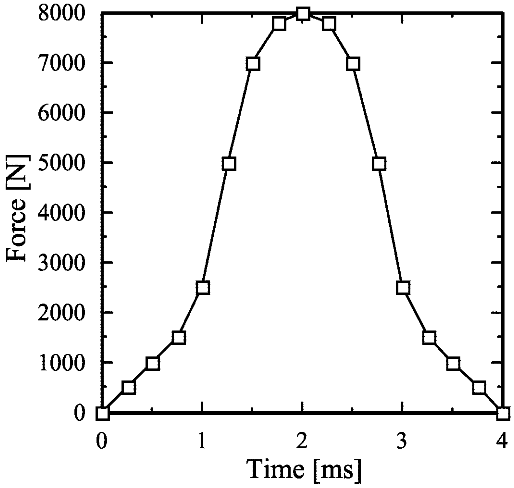


Fig. 5. 4 ms impact pulse time history (approximate half sine wave) used to simulate the cadaveric impact experiments of Nahum et al., 1977

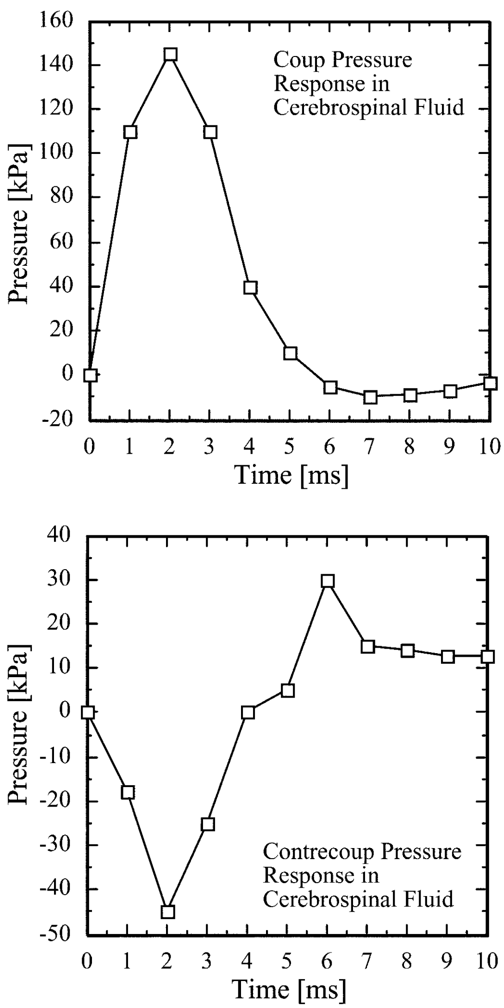


Fig. 6. Coup and contrecoup pressure time histories from the cadaveric impact experiments of Nahum et al., 1977

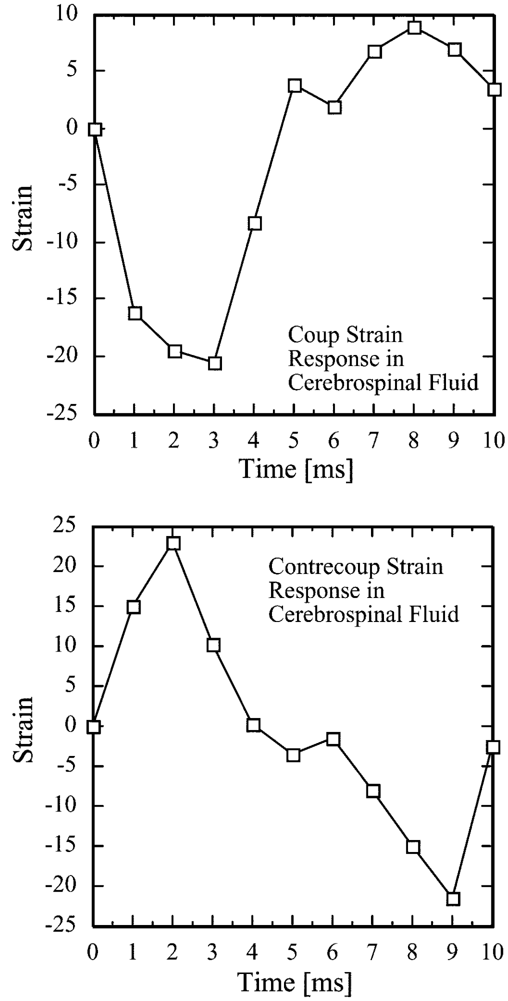
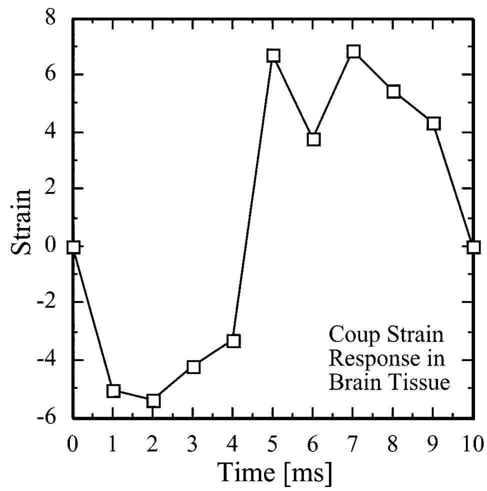


Fig. 7. Maximum levels of strain predicted for coup and contrecoup regions within the cerebrospinal fluid using Model 3 (cf. Figs. 3 and 4) due to the frontal impact pulse of Fig. 5

These negative strains correspond to the positive pressure pulse of the experimental data which indicates a period of compressive stress. The predicted response of the computational model also indicates that a period of tension, represented by positive strains, will exist in the coup site after approximately 4 ms. This agrees well with the qualitative experimental results of Nahum et al. which indicate that negative pressure (i.e., tension) also exists in this region for this period after the impact.

The response of the computational model predicted tensile strains within the cerebrospinal fluid at the contrecoup site for the duration of impact. Again, these results were in close agreement with the experimental findings which showed negative pressure at this site (i.e., tensile stresses) followed by a period of positive pressure after the force has been removed. This compressive region (i.e., positive pressure) was also predicted by the computational model as indicated by negative strains once the impact had been removed.

The strain levels in the brain were also predicted by the present computational model and the time history responses of the coup and contrecoup sites are shown in Fig. 8. At $t = 0.5$ ms, compressive strains were predicted



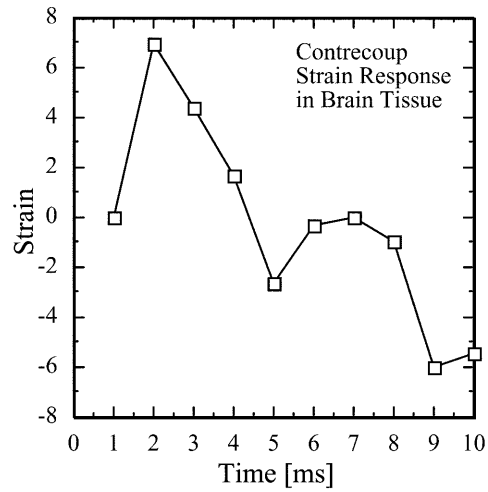


Fig. 8. Maximum levels of strain predicted for coup and contrecoup regions within the brain tissue using Model 3 (cf. Figs. 3 and 4) due to the frontal impact pulse of Fig. 5

at the coup site. These strains were of a lower magnitude than those predicted in the surrounding cerebrospinal fluid. No strains were predicted at the contrecoup site of the brain at this time of 0.5 ms although the cerebrospinal fluid in that region did predict the presence of tensile strains. At $t = 1$ ms, the strain within the coup site remains compressive but is of an increased magnitude as the force increases; the response of the complete model at this time of 1 ms is shown in Fig. 9. Tensile strains are predicted for the contrecoup site as being of a lower magnitude than those predicted for the adjacent cerebrospinal fluid.

After $t = 1.5$ ms, both compressive and tensile strains become apparent throughout a larger area, the magnitudes of strain increasing until $t = 4$ ms with the cerebrospinal fluid always predicting a greater level of strain than that predicted within the brain. After $t = 4$ ms the levels of strain decrease as expected since the impulse force has been removed. However, the regions affected by the tensile and compressive strains continue to grow throughout the remainder of the analysis and tensile strains at the coup site give way to compressive strains while compressive strains at the contrecoup site give way to tensile strains.

6 Conclusions

Three separate two-dimensional plane strain finite element models of the head, each of which incorporated the skull, the cerebrospinal fluid and the brain, were constructed. The first two of these models represented the coronal plane of the head as being elliptic whilst the third model was geometrically representative of an actual human head. This third most sophisticated model was taken in an off-centre mid-sagittal plane in an anterior–posterior direction. These three models were used to investigate the dynamic response of the human head when subject to direct translational impact events. The physiological conse-

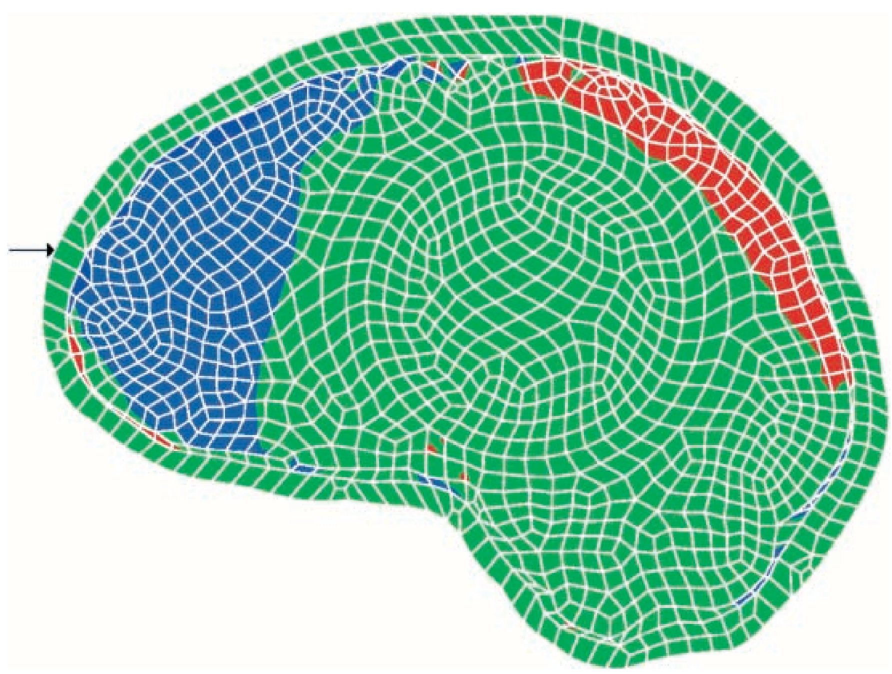


Fig. 9. Response of the elastic head model, Model 3 (cf. Figs. 3 and 4), after 1 ms due to the frontal impact pulse of Fig. 5. Negative compressive strains are seen in the cerebrospinal fluid and brain tissue at the impact (coup) site whilst positive tensile strains exist in the cerebrospinal fluid and brain at the opposite (contrecoup) region. The impact site is indicated by the arrow

quences of modelling the brain as elastic were established. The following conclusions can be made:

1. Correct estimates of the levels of neurological trauma can only be predicted by including the localised distribution of cerebrospinal fluid surrounding the brain tissue.
2. Some of the impact energy arising from an impact event is absorbed by the cerebrospinal fluid, which can be considered as acting partially to protect the brain tissue.
3. Compressive and tensile strains have been predicted at the coup and contrecoup sites for a frontal impact event by a simple elastic analysis. These correspond directly to coup and contrecoup injuries such as are witnessed in clinical studies which involve translational (linear) acceleration.
4. The present elastic analysis predicts that compression (negative strain or positive pressure) develops at the site of coup contusion with tension (positive strain or negative pressure) developing at the contrecoup site immediately following frontal impact.
5. Accurate modelling of the material properties of the intracranial contents is essential if the correct magnitude and distribution of strains is to be predicted. Excessively stiff or compliant systems will not permit the prediction of an accurate response.

Future publications in this area will address other impact scenarios as well as the consequences of modelling the constitutive response of brain matter as being viscoelastic. This will provide additional insight into the rate dependent nature of the response of brain tissue.

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