Physical Model Studies of Cortical Brain Deformation in Response to High Strain Rate Inertial Loading

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ABSTRACT

Acute subdural hematoma (ASDH) is a common form of severe head injury and is characterized by a high mortality rate. Studies in our laboratory have shown that ASDH can be produced in the primate by subjecting the head to short duration, sagittal plane loading. While these animal experiments provide an absolute confirmation of injury, they yield no information regarding the changes in mechanical field parameters that occur during the dynamic loading period. Developing tolerance levels for ASDH in man requires integrating the results from these primate studies with information from two parallel studies - measurement of the superior margin deformation that occurs in response to a range of inertial loads, and the development of a failure criterion for the cortical vasculature.

The objective of this study was to measure the response of an inanimate model of the head subjected to a range of short duration, sagittal plane loading conditions. Human or primate skulls were cut parasagittally and filled with an optically transparent gel exhibiting static mechanical properties in the range of values reported for primate brain tissue. The model was subjected to a range of dynamic loading conditions both within and outside the region of scaled loading levels associated with ASDH in the primate studies. The motion of an orthogonal grid located parasagittally within the surrogate brain tissue was used to measure the cortical deformation occurring in response to an inertial loading level.

Peak superior margin strain (ε) was found to increase as the peak angular deceleration increased, and was maximum in the frontal region. This regional variation of cortical strain indicates the disruption of parasagittal bridging veins is most likely to occur in the frontal region, an hypothesis supported by pathological information from the primate studies which showed that subdural hematomas were frontally predominant. Further, the results from this physical model study was compared to tissue failure relationships available for parasagittal bridging veins. The inconsistency found between the predicted injury outcome using two different failure criteria and the scaled primate data emphasize the need for a more comprehensive study regarding the biomechanics of ASDH, focusing on extending the physical modeling work presented in this report and developing tissue failure criteria for perfused parasagittal bridging veins across a range of age groups.

INTRODUCTION

Observations from primate studies performed in our laboratory have indicated that a series of injuries (acute subdural hematoma, mild to severe concussion, and prolonged coma) can be produced by subjecting the primate head to non-contact, inertial loading [1, 4]. Moreover, these studies indicate that the type of injury produced depends strongly on the temporal and directional characteristics of the applied load [5]. Short duration inertial movement of the head in the sagittal plane produced rupture of the parasagittal bridging veins, whereas diffuse axonal injuries located in the deep white matter were produced by using longer duration, coronal plane loading.

These animal experiments serve as an absolute confirmation of the injury type, but do not afford the opportunity to measure the mechanical field parameters. A key in understanding the mechanical conditions needed to initiate these injuries lies in the ability to measure the mechanical deformation at the site of injury during the dynamic loading period. In this regard, inanimate physical models offer the opportunity to measure the response of surrogate brain tissue to applied loading. Previous modeling investigations have focused on using physical models of the head to measure the spatial and temporal distribution of coronal plane deformation in response to inertial loading [10, 13, 14]. The information derived from this study and the University of Pennsylvania animal studies was integrated to suggest a tolerance level for gradations in diffuse axonal injury in the primate and man [9].

The purpose of this report is to study the mechanical response of an inanimate model of the head that was subjected to a range of short duration, sagittal plane inertial loads. The results from this investigation will be used to study the relationship between cortical brain deformation and inertial loading level and will also yield empirical evidence on the variance of cortical brain deformation as a function of anatomic location. The latter issue addresses a hypothesis which has been offered recently [6] regarding the attachment angle of the bridging veins to the sagittal sinus and the critical role of this angle in the biomechanics of acute subdural hematoma. A recent study [2] indicates that the attachment angle over much of the superior margin is closer to 90° than previously thought, indicating this angle may not be the most important parameter to consider when formulating an injury specific tolerance level for subdural hematoma. Rather, the preferential failure of the parasagittal bridging veins is most probably related to the geometry of the brain, the temporal nature of the applied load, and the constitutive properties of the vasculature.

MATERIALS AND METHODS

Construction of physical model

The physical model used in this study (Figure 1) followed the same general procedures used in previous investigations by Thibault et al. and Margulies [10, 14] An adult human skull (Carolina Biological Supply, Raleigh, NC USA) was prepared by cutting the skull 1.5 cm lateral to the sagittal midline and machining the foramen magnum to facilitate the insertion of a surrogate spinal column. The interior of the skull and spinal column were coated with flat white enamel to enhance photographic resolution during high speed filming. A cylindrical aluminum can was fabricated in such a manner to encase the skull/spinal column assembly, and a polymer/resin mix was used to fix the skull in position inside the aluminum can.

The interior of the skull/ spinal column was cleaned in preparation for the initial layer of surrogate brain tissue. A polymer gel (Sylgard Medical Gel, Dow Corning, Midland MI, USA) with mechanical properties similar to brain tissue [8] was poured into the model to a level 1.0 cm from the sagittal midline. After the gel had cured, an orthogonal black enamel grid (7 mm spacing) was painted on the gel

surface and allowed to dry thoroughly to permit any paint solvents to evaporate. After drying, a second layer of gel was poured to the level of the sagittal midline and a second grid was painted in the spinal column region. After full drying of the enamel, the final layer of gel was poured and allowed to cure. This process does not create mechanical discontinuities within the surrogate, i.e. adjacent interfaces are self-adherent and do not separate during testing. After curing of the final gel layer, the completed model was stored in a cool, dry environment to minimize entrapment of water vapor which results in an alteration of the refractive index of the gel.

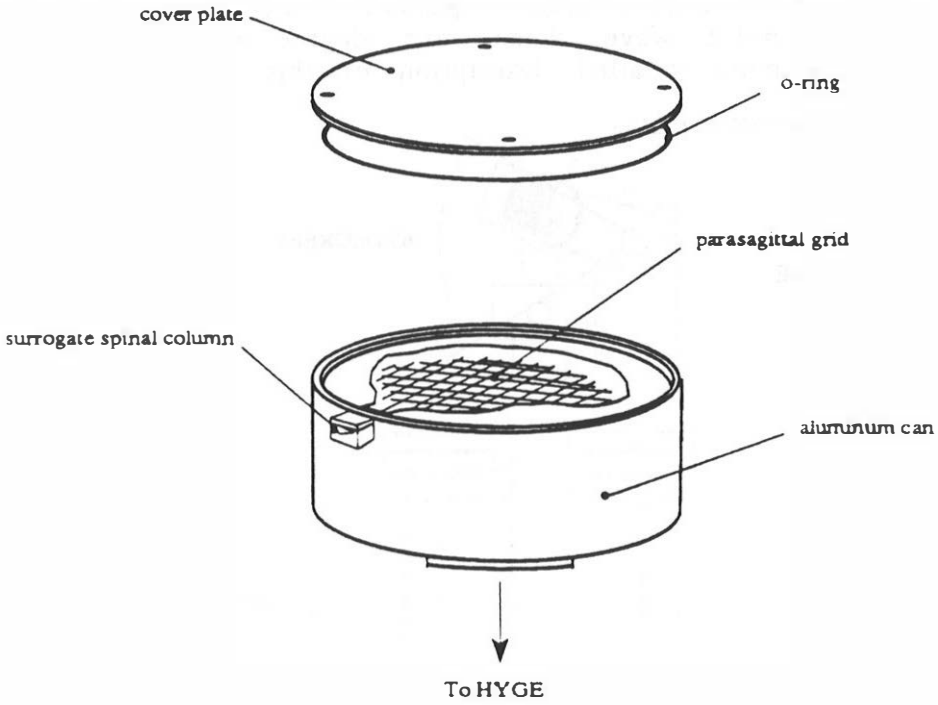


FIGURE 1

Mechanical testing of surrogate brain tissue

Due to both changes in the environmental variables that occur during the gel curing process and the physical characteristics of the gel, the mechanical properties of the gel will differ slightly from the constitutive properties of primate brain tissue it is designed to simulate. A normalization technique was used to normalize the field variable computations to a single value of the constitutive properties based on primate brain tissue.

The technique used to determine the mechanical properties of the gel was based on the rigid indentor technique used in previous studies [3, 8]. The measured force (F) caused by advancing a rigid teflon indentor (diameter d_O) a distance h into the face of a gel sample (elastic modulus E_{gel}, Poisson's ratio υ=.5) can be used to calculate the elastic modulus of the gel (Egel) [15]:

$$E_{gel} = \frac{(1 - \nu^2) F}{d_o h}$$

The elastic modulus of the gel (E_{gel}=.417 psi) was compared with the elastic modulus of primate brain tissue (E_{brain}=.437 psi) [3] to yield a scaling factor that was used to normalize the measured strains in the physical model with respect to primate brain tissue:

$$\varepsilon_{norm} = \frac{E_{gel}}{E_{brain}} \varepsilon_{gel}$$

Acceleration Apparatus

The device used to accelerate the physical models has been used successfully in previous animal and physical model experiments [4, 10, 13]. The device consists of a six-inch diameter Bendix HYGE actuator and a linkage assembly which delivers a distributed inertial load to the primate head or physical model (Figure 2). The peak acccleration/deceleration, pulse wave shape, and degree of excursion can be modified as desired. A more detailed description of this system can be found elsewhere [4, 10].

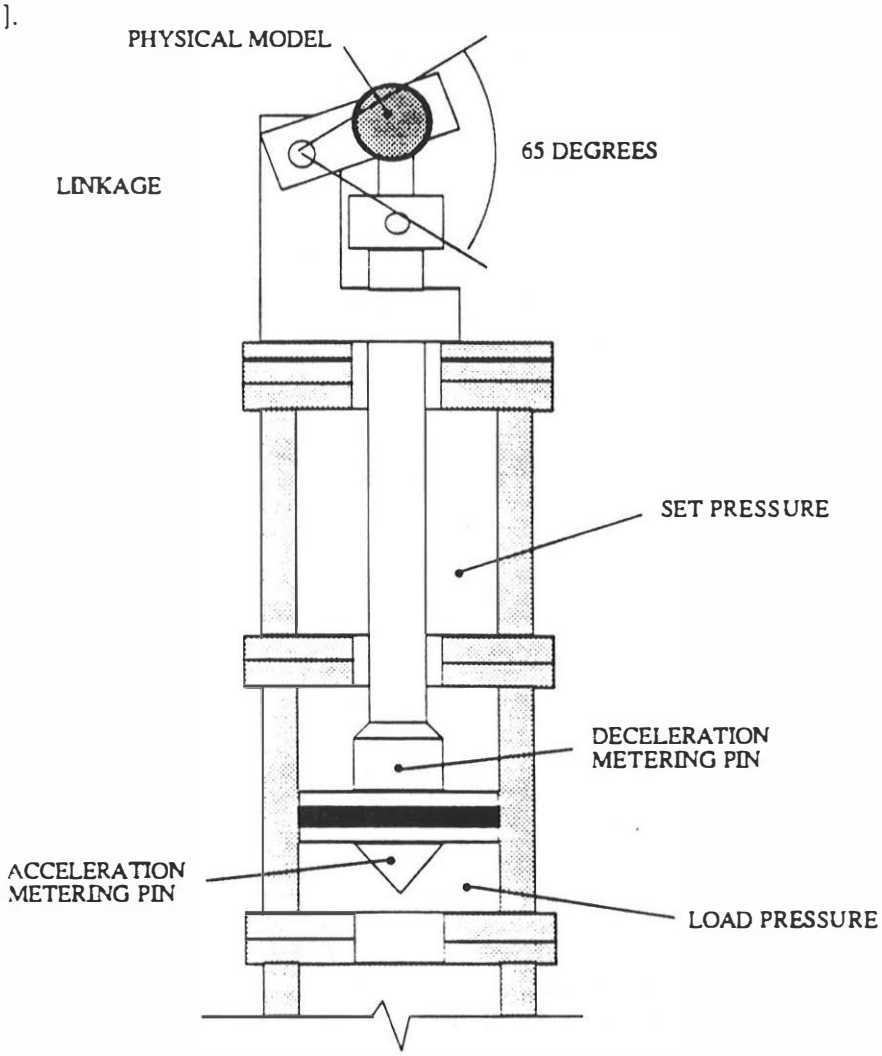


FIGURE 2

A uniaxial accelerometer (Endevco Instruments, San Juan Capistrano CA, USA) mounted on the linkage arm was used to record the magnitude of the acceleration pulse in the direction tangent to the angular motion of the model. The acceleration data was amplified, stored on a recorder (Endevco Instruments) and plotted. Later, the acceleration trace was digitized and stored on computer disk.

Data Analysis

The motion of the physical model was filmed using a HYCAM high speed camera (Redlake Industries Inc.) operating at a rate of 6600 frames/sec. Photographs were developed from the high speed film frames, and the position of the grid intersections in each photograph were recorded and stored on computer disk.

Positions of specific grid intersections were used to determine strain and strain rates in the desired region of interest for the duration of the experiment.

RESULTS

The physical model in this study was subjected to distributed inertial loading that was intended to simulate the high strain rate loading conditions that were used in previous primate studies [1, 4]. An example of the recorded tangential acceleration is shown in Figure 3. As can be seen from this figure, the acceleration wave shape can be described as a biphasic, with a predominant deceleration occurring over a relatively short pulse duration (approx. 6 ms).

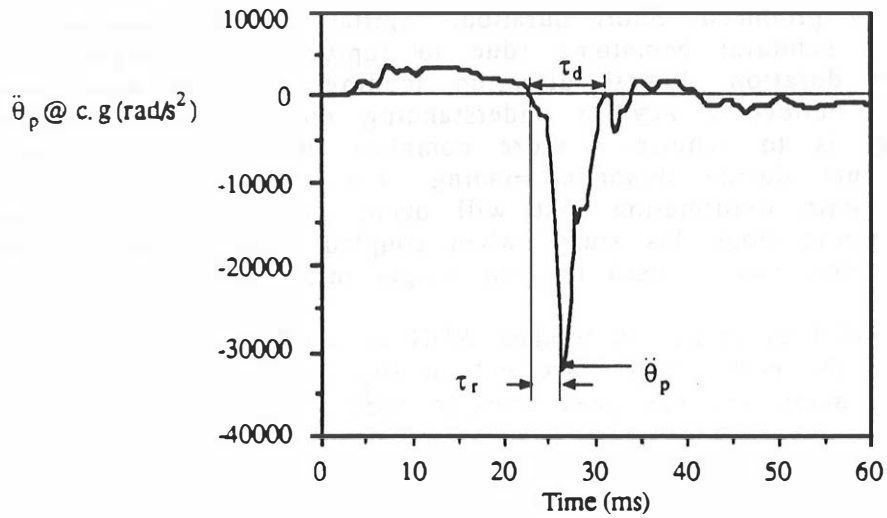


FIGURE 3

Three variables will be used in this report to characterize a given loading level: the peak angular deceleration (θ_p, rad/s^2) measured at the model c.g., the time period over which the deceleration occurs (τ_d, ms), and the elapsed time from the beginning of the deceleration pulse to the maximum deceleration (τ_r, ms). The values for θ_p, τ_d, and τ_r for the three loading levels used in this study are shown in Table 1.

TABLE 1

Cylinder set pressure (psi)	θ_p (rad/s^2)	τ_r (ms)	τ_d (ms)
15	13900	3.2	6.2
25	16200	2.8	6.2
35	32100	2.0	5.8

The cortical deformation measured in this study was described in terms of strain (ε), which was defined as:

$$\varepsilon(t) = \frac{(l(t) - l_o)}{l_o}$$

where $l(t)$ was the length of a grid segment at time t, and l_o was the original length of the line segment at time t=0. The original length of the line segment was defined as the distance of a line extending perpendicular from the inner surface of the skull to a specified grid intersection. This measurement method was intended to reflect the approximate the attachment angle of the bridging veins to the sagittal

sinus over much of the cortex (90°, [2]), thereby providing a reasonably accurate representation of the elongation witnessed by the parasagittal bridging veins. The strain for three superior margin locations (frontal, parietal, and occipital regions) were measured at each loading level. Results from these measurements will be discussed in the next section.

DISCUSSION

In its present form, the head injury criterion (HIC) index does not differentiate between the most common forms of head injuries (concussion, prolonged coma, acute subdural hematoma). A series of primate studies performed in our laboratory have shown that temporal and directional characteristics of the inertial loading applied to the primate head synergistically contribute to the type and severity of injury produced. Short duration, sagittal direction loading produced concussion and acute subdural hematoma (due to rupture of parasagittal bridging veins), while longer duration, lateral direction loading produced concussion and prolonged coma. We believe a key in understanding the differences exhibited in these primate studies is to acquire a more complete understanding of the brain deformation that occurs during dynamic loading. This study will focus on the changes in cortical brain deformation that will occur in response to a range of inertial loads. The results from this study, when coupled with a failure criteria for parasagittal bridging veins, can be used to gain insight into the onset of SDH in the human.

The loading conditions needed to produce SDH in the primate (Figure 4) can be described in terms of the peak angular acceleration (θ_p) and peak change in angular velocity ($\Delta\theta$). The values for the peak angular acceleration and peak change in angular velocity shown in in this figure have been scaled to a brain mass of 1200 grams using Holbourn's scaling relationship [11]. Also shown in this figure are the loading levels used in the present

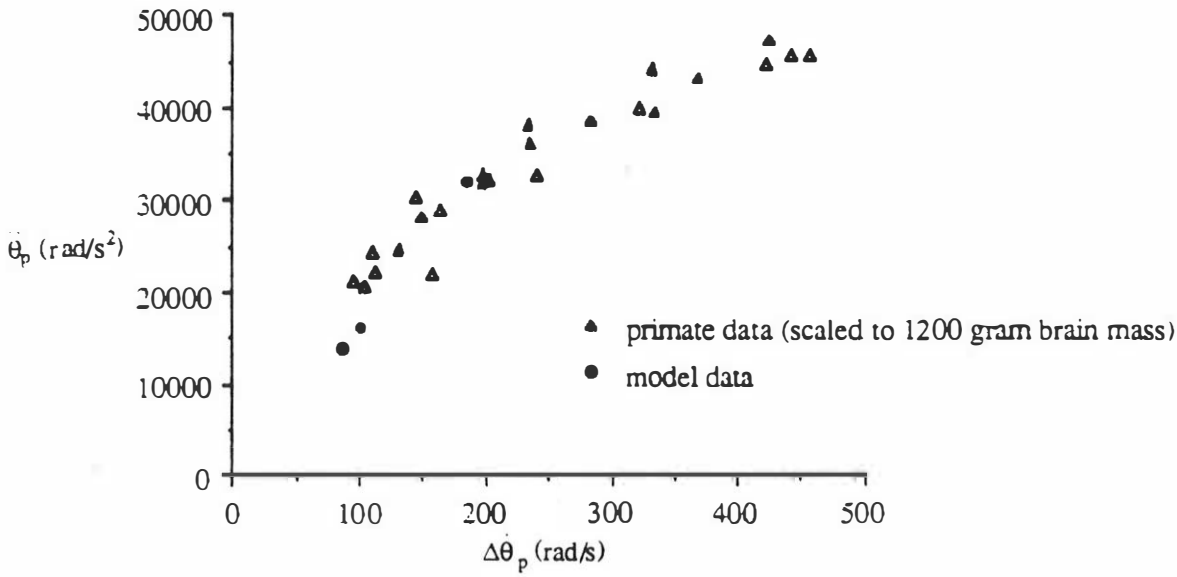


FIGURE 4

modeling study (scaled to 1200 gram brain mass), which are found both outside (15 psi set pressure, 25 psi set pressure) and within (35 psi set pressure) the region associated with acute subdural hematoma in the scaled primate data set.

The measured cortical deformation at these three loading levels using the sagittal section skull model are shown in Figures 5 and 6. Figure 5 portrays the temporal variation of cortical deformation at three different anatomic locations. It can be seen that the site of maximum strain is located in the frontal region (ε_{peak}=.98), suggesting that the rupture of parasagittal bridging veins is most likely to occur in this region. Such a hypothesis is supported from the University of Pennsylvania primate studies cited earlier [1], where the location of hematomas were frontally predominant.

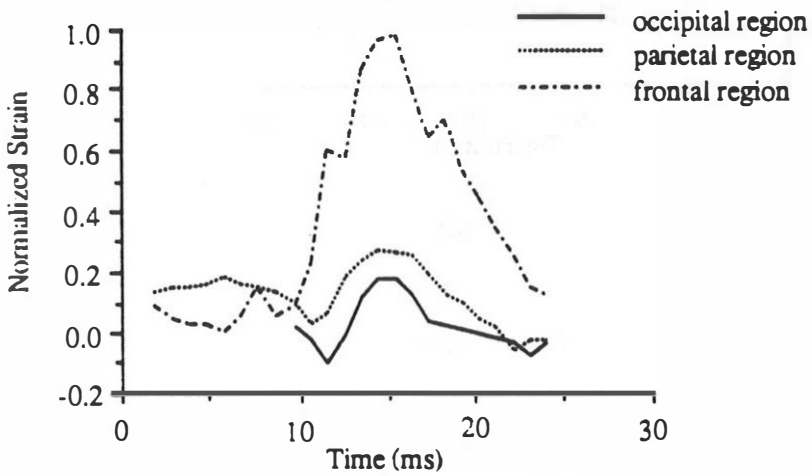
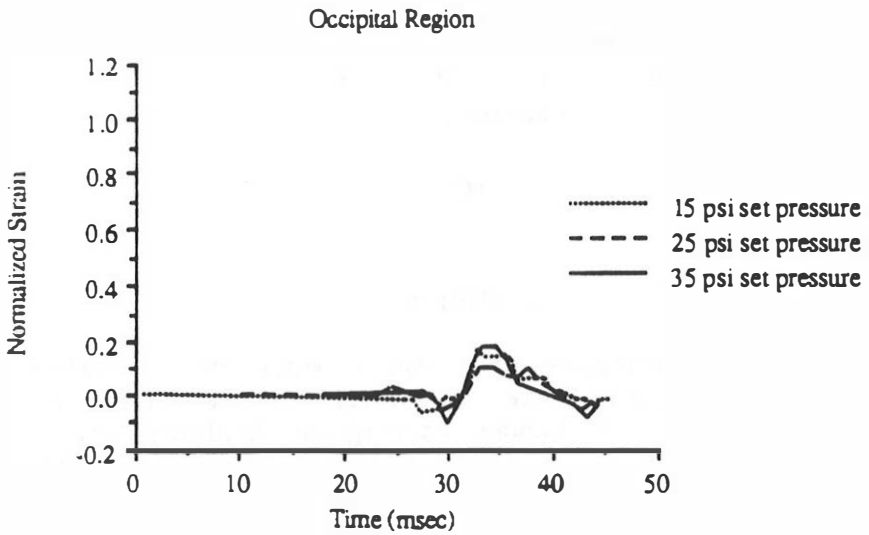
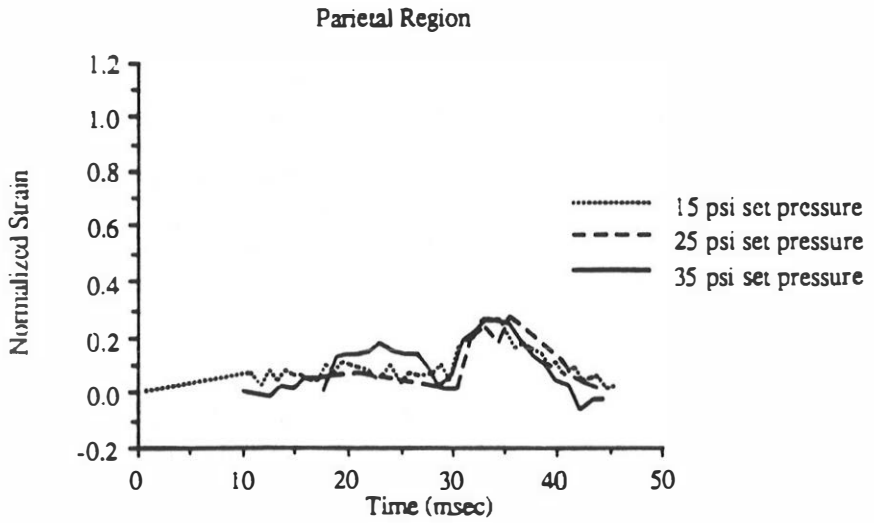


FIGURE 5

Figure 6A-C displays the stretch ratio of elements located in the occipital, parietal, and frontal regions for the three loading levels used in this study. Strain was maximum in the frontal region for all three loading levels, ranging from ε_{peak}=.33 to ε_{peak}=.98, and increased as the peak angular acceleration increased.



6A



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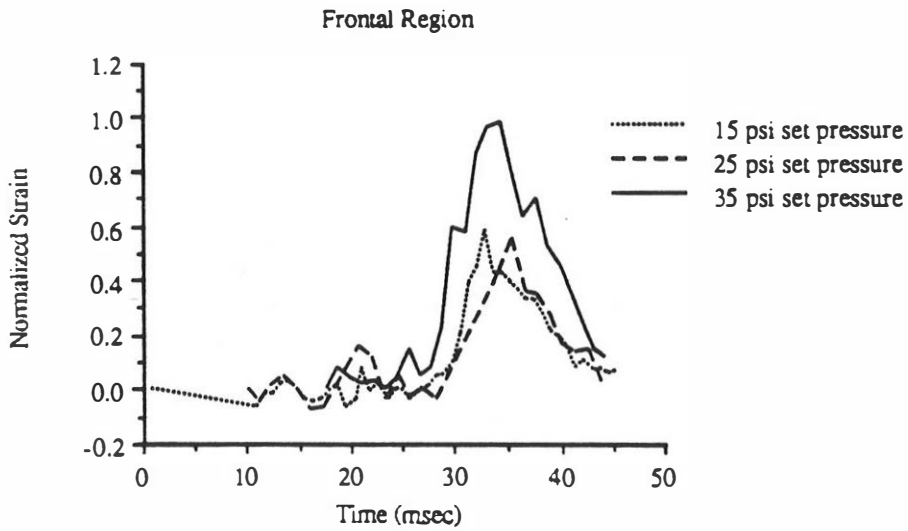

6C

FIGURE 6

It is informative to integrate the results from this modeling study with available information concerning the structural failure limit of parasagittal bridging veins. The response of human parasagittal bridging veins subjected to dynamic elongation similar to conditions depicted in figures 5 and 6 has been investigated by two groups which have formulated two distinct failure criteria. Lowenhielm measured the ultimate failure limit of isolated bridging veins over a range of strain rates (1 - 1000 s^{-1}) and found the failure limit to be dependent upon the strain rate [7] . Low strain rate (1 s^{-1}) elongation produced a much larger failure limit (ε_{ult}=.7-.9) when compared to high strain rate testing (ε=1000 s^{-1}, ε_{ult}=.2). Conversely, Haut and Lee measured the failure limit of human parasagittal bridging

veins and found the failure limit (ε_{ult}=.51-.55) to be relatively insensitive to the elongation rates used (.1 - 250 s^{-1}) [6].

The approximate strain rates and peak strains for the line elements located in the frontal region are shown in Table 2. Applying the Lowenhielm failure criteria using these values suggest that bridging vein disruption will occur at all loading levels, an outcome not predicted by scaling the results of the primate experiments. When the Haut and Lee failure criteria is used, it predicts that a hematoma will not occur for the lowest loading level, but will occur for the highest loading level. The intermediate loading level, however, appears to be capable of producing injury, an outcome which is not predicted by the scaled primate data.

TABLE 2

Set Pressure (psi)	Peak Strain	Strain Rate (s^{-1})
15	.33	176
25	.56	84
35	.98	155

These discrepancies between physical modeling results, scaled primate data, and bridging vein failure relationships emphasize the need for a comprehensive study encompassing physical modeling, analytical simulations, and isolated tissue testing to develop a more consistent understanding of the tolerance levels for ASDH in the human. Physical models currently being developed in our laboratory will be used to study the function of the tentorium cerebri, which may affect deformation in the occipital region, and to quantitatively study the relationship between simulated flow of surrogate brain tissue from the cranial cavity and cortical deformation.

Possibly the most critical issue, though, is to clarify the failure relationship for parasagittal bridging veins over a range of strain rates. In addition, more variables should be considered when testing vein specimens. Currently, no information is known regarding the failure criteria of bridging veins over a range of age groups. It has been shown that the incidence of subdural hematoma is much larger in older population groups, a statistic that could be due in part to a change in the mechanical properties of the bridging veins [12]. Furthermore, future mechanical testing of isolated bridging veins should include perfusion of the vein during testing. The inertial and viscous effects from the perfusate, combined with a peripheral resistance representing the cerebrovasculature, may affect the measured failure limit. Studies are currently in progress in our laboratory which will test perfused and non-perfused bridging veins from a number of age groups (neonate, pediatric, young adult, mature adult) in an attempt to determine the strain rate dependence of bridging vein failure across the population.

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