EFFECT OF CEREBRAL VASCUlations ON THE MECHANICAL RESPONSE OF BRAIN TISSUE: A PRELIMINARY STUDY

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1. INTRODUCTION

Traumatic brain injury (TBI) constitutes a significant portion of all injuries occurring as a result of automotive and sports related injuries. Over the years, many studies devoted to an increased understanding of clinical symptoms, pathological evidence and injury biomechanics for such injuries. However, the precise causal mechanism is not fully understood.

Typically, brain tissues are described as incompressible because of their high water content [1]. This description neglects the fact that brain tissues consist of a complex network of neurons and blood vessels interspersed within a matrix of supporting cells. Upon loading, the vascular structure may contribute to some of the load-bearing properties of the brain. Although there are a considerable number of studies on the mechanical properties of brain tissue and blood vessels [2-8], little information exists concerning the effect of cerebral vessels on brain tissue response when subjected to dynamic loading.

Computer modeling is becoming an important tool for the study of impact brain injury. A well-validated computer model can be used to study impact events that are too complex or costly to conduct in a laboratory setting. Such a model can provide an understanding of the basic brain injury mechanisms. Over the years, several brain injury models have been developed at Wayne State University (WSU) [9,10]. Despite of being the state-of-the-art, these models share a common problem in that a better understanding of the mechanical properties of brain tissues is needed.

In a study reported by Arbogast et al. [11], brain tissues were dissected from adult pigs and vibratory shear forces were applied through two parallel plates. Shear moduli were calculated for these tissues as a function of frequency. Although the design of this study was quite elegant, its results did not fully reflect the tethering effect of the blood vessels.

To compensate for the effect of vascular structures, shear moduli used for modeling the brain at WSU were considerably higher than those reported by Arbogast et al. [11].

Although the most recent WSU brain injury model consists of over 220,000 nodes, it still lacks the explicit modeling of the cerebral blood vessels. As an initial attempt to incorporate blood vessels into this model, simplified finite element models of sagittal sections of the brain with and without explicit modeling of the main cerebral vessels and branches were developed. An attempt was made to gain some insight into how the inclusion of vessel elements would modify the local tissue responses to impact. The stress and strain response of brain tissue were computed and then compared in order to assess the contribution of the major vessels in relation to the local response of the brain for a given set of loading conditions. The findings of this preliminary study may serve to advance our understanding of the underlying mechanism of focal injury to the brain and diffuse axonal injury (DAI), which involve significant damage to the axons and vasculature of the central nervous system (CNS).

2. METHOD

2.1. Model Description

In this study, two finite element models of a half-circular disk to approximate the cerebrum were developed to represent a parasagittal section of the human head. These 2D models had a diameter of 170 mm, similar to an average size of a human adult head. Model I consisted of the skull, dura, cerebrospinal fluid (CSF), pia mater and brain tissue as shown in Figure 1. In addition of all the features in Model I, two types of simplified cerebral blood vessels, parasagittal bridging vein and a major branch of anterior cerebral artery, were added to form Model II (Figure 2).

The parasagittal veins serve to drain the blood from superior regions of the cerebral cortex into the superior sagittal sinus. These ascending veins were formed by connecting a node on the cortical surface to a node on the inner surface of the dura, crossing the subarachnoid space. The purpose of modeling the bridging veins was to take into account the tethering effect of these vessels when there is a relative motion between the brain and the skull. To model the cerebral arteries, the main branches of anterior cerebral arteries were arranged along the radial direction of the brain. The anterior cerebral arteries originate from a bifurcation of the internal carotid artery, which supplies the medial surface of the brain [13]. These blood vessels (arteries and veins) were modeled as beam elements that can take tensile and bending loads. It is believed that pressurized vessels are capable of resisting bending load.

The skull was modeled as single layer rigid shell elements so that rotational loading could be applied through the center of the gravity of the head. The dural and pia mater were modeled with membrane elements covering the inner surface of the cranium and outer surface of the brain, respectively. The CSF layer was modeled as solid elements occupied in subarachnoid space while the ventricular system was not included.

2.2. Material Properties

Identifying proper brain tissue properties continues to be the greatest handicap in computational modeling. The earlier finite element models of the brain adopted linear elastic material constitutive laws [14-16]. In recent studies, linear viscoelastic material laws were used to model brain tissues [9,17-21]. In general, brain tissue is considered to be a highly damped material, thus, a linearly visco-elastic material model was chosen to model the brain tissue. The behavior of this material was characterized as visco-elastic in shear, while the compressive behavior was considered as elastic. The shear characteristics of viscoelastic behavior of the brain was expressed by:

\[
G(t) = G_s + \left(G_p - G_s\right) e^{-\beta t}
\]

where \(G_s\) is the short-term shear modulus, \(G_p\) is the long-term shear modulus, \(\beta\) is the decay constant and \(t\) is the duration.

Arbogast et al. [11] studied porcine brain tissue, and computed the average instantaneous shear modulus to be 1,036 Pa and 681 Pa for white matter and gray matter, respectively. In this study, the shear moduli used for both models were calculated from data.
reported by Arbogast and Margulies [12]. The bulk modulus of
the brain tissue was considered to be similar to that of the water
with a value of 2.10 GPa [4]. In this study, the bulk modulus of
the brain was assumed to be 2.19 GPa, the same value used during
the development of previous versions of WSUBIM [9,10].

In this model, an elastic modulus of 300 kPa was assumed for
all bridging veins [22,23]. Because only minimal information exists
on the material properties of cerebral blood vessels, it is assumed
that they possess the same mechanical properties as the bridging
veins. A cross sectional area of 0.22 mm² was chosen to model
both veins and arteries based on the geometric data [23,24].

Figure 1. Simplified sagittal slice of the head model.

Vessels
Brain
CSF

Figure 2. Simplified sagittal slice of the head model with
explicit modeling of the major anterior cerebral arteries on the
medial surface of the brain and parasagittal bridging veins at
the subarachnoid space.

Figure 3. Prescribed head angular acceleration time history.

2.3. Loading Condition

A rotational impact was applied to the rigid skull of both
models at a point that represents the center of the gravity of the
head. The angular acceleration time history used by Bandak and
Eppinger [19] was modified for this study. This pulse was formed
by two sinusoidal curves of different frequency and amplitude. The
acceleration component was a half-sine with a peak value of 5,000
rad/s² and a duration of 5 ms while the deceleration component was
a half-sine with a peak value of 2,500 rad/s² and a duration of 10
ms. Figure 3 shows the time history of the prescribed angular
acceleration loading condition. It had been postulated that a peak
angular acceleration of 4,500 rad/s² was the threshold for the
initiation of parasagittal bridging veins rupture [25]. Predicted
shear stress and shear strain were selected as response variables to
assess the model response to the given impact condition. Plane
strain was assumed for the models.

3. RESULTS

Figure 4 (a) shows the spatial distributions of the shears stress
predicted by Model I at 15 ms, the end of the simulation. Peak
stresses were observed at anterior and posterior corners. The
predicted maximum shear strain and shear stress from Model I
were 0.28 and 210 Pa, respectively. Figure 4 (b) illustrates the
contours of maximum shear stress predicted by Model II at the
same time. As depicted in this figure, a higher shear stress
concentration with a different distribution pattern was observed as
compared to the shear response from the Model I. The maximum
stress was localized at the region adjacent to the cerebral arteries
crossing the brain. High shear stress also occurred at the cortical
surface of the brain and in the subarachnoid space close to the
bridging veins. In contradiction to Model II, the shear stress

\[
\text{Max Shear Stress} \begin{bmatrix}
0 & 3.75e-08 & 7.5e-08 & 1.125e-07 & 1.5e-07 & 1.875e-07 & 2.25e-07 & 2.625e-07 & 3e-07
\end{bmatrix}
\]

Figure 4. Predicted maximum shear stress from the brain for (a)
Model I and (b) Model II (with blood vessels).
generated in the brain tissue was more uniformly distributed when vascular structures were absent in the model.

The maximum shear strain and shear stress predicted by Model II was about 0.79 and 560 Pa, significantly higher than those predicted by the model without blood vessels. Because blood vessels were stiffer than surrounding brain tissues, they reduced deformations that were perpendicular to the axis of the vessels. As a result, higher shear stresses were observed along the boundary of the vessels and distributed throughout the brain.

4. DISCUSSION AND CONCLUSIONS

Two simplified finite element brain models, with and without inclusion of blood vessels, were developed and angular acceleration was applied to both models to investigate the effect of vasculature on the shear response of the brain. In previous versions of the WSUBIM, shear moduli adopted for the models were higher than those measured directly using dissected brain tissues. The rationale for using a higher value was to account for the tethering effect of the vasculature. In this study, comparisons were made between models with and without simulations of blood vessels while the material property used to represent the brain was taken directly from the measured values without the effect of tethering. Results from this preliminary study reveal that the existence of vasculature greatly affects the shear stress pattern in the brain due to rotational acceleration. Because previous studies have suggested that DAI is due to a strain of axon [26-28], more studies are needed to determine if blood vessels indeed contribute to the occurrence of DAI in vivo.

The shear stress pattern predicted by the model was highly localized when prominent blood vessels were explicitly modeled. The induced local shear stress was concentrated near the region of the blood vessels and at the subarachnoid space on the brain surface. Based on this preliminary study, we hypothesize that the vascular architecture plays a role in the mechanical strength of brain tissues in vivo. Testing brain tissues in vitro in order to obtain material properties of the brain may not be able to determine the mechanical contribution of vasculature to the brain in vivo. If computing power is not a problem, future efforts in the head injury research should incorporate the vascular structure into the computer model to better represent the human brain.

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REFERENCES