DISCUSSION on "Rotation - Translation Duality in Head Trauma ? Perceptive and objective evidence"

David F. Meaney Department of Bioengineerng University of Pennsylvania

This paper presents a continuation of previous work using mechanical impedance measurements for the purpose of identifying and/or clarifying a possible new injury mechanism (brain/skull decoupling). The hypotheses, results, and conclusions from the work can be stated as follows:

- <u>Hypothesis:</u> Diffuse brain injuries occur from mechanical loading conditions distinctly different from those that cause focal brain injuries such as subdural hematoma and cerebral contusion. Decoupling, or separation, of the brain from the skull is responsible for creating intracerebral motions and deformations capable of tearing parasagittal bridging veins (subdural hematoma, SDH) and causing cerebral contusions (CC). In comparison, motion of the brain with the skull will affect tissue structures throughout the brain, thereby causing diffuse axonal injury (DAI).
- <u>Hypothesis:</u> Mechanical impedance measurements are instrumental in detecting the transition point where brain/skull decoupling begins to occur. A reduction in the apparent mass during mechanical impedance testing signifies a transition from oscillating the skull, soft tissues, and brain to oscillating only the skull and soft tissues.
- <u>Results:</u> Reported human volunteer tests indicate that a brain-skull decoupling occurs between frequencies of 100 to 150 hz. Above these frequencies, the apparent mass of the system drops and the skull and brain move relative to each other.

Human volunteers experienced brief periods of nausea and dizziness at low excitation frequencies (<100 hz), interpreted to be due to the motion of the brain from the excitation force. Above the frequencies of 300hz, however, no discomfort was felt. The authors attribute this to the decoupling of the brain from the forced skull motion.

<u>Conclusions</u>: Reported results suggest that the brain/skull decoupling phenomena should be strongly considered when formulating new brain injury tolerance criteria. Spectral analyses of the impacting force can yield a prediction of the brain/skull decoupling occurring from a prescribed impact force. Forces with significant frequency components above 150 hz may yield focal injuries consistent with the decoupling of the brain/skull structure. These injuries include subdural hematoma and cortical contusion. Conversely, forces containing spectral energies much below 150 hz may yield diffuse brain injuries due to the transmission of the mechanical energy in the impact force to the cerebral mass.

In all, the report continues previous lumped parameter modeling approaches to assess the utility of mechanical impedance measurements in either understanding brain injury mechanisms or identifying injury tolerance levels. A primary advantage of lumped parameter modeling approach is the ability to link modeling efforts to the measured mechanical impedance response of skull/brain structure. Additional levels of complexities (i.e., additional model components such as springs, dampers, masses) can be added to more closely reproduce experimental findings and understand the resonance and anti-resonance behavior measured experimentally. A primary disadvantage, though, of the discrete modeling approach is the inability to describe the continuum response of either the brain or skull. As such, results from such lumped parameter studies are primarily used to identify and model gross motions of the skull and brain, as well as determine structural damping characteristics of the skull, scalp and brain.

In this report, the authors recognize the advantages and challenges associated with lumped parameter modeling, and present a summary of findings intended to support the importance of brain-skull decoupling. The novel data presented from human volunteer studies appears to be consistent with previous results obtained in both cadavers and animal experiments. Prudenz and Sheldon (1946), one of the first investigative teams to quantitatively describe the relative skull-brain motion phenomena in the nonhuman primate, discovered the magnitude of relative motion depended upon direction of impact force. Lateral blows to the exposed skull created little movement of the sulci located along the superior brain surface, while sagittal impacts caused noticeable motion of the brain relative to the calvarium. Stalnaker (1978) and colleagues measured a distinct brain/skull decoupling that occurred both during and immediately following head impacts on cadavers repressurized to normal physiologic conditions. While it was found that quality of pressurization determined the extent of the brain/skull decoupling, Stalnaker noted that good pressurization did not eliminate the phenomenon entirely but only limited the amount of brain/skull decoupling. Taken together, these previous investigations and the current human volunteer studies suggest that brain decoupling does indeed occur to some extent for a forced vibration of the skull/brain structure. In turn, the phenomena may have some contribution to the incidence of subdural hematoma and cerebral contusion in specific cases. However, these results cannot be extended to those instances where brain injuries are due to very focal loading; an example would be cerebral contusions occurring from a depressed skull fracture.

The authors should be cautioned, also, when attempting to relate the results from this study to predict the occurrence of diffuse axonal injury as a consequence of a given impact force. The nature of the lumped parameter modeling approach prevents one from describing the distribution of stress and/or strain throughout the brain that is caused by a specific impact force. Diffuse axonal injury is comprised of axonal damage noted in specific regions of the brain and brainstem. As such, prediction of diffuse axonal injury from mechanical parameters will require a detailed distribution of the strain and/or stress behavior in these brain regions in response to a given impact force. While the data contained in this report presents some intriguing possibilities, it must be considered preliminary to suggest that the brain/skull decoupling phenomena can be directly linked to the presence or absence of diffuse axonal injury. Indeed, our experience with animal models of brain injury indicate other factors, such as

the directional loading dependence, may be equally important in predicting brain injury occurrence.

These directional loading factors have been studied in detail using physical surrogates, with results suggesting that intracranial deformation patterns were critical data necessary to separate loading conditions responsible for DAI and SDH (Gennarelli et al., 1987). Animal tests confirmed the directional dependence, with lateral noncentroidal or centroidal head rotations creating the intracerebral strains necessary for axonal injury throughout regions of the brain and brainstem (Margulies et al., 1990; Meaney et al., 1993). In comparison, subdural hematomas and contusions are more likely to occur from sagittal plane motion of the nonhuman primate head (Abel et al., 1978). Interestingly, we have documented cases in which tearing of parasagittal bridging veins has occurred in concert with diffuse axonal injury. In these cases, it appears enough brain/skull decoupling occurred to cause a subdural hematoma, and that sufficient strains were developed intracerebrally to create axonal injury patterns consistent with other DAI cases. Therefore, brain/skull decoupling should not be viewed as a distinct transition from one class of injuries to another class of injuries, but perhaps may only raise the likelihood of one form of injury over another. This approach also appears to be more appropriate upon viewing the distribution of brain injuries in moderate to severly head injured patients, where an admixture of injuries occurs in most patients.

Finally, the important human volunteer information contained in this report will add to a slowly expanding database of information relating direct or indirect mechanical loading of the human head to a measured physiologic, neurologic, or behavioral response. However, the difficulties presented in interpreting the human volunteer response may limit the applicability of these methods in definitively assessing injury mechanisms. By their nature, the volunteer tests do not examine injurious loading conditions. Rather, the minor changes noted in the volunteers are considered to be the initial signs of more serious injuries that would occur at higher loading conditions. It is important to note, though, that the phenomena observed in volunteer tests, such as the appearance of nausea and dizziness under low excitation frequencies, may not appear from a cerebral mass excitation but may be due only to an overstimulation of the vestibular system. To clearly document the brain regions affected by the low amplitude loading used in volunter studies, future investigations should consider more comprehensive techniques, such as the rapidly evolving functional magnetic reasonance imaging tools. Use of such tools will allow a more definitive interpretation of the deficits appearing in volunteers, and may lend credence to new injury mechanisms.

Taken together, this study offers interesting paths to pursue in order to examine the role of brain-skull decoupling. Possible paths include the following:

1. To conduct detailed set of experimental studies which will document the appearance of brain/skull displacement and response to a forced mechanical excitation for frequencies both below and above the transition point for brain/skull decoupling. Although this has been conducted for an in vitro model using a bovine brain, techniques are available (e.g., cranial window, lucite calvarium) to quantitatively describe the in vivo movement of the animal brain caused by an impact force.

2. To relate the magnitude of brain-skull displacement to the appearance of specific brain injuries such as tearing of parasagittal bridging veins and cerebral contusions. The amount of displacement needed to create these injuries can be defined using animal surrogates, and may be appropriately scaled to the human.

3. To further clarify the human response to the forced vibratory oscillation used in this study. Volunteer response data can be expanded to include new imaging techniques that will document functional deficits appearing from a given excitation input, as well as including a more complete evaluation of the subjects following testing.

4. To offer the results from the above experiments to experimental and computational biomechanicians interested in advancing new models for studying brain injury. Establishing detailed data on the brain-skull decoupling will provide vital information for modeling the boundary condition between the skull and brain surface in the next generation of physical models and finite element models. In turn, these models can be tested under a range of loading conditions not possible to pursue in human volunteer tests, with results being used to estimate the human tolerance for subdural hematoma, cerebral contusion, and diffuse brain injuries.

References

- Abel, J., T. Gennarelli and H. Segawa (1978). Incidence and Severity of Cerebral Concussion in the Rhesus Monkey Following Sagittal Plane Acceleration. <u>Proc. of the 22nd Stapp Car Crash Conf.</u> pp. 33-53
- Gennarelli, T. A., G. Spielman, T. Langfitt, P. Gildenburg, T. Harrington, J. Jane, L. Marshall, J. D. Miller and L. Pitts. (1982) Influence of the Type of Intracranial Lesion on Outcome From Severe Head Injuy. <u>J. Neurosurg.</u> 56: 26-32.
- Gennarelli, T., L. Thibault, et al. (1987). "Directional Dependence of Axonal Brain Injury Due to Centroidal and Non-Centroidal Acceleration." <u>Proc. of 31st</u> <u>Stapp Car Crash Conf. SAE:</u> 49-53.
- Gennarelli, T.A.;Thibault, L.E.; Adams, J.H.; Graham, D.I.; Thompson, C.J.; and Marcinin, R.P. (1982) Diffuse axonal injury and traumatic coma in the primate. <u>Ann. Neurol.</u> 12: 564-574.
- Margulies, S.S., Thibault, L.E., and Gennarelli, T.A. (1990) Physical model simulations of brain injury in the primate. <u>Journal of Biomechanics</u>, v. 23, no. 8, pp. 823-836.
- Meaney, D.F., Thibault, L.E., Smith, D., Ross, D.T., Gennarelli, T.A., (1993) Diffuse axonal injury in the miniature pig: Biomechanical development and injury threshold, <u>AMD v. 169</u>, pp. 169-175, 1993

Prudenz, R.,C. Sheldon, (1946) The lucite calvarium - a method for direct observation of the brain. II. Cranial trauma and brain movement. <u>J.</u> <u>Neurosurg</u> 3:, 487.

Stalnaker, R., Melvin, J., Nusholtz, G., Alem, N., Benson, J., (1977) Head Impact Response, SAE paper no. 770921