ROTATIONAL BRAIN INJURY TOLERANCE CRITERIA AS A FUNCTION OF VEHICLE CRASH PARAMETERS

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ABSTRACT

Diffuse brain injury is a form of severe brain injury which occurs primarily in vehicular accidents, and is responsible for 35% of the deaths in severely head injured patients. A coordinated series of animal experiments, physical and analytical simulations, and isolated tissue tests have been used to formulate a tolerance criterion for concussion and more severe forms of diffuse axonal injury in man, but the relationship between these tolerance criteria and measurable vehicle crash parameters has not been studied. In this report, the kinematics of the occupant in an idealized side impact are studied using the Crash Victim Simulator/Articulated Total Body (CVS/ATB) program. An emphasis was placed on relating peak coronal plane rotational acceleration and rotational velocity of the head to the change in velocity (ΔV_y) describing the crash. Results using a three ellipsoid model of the head-neck-upper torso (no head contact) indicated substantial changes in torso velocity (ΔV=36 mph) were necessary to attain the maximum non-injurious loading conditions measured in human volunteer tests. Higher crash velocities were needed to exceed the tolerance for concussion (DV=46 mph), and yet higher velocities for mild to severe DAI. These results suggest the importance of head contact to generate the inertial loading conditions to cause diffuse brain injuries in minor to moderate collisions. The conclusions regarding the importance of head contact in producing diffuse brain injuries is supported by recent epidemiological studies.

INTRODUCTION

Traumatic brain injury is a primary cause of death in victims of motor vehicle accidents (MVA), and accounts for 75,00-100,000 annual fatalities in the United States. Compounded by the 500,000 head injuries requiring hospitalization and estimated 1.5 million untreated injuries, traumatic brain injury in the United States is responsible for an estimated $25 billion in annual costs due to hospitalization, rehabilitation, and disability.

Diffuse brain injury is a form of severe brain injury which occurs primarily in vehicular accidents, and is responsible for 35% of the deaths in severely head injured patients. (Gennarelli and Thibault, 1982; Gennarelli et al., 1981). However, the complex head motions occurring in the automotive crash environment have presented challenges in establishing the human tolerance to various forms of diffuse brain injury. In light of these factors, an alternative approach of animal, physical model, and analytical simulations has been utilized to formulate tolerance criteria for concussion and various grades of DAI in the primate based on single plane rotational measures (Ommaya et al., 1967; Ommaya and Hirsch, 1971; Gennarelli et al., 1982; Thibault et al., 1982; Margulies and Thibault, 1989; Margulies et al., 1990; Margulies and Thibault, 1992). However,

the manner in which these proposed tolerance criteria relate to the automotive collision environment remains unclear.

To this end, the objective of this study was to establish the relationship between the severity of an automotive collision, as measured by the change in velocity (ΔV_y), and the tolerance to various grades of diffuse brain injury. In the first part of the investigation, the gross kinematics of the head-neck-upper torso structure during a side impact collision is studied using available techniques. As a simplification, no head contact is assumed to occur with interior structures. Parametric analysis reveals the sensitivity of the model output parameters (peak angular acceleration, peak change in angular velocity) to size and strength of the head-neck. Once analyzed, the model is exercised to determine the critical velocities needed to produce conditions capable of producing moderate to severe diffuse axonal injury, as well as milder forms of DAI, in the occupant.

METHODS

In this report, the kinematics of the head-neck structure was studied using a three ellipsoid representation of the head-neck-upper torso depicted in Figure 1. Size, mass, and mass moments of inertia for the ellipsoidal segments were determined using GEOBOD software, and were structured to describe the anthropomorphic dimensions across a broad range of male and female subjects. All calculations of the kinematics were performed using the Crash Victim Simulator/Articulated Total Body (CVS/ATB) program. A more detailed description of CVS/ATB can be found elsewhere (Obergefell et al., 1987; Bartz, 1972).

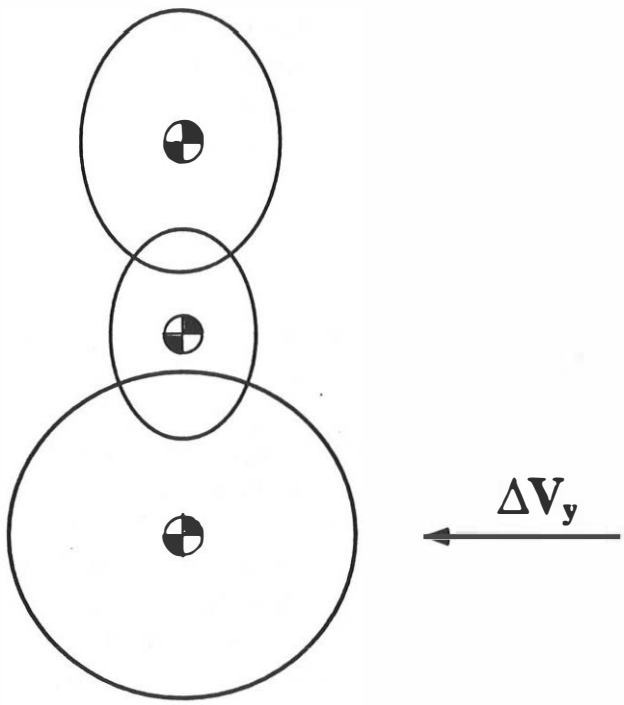

FIGURE 1 - Three ellipsoid model of the head, neck, and upper torso used tp estimate inertial loading conditions experienced by the head during a lateral change in velocity of the torso

Ellipsoids were connected with nonlinear springs representing the head pivot (K_{hp}) and the neck pivot (K_{np}), with viscous components (B_{hp}, B_{np}) to provide joint damping. Torque generated in the springs was related to the change in angle between the two consecutive segments according to the formula:

$$\tau = K_1(\theta_t - \theta_{t+1}) + K_2(\theta_t - \theta_{t+1})^2 + K_3(\theta_t - \theta_{t+1})^3 \qquad [1]$$

where the constants K_1, K_2, K_3 are derived from a Hybrid III neck description incorporated into GEOBOD. While the joint parameters for the human neck may vary (Deng, 1989; Nilson, 1992; Wismans and Spenny, 1984; Wismans et al., 1986), the values used in this study were chosen to be within the range used in previous studies. Potential energy stored in the joints and segments, combined with the kinetic energy of the system due to the rotational and linear motion, yields expressions that are part of the equations of motion derived using a Lagrangian formulation:

$$\frac{d}{dt}\frac{\partial T}{\partial \dot{\zeta}_i} - \frac{\partial T}{\partial \zeta_i} + \frac{\partial V}{\partial \zeta_i} + \frac{\partial D}{\partial \zeta_i} = F_i \qquad [2]$$

where T is the system kinetic energy, V is the potential energy, D is the energy dissipated in the joints, F_i is the external forces acting on the system, and ζ_i refers to the system variables (e.g. θ_{head}, θ_{neck}, θ_{torso}).

All segments were assumed to be aligned and initially at rest. Contact between segments during the simulation period was allowed, and modeled using a supplied segment-segment contact function. Moreover, each segment was allowed to move freely with the exception of the upper torso, which was modeled as being accelerated from rest to a final velocity (ΔV_y) within a fixed time period (50 milliseconds). No rotation of the upper torso was allowed during this deceleration phase, representing the constraint provided by the door panel during impact. Additionally, the deceleration was assumed to follow a sinusoidal pattern. For all simulations conducted, peak head angular acceleration, peak change in head angular velocity, and peak change in head angular displacement were recorded for different crash conditions (ΔV_y).

RESULTS

In accordance with the objectives of this study, simulations were conducted for a range of anthropomorphic sizes and collision velocities. An emphasis was placed on determining the threshold velocities needed to produce specific forms of rotational brain injury and the variations that may occur due to parameter estimation.

Simulation results at low velocities (Figure 2) indicate a rigid body rotation of the head-neck complex in the initial phase of loading (t < 24 milliseconds), followed by an articulating movement of the head-neck until maximum angular excursion occurs (t = 54 milliseconds, θ_{head}=100 degrees).

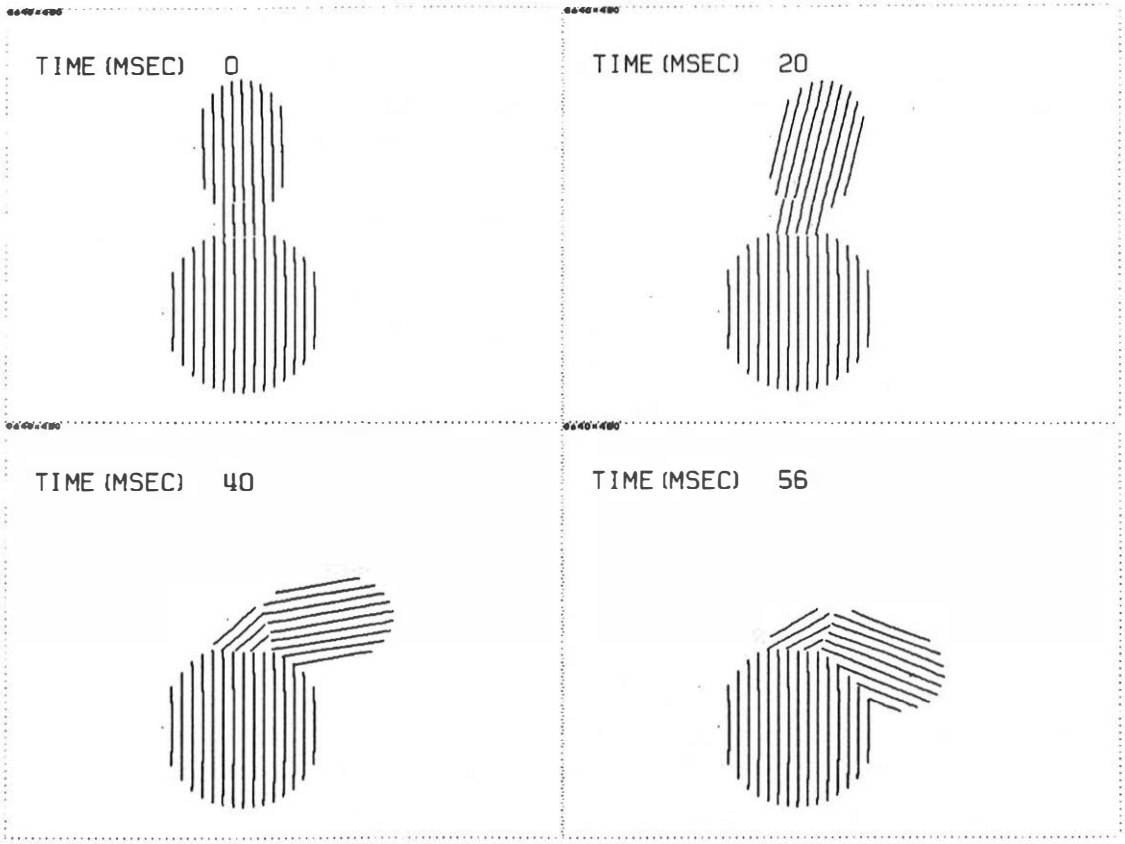

FIGURE 2 - Simulation response using 50th percentile male anthropomorphic size, and a lateral change in velocity of 20 mph

Head angular acceleration is biphasic, with the maximum deceleration occurring as the head contacts the upper torso (t=54 ms, Figure 3a). Maximum angular velocity is achieved during the articulating phase, decreasing to zero as the head is decelerated against the torso (Figure 3b).

Due to its temporal occurrence, maximum head angular acceleration was dependent upon the stiffness assigned to segment-segment contact in the simulations, with notable variations occurring when the contact function is changed for a given collision velocity (Figure 4a). However, the variation is not linearly related to the contact function

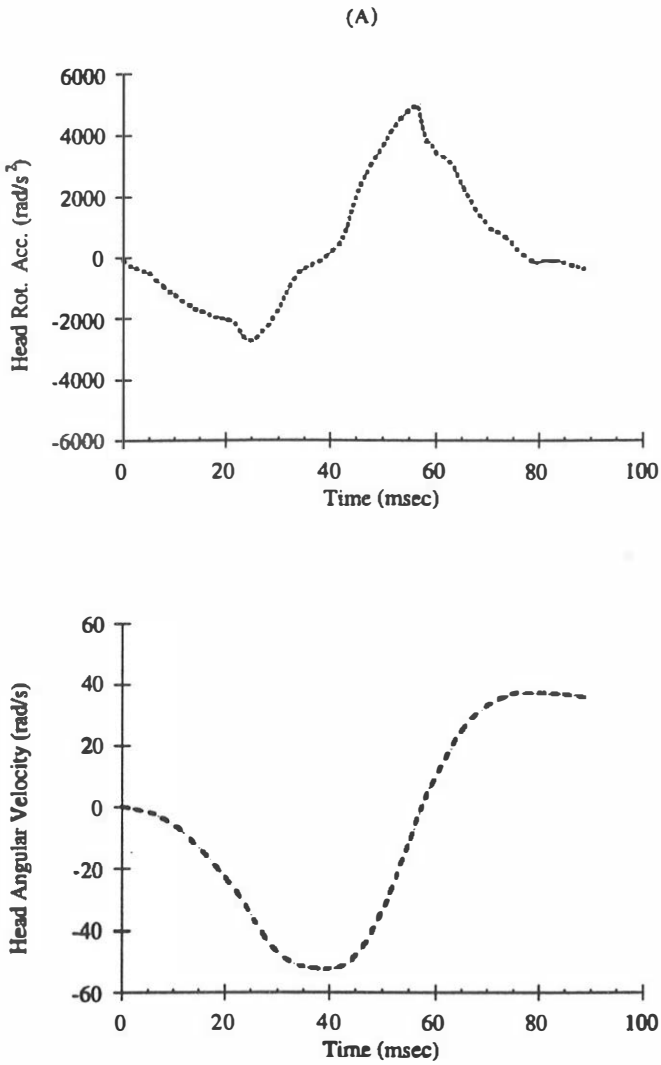

FIGURE 3 - Head rotational acceleration and rotational velocity over time; 50th percentile male dimensions.

description; rather, decreasing the stiffness of the segment interactions significantly (25% of original stiffness) decreased the maximum deceleration only slightly (70 % of original maximum rotational acceleration). In comparison, peak angular velocity varied much less for varying contact descriptions (Figure 4b), primarily because the peak angular velocity is achieved prior to any head contact with the upper torso segment. Since the most significant head accelerations and velocities were achieved for the stiffest segment-segment interactions, these stiff descriptors used in later simulations to generate a conservative estimate of tolerance threshold for various grades of DAI.

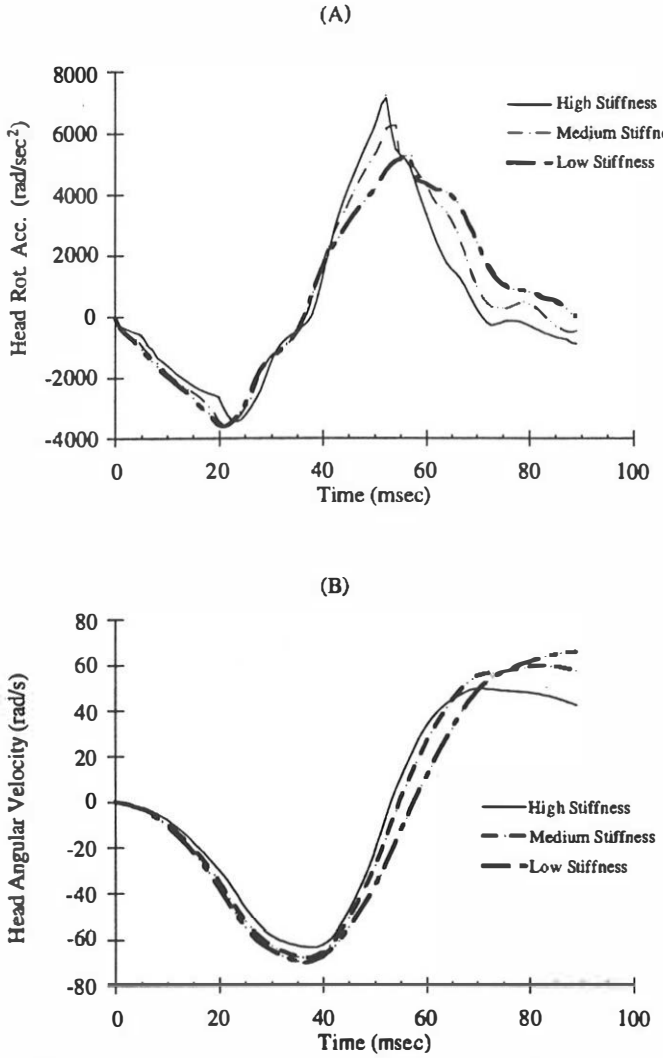

FIGURE 4 - Peak rotational acceleration is dependent upon the segment-segment stiffness characteristics. A conservative critical velocity threshold was developed using the parameters yielding the highest rotational accelerations at a given change in velocity (DV)

For a given stiffness description, Figure 5 indicates that the peak head angular acceleration (A) and peak change in angular velocity (B) increase nonlinearly with increasing collision velocity. Increases in collision velocity appear to influence the behavior of the nonlinear relationship for low velocities, and may be in part due to the increase in angular excursion at these levels. If the head and neck are assumed to angulate about a fixed point (i.e., C7/T1 junction) sinusoidally, it can be shown that the maximum head rotational acceleration ($\ddot{\theta}_{pk}$) is dependent upon the vehicle velocity (ΔV_y) and peak angulation (θ_{max}) and radius to the center of rotation (R) according to the formula:

$$\ddot{\theta}_{pk} = \left[\frac{\Delta V}{R}\right]^2 \frac{1}{\theta_{max}} \qquad [3]$$

As the velocity increases in the CVS/ATB simulations, the angular excursion reaches a maximum (θ_{max}=100 degrees) and the peak values of head rotational

acceleration for this simplified model approach the values calculated in the simulation (dashed line, Figure 5a). However, this simplified model does not include either an articulating head pivot or contact with the upper torso, factors that may account for the higher results obtained for the CVS/ATB calculations.

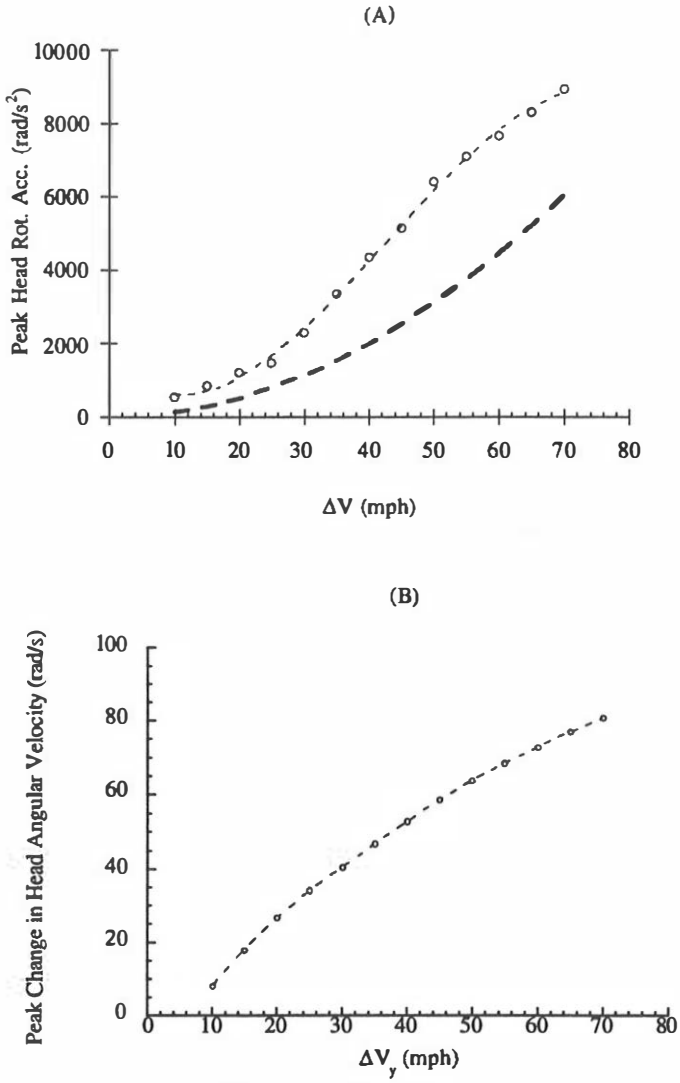

FIGURE 5 - Increasing collision velocities cause both the peak rotational acceleration and rotational velocities to increase in a nonlinear fashion. Estimates of peak rotational acceleration were larger than those calculated from a rigid body analysis (eq. 3), and is likely due to the articulation and segment-segment interaction present in the model

By conducting a number of test simulations across a broad range of occupant sizes and collision velocities, relationships were derived for the peak head rotational acceleration and peak head angular velocity as a function of crash severity (ΔV_y). We have reviewed previous human volunteer and animal studies, and have found estimates of the non-injurious maximum rotational velocity and rotational acceleration (Ewing et al, 1973; Pincemaille, et al., 1989). Shown on Figure 6 is a shaded region depicting the range of head loading conditions in the human volunteer studies conducted by Ewing (1973), and points corresponding to the maximum head rotational acceleration and rotational velocity (lateral flexion) encountered by boxers during a three round boxing match. In both studies, none of the volunteers experienced a loss of

consciousness. When compared to the CVS/ATB results, it was determined that a change in torso velocity of at least 32 mph was necessary to exceed the non-injurous loading levels measured in these human volunteer tests.

Using thresholds for cerebral concussion (Ommaya 1967; Ommaya and Hirsch, 1971), it was found that changes in torso velocity greater than 45 mph were necessary to produce concussive head-neck motions. More severe forms of diffuse axonal injury will require larger inertial loading conditions (Margulies et al., 1991), and will therefore have larger critical changes in velocity.

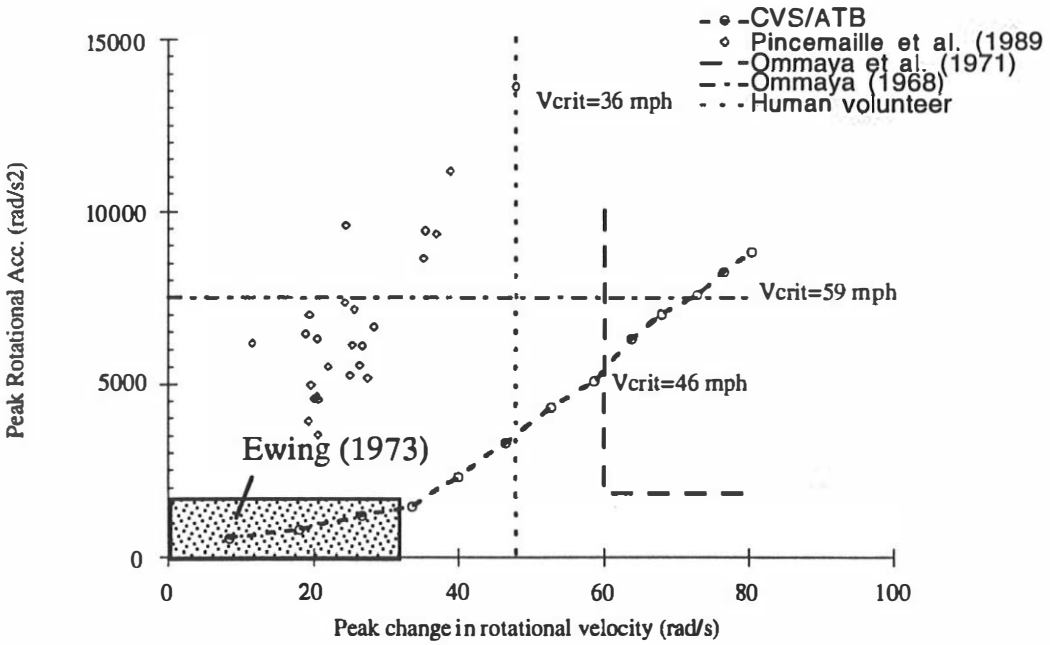

FIGURE 6 - Estimates of the critical collision velocities needed to exceed the proposed rotational brain injury tolerance criteria. Significant velocities (>32 mph) were necessary to exceed non-injurious loads experienced by human volunteers, while even higher velocities were necessary to exceed proposed criteria for concussion and diffuse axonal injury.

DISCUSSION

The objective of this investigation was to relate measurable crash parameters to the human tolerance for specific grades of diffuse axonal injury. Simulations of head-neck motions using CVS/ATB indicated the maximum head angular acceleration and peak change in angular velocity decreased as subject size increased, and increased in a nonlinear fashion with the change in torso velocity. Results for moderate to severe collision velocities were explained in part by a simplified model of the head-neck structure, but the simplified model could not fully simulate the contact with the torso or the articulation of the head-neck. Due to the limitations of the simplified model, results from the CVS/ATB simulations were used to formulate the threshold velocities to exceed non-injurious loads measured in human volunteer studies ($(\Delta V_y)_{crit}$ = 32 mph) and

to excced proposed tolerance levels for milder forms of DAI, such as concussion ($(\Delta V_y)_{crit}$ = 46, 59 mph).

Several factors deserve mention when interpreting these threshold levels. First, it was shown that the stiffness of the segment-segment interactions could influence the calculated peak head angular accelerations, but did little to influence the head angular velocity. More compliant segment contact functions caused peak head angular accelerations to decrease, although not directly proportional to the decrease in stiffness. Consequently, if the segment-segment interactions are considered too stiff, the estimated critical collision velocities for mild, moderate, and severe DAI may prove an underestimate of the actual velocities needed to produce these forms of brain injury in the occupant. Second, the ellipsoidal description for the head and neck pivot does not fully encompass the nonlinear behavior of the neck, but rather is a necessary simplification of the neck structure that may not exactly replicate the neck under the test conditions studied. However, many recent studies (Deng (1989), Wismans and Spenny (1983, 1984), Wismans et al. (1986, 1987), Mendis et al. (1989), Nilson (1992)), have shown that a two segment model of the head and neck is a reasonable tool to study the kinematics and kinetic response of the neck during dynamic loading. More advanced, multi-element models (Deng (1989), Nilson (1992), Merrill et al., (1984), Deng and Goldsmith (1987), Kabo and Goldsmith (1983)) have proved valuable to model anthropomorphic dummy necks and to investigate individual joint and muscle forces during impact/impulsive loading, but have yielded similar head kinematic and kinetic results to two segment models.

Perhaps the most notable factor to consider when interpreting these findings is the derivation of the injury tolerance levels for concussion and more severe forms of DAI. It was found that a critical torso velocity of 32 mph was needed to exceed the non-injurious head motions measured in human volunteer tests. Higher critical velocities (ΔV = 45mph) were calculated for concussion. However, the threshold level for concussion should be considered an approximate, since it is based on scaling results from animal experiments, and does not attempt to exactly predict the scaling relationship between the man and the nonhuman primate. Similarly, injury tolerance levels used in deriving the velocity threshold estimates for various grades of DAI have been based, to date, on a coordinated series of animal experiments, physical and analytical simulations, and isolated tissue experiments. Animal experiments have confirmed the relationship between both head kinematics and kinetics to severity and type of brain injury, while physical and analytical model simulations have related macroscopic head motion parameters used in these experiments to intracranial tissue deformation. In turn, spectrum of stretch induced electrophysiological and pathophysiological dysfunctions noted in isolated neural tissue experiments have provided the basis for relating intracranial deformations to injury (Galbraith 1993, Thibault et al. 1990, Saatman and Thibault 1991). Similar to concussion, these injury threshold levels are approximate and may need further investigation.

The estimated velocities outlined in this report to exceed proposed concussion and DAI tolerance levels underscores the significance of head impact in minor to moderate collisions (ΔV<25 mph). Specifically, head impact appears necessary in these collisions to produce inertial loading conditions sufficient for moderate to severe diffuse axonal injury. Indeed, it has been shown (Morris et al., 1992) that diffuse brain injuries appear frequently in very minor lateral

collisions (ΔV_{50}=18mph), an observation that is likely due to the high incidence of head impact with interior structures and side glass in these collisions. While these impacts may impose additional focal brain and skull injuries such as cerebral contusion, epidural hematoma, and skull fracture, it is probable that these impacts may cause head motions that exceed the critical thresholds for diffuse brain injuries.

Finally, it should be emphasized that this report has simulated only an idealized side impact collision, and has not attempted to investigate more complex off-axis vehicle impacts. The head motions induced during these off-axis vehicle impacts will involve coronal, sagittal, and horizontal plane motions, and have not been sufficiently explored experimentally to propose DAI tolerance levels such multi-planar motions. For example, while it is known that sagittal plane impulsive head motions can produce tearing of parasagittal bridging veins (Abel et al., (1978)), the incidence of other brain injuries occurring as a result of this head motion has not been fully determined. Critical in evaluating multiplanar head motions will be assessing the influence of intracranial partitioning membranes (e.g. tentorium cerebelli, falx cerebri) on the distribution of strains in the brain, and the manner in which these strains relate to the dysfunction of neural and vascular tissue. As multiplanar motions are investigated more rigorously in the laboratory, the more complex loading conditions in the automotive crash environment can be more fully understood.

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