
THE BIOMECHANICS OF TRAUMA

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CHAPTER 13

Biomechanics of Head Injury: Experimental Aspects

Ayub K. Ommaya

INTRODUCTION

The experimental study of head injury mechanisms is unique because it examines the effects of damage to a part of the body containing the organ that controls all other parts, as well as itself and the flow of information into and out of the body as a whole via its sensory receptors and motor effectors. The highest level of brain function constitutes the state of consciousness that may be defined as a state of self-reflective and intentional actions, one measure of which is the capacity to receive, record, and recall new information over time (memory). Experimental and theoretical studies of head injury use a combination of biomechanical and physiopathologic techniques not only to prevent (or minimize) the effects of obvious and visible damage, such as skull fractures and brain contusions, but, most importantly, to prevent the loss or disturbance of consciousness. In general, such disturbances of consciousness are produced either directly (and dynamically) via the mechanisms of reversible or irreversible concussive brain injury on neural elements or indirectly (and statically) via either the relatively slower volumetric distortions and pressure gradients induced by skull fracture, intracranial bleeding, and cerebral edema or by the onset of delayed tissue destruction secondary to vascular and metabolic factors (including hypoxia and ischemia), many of which are as yet unknown but potentially reversible. This latter aspect, important as it is for the final outcome, will not be reviewed in this chapter. The primacy of the traumatic disturbances of con-

sciousness are clearly recognized in most current classifications of head injury types and in most systems of grading head injury severity.

Any listing of the types (and lesions) of head injuries, such as is given in Table 1, shows only their anatomic loci and, while suggesting some of their physiopathologic effects, does not clearly indicate their effects on consciousness. It is important, therefore, to relate each injury type to its mechanism and to show how the functional effects follow, i.e., to connect the trauma input biomechanics to the physiopathologic response and its contribution to the outcome for the patient. The two most commonly used measurement tools for estimating head injury severity are the Glasgow Coma Scale and the Abbreviated Injury Scale. The first is used primarily by clinicians to record the intensity of coma and, by repeated use, can also show its duration. The second is a simple estimate of the threat to life caused by the injury at an early stage of the trauma and is used primarily by motor vehicle accident investigators. It is useful, therefore, to attempt to bridge the gap between these two disparate methods of grading head injury severity, which I will do later in this chapter.

TABLE 1. TYPES OF HEAD INJURY LESIONS

-
- I. Scalp
 - A. Bruise (leakage of blood from a vessel into adjacent tissue)
 - B. Abrasion (traumatic removal of some outer layers of scalp)
 - C. Laceration (cutting injury, tearing of scalp)
 - D. Avulsion (extreme laceration causing peeling of whole scalp)
 - II. Skull
 - A. Suture separation (diastasis, more often in younger skulls)
 - B. Indentation (e.g., ping-pong fracture, usually in younger skulls)
 - C. Linear fracture (may occur at points remote from impact location due to tensile stresses generated by sudden return of skull to its original shape after deformation)
 - D. Depressed fracture (may be accompanied by perforation, fragmentation, or comminution of skull)
 - E. Crushed skull (massive comminution, usually due to extreme static loading)
 - III. Extracerebral bleeding (focal or diffuse)
 - A. Subarachnoid hemorrhage
 - B. Epidural hematoma (with skull fracture in 90% of cases)
 - C. Subdural hematoma (with skull fracture in 50% of acute cases—usually due to torn bridging veins)
 - IV. Brain tissue damage (neural and/or vascular)
 - A. Brain concussive injuries (grades I through VI, with grade III including classic cerebral concussion and associated with increasing intensity and distribution of DAI—see Table 2)
 - B. Brain contusions (bruises of brain tissues located at any site, e.g., cortical, intracerebral, brain stem)
 - C. Intracerebral hematoma (visible intracerebral blood clots)
 - D. Cerebral laceration (visible tearing of brain tissues)
-

CURRENT CONCEPTS OF HEAD INJURY MECHANISMS

We have previously presented a paradigm for head injury mechanisms (Fig. 1).¹ This paradigm will be used to discuss the current concepts of how the various types of head injuries listed in Table 1 are produced and will also serve to summarize the current hypotheses seeking to integrate knowledge in this field. Modern concepts owe much to the work of Holbourn, Gurdjian, Symonds, and Sabina Strich, although two of the most fundamental facts about the nature of head injuries are very ancient. Thus, Al-Razi (Rhazes) and Abu Ibn Sina (Avicenna) on the basis of clinical observations alone were probably the first to recognize and distinguish between the causation and effects of skull fracture, brain wounds, and cerebral concussion in the tenth century.² Holbourn was probably the first physicist who, on the basis of physical and theoretical modeling, was able to present a rational and testable hypothesis for the mechanics of head injuries.³ Gurdjian, working closely with Lissner and Evans, pioneered the experimental study of skull fractures and brain injuries using linear accelerometers. Their work, later developed by others, led to the Wayne State Curve and via the work of Gadd to the current head injury criterion (HIC).⁴⁻⁷ Strich made the original observations in 1961 on diffuse axonal injury (DAI) in severely head-injured patients, a lesion she postulated to be a significant factor in the pathogenesis of symptoms and signs in the final outcome after head injuries of lesser severity.⁸ This prediction was supported by Oppenheimer, who in 1968 found histologic indicators of such tissue damage in patients with minor concussive head injuries who died of other causes.⁹ The classic review of theoretical, clinical, experimental, and pathologic data on concussive brain injuries by Sir Charles Symonds in 1962 was the first statement of hypothesis that has proved to be very fruitful in generating new experiments, namely that the term "cerebral concussion" or "concussive brain injuries":

should not be confined to cases in which there is immediate loss of consciousness with rapid and complete recovery, but should include the many cases in which the initial symptoms are the same but with subsequent long continued disturbances of consciousness often followed by residual symptoms . . . concussion in the above sense depends upon the diffuse injury to nerve cells and fibers sustained at the moment of the accident. The effects of this injury may or may not be reversible.¹⁰

Referring to our paradigm for head injury mechanisms (Fig. 1) and to the list of head injury types (Table 1), we can now elaborate the current concepts for such mechanisms. Applied loads to the head may be either static (occurring at time durations exceeding 200 msec) or dynamic (time durations less than 200 msec and usually in the range of 5 to 50 msec). Static loads causing head injury occur infrequently, e.g., when the jack of an automobile fails and allows the car to crush the head of the victim trapped underneath. A key observation in such patients is that in spite of severe

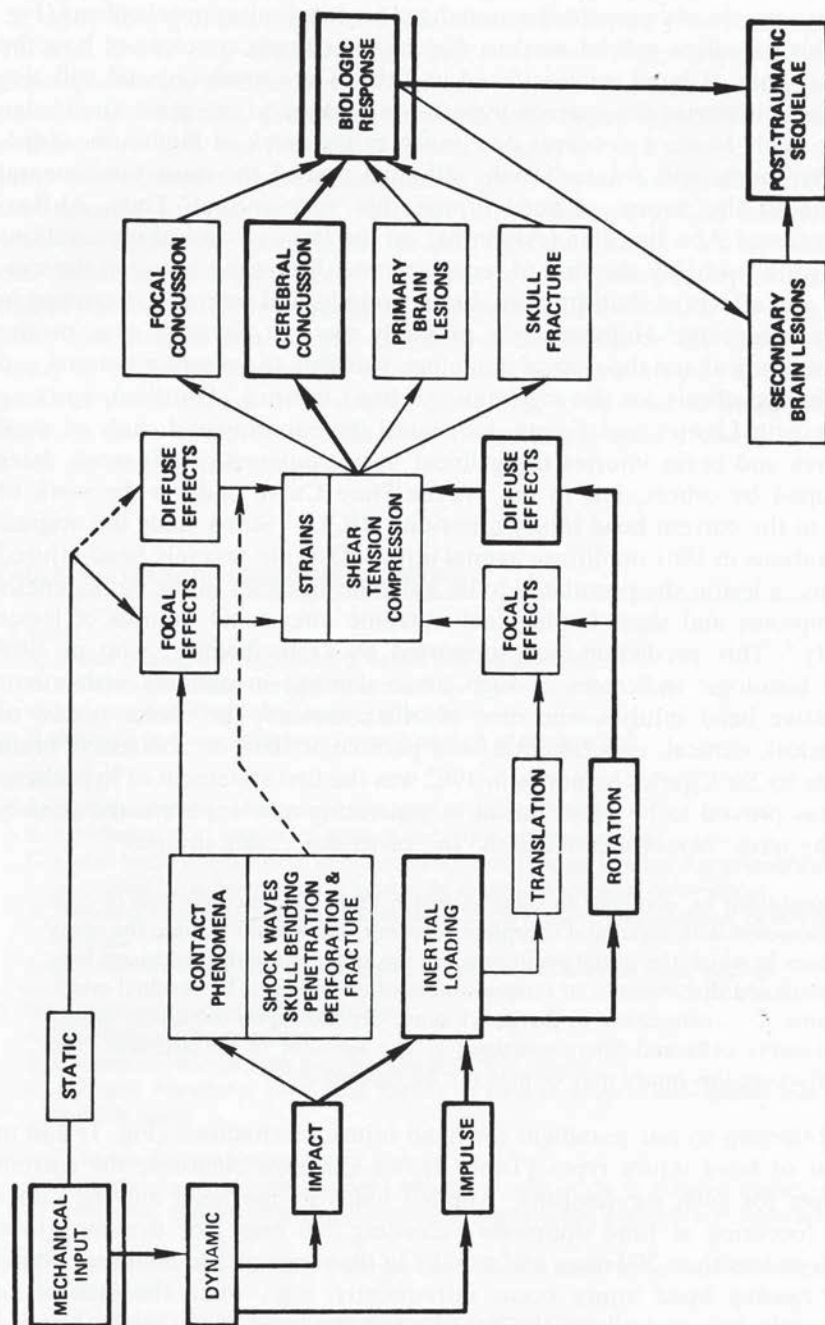


Figure 1. A paradigm for head injury biomechanics. (From Ommaya and Gennarelli.)

skull fractures and brain contusions with spinal fluid leakage through ears and nose, disturbance or loss of consciousness is seldom seen and the dissociation between the severe focal injuries and the absence of coma is striking.¹¹ This is strong supportive evidence for the concept that concussive brain injuries (Table 1, IVA) require some mechanism for which focal injuries to the brain and its adnexae (including skull fractures) are not necessary. Impacts to the head, e.g., in falls, automotive accidents, and assaults, are by definition dynamic events that include *two* components: the contact phenomena and inertial loading of the head and its contents. It should be noted that while contact phenomena can only be produced by direct impacts, inertial loading can be produced also by indirect impacts or impulsive loading of the head, for example, during the violent flexion-extension movements of the head after a rear end car collision. Of course inertial loading is always a highly significant component of all direct impacts when the head is free to move, and the rate of onset and/or duration of such loading pulses may be critical factors in the genesis of the types of brain injuries that result. Again with reference to our paradigm, all the items in categories 1, 2, and 3, and IVB, C, and D in Table 1, can be produced as focal injuries induced by contact phenomena alone. Although shock waves are listed as one of the contact phenomena, thus raising the possibility of diffuse brain injuries caused by this mechanism, it has been argued by Holbourn and others that the extremely short transit times for such waves (in microseconds) are exceeded by an order of magnitude by the impact durations and, therefore, should be ignored.

Figure 1 also indicates that inertial loading results in two types of head motion: translation and rotation. Holbourn's original hypothesis predicted that because of the incompressible nature of brain tissue, translation would be harmless and only rotation could initiate the diffuse tensile and shear strains required to produce the diffuse effects required to generate the concussive brain injuries.³ Our experimental test of this prediction refutes the notion of the innocuous nature of translation but does support the critical but not exclusive role of rotation in causing diffuse effects and cerebral concussive injuries, as will be described below.^{1,12}

Other concepts that have been advanced for the causation of concussive brain injuries include the idea of intracranial pressure gradients causing a primary and direct (or focal) effect on the brain stem and also by cervical cord stretch, an effect on the lower brain stem, or spinomedullary junction.^{4,13} While there is little doubt that a traumatic focal lesion in the brain stem, particularly in the rostral midbrain zone, would certainly cause severe and usually prolonged disturbances of consciousness, it is much more difficult to explain how such a mechanism as a *primary* event could explain at least two sets of clinical and pathologic data. First, there is the well-established one-way dissociation between what may be called the *amnesic* and the *paralytic* phenomena of concussive brain injuries. Thus, the amnesic or memory func-

tions appear much more fragile than the more robust sensorimotor control functions of the brain. In lesser degrees of head injury, it is possible to see amnesic disturbance without the victim falling down unconscious, although when such a knockout does occur, amnesia is always present. Second, when careful autopsy studies of severely head-injured patients are made, it is not possible to find primary brain stem damage *without* extensive associated damage elsewhere in the brain.¹⁴ It is for this reason that we introduced the *centripetal theory of cerebral concussion and concussive brain injuries*, which invokes the geometric structural and material properties of the cranium and its contents, to state that the diffuse effects of the rotational component of inertial loading are produced by a centripetal progression of strains from the outer surfaces to the core of the brain (coinciding with the midbrain and basal diencephalon). These strains are exaggerated and enhanced at all regions where abrupt changes in material and structural properties occur, e.g., in relation to dural partitions, bony protuberances (e.g., the sphenoid wing), and vascular tethers.¹ This hypothesis explains most of the available clinical pathologic and experimental data, including the phenomenon of one-way dissociation of amnesic and paralytic phenomena in concussive brain injuries. We have stated this hypothesis as follows: Concussive brain injuries include cerebral concussion and constitute a graded set of clinical syndromes following head injury wherein increasing severity of disturbance in level and content of consciousness is caused by mechanically induced strains affecting the brain in a centripetal sequence of disruptive effects on function and structure. These effects begin at the surface of the brain and extend inward to the diencephalic-mesencephalic core at the most severe coma-producing levels of trauma.

Our proposed classification of six grades of increasing severity of cerebral concussion thus produced is shown in Table 2, where a correlation with AIS level, pathologic data, and outcome at 1 month is also proposed. Because of the variable material properties, inhomogeneities, and anisotropy of the neural tissues, it is not proper to assume that strain distributions from the cortex through subcortical gray and white matter will necessarily correlate with visible tissue tears. Indeed, without a clear understanding of the precise relative fragility of axons, synaptic clefts, and neuronal bodies with which one could assess the three-dimensional distribution of all known structural lesions (including DAI), a definitive test of this hypothesis cannot be achieved. The classic case of cerebral concussion is thus understood as being in the middle of a range of concussive syndromes of lesser and greater severity. On the one hand may be found less severe cases where memory disturbances occur without loss of motor control, and consciousness is only partially impaired (grades I to II). In such cases, we suggest that significant strains did not reach the reticular activating system in the rostral brain stem. On the other hand are the more severe cases in coma with greater degrees of diffuse irreversible damage. When such diffuse impairment reaches a

TABLE 2. AIS—CONCUSSIVE BRAIN INJURY GRADING CORRELATION

AIS Level	Concussive Brain Injury Grade	Clinical Descriptions	Pathologic Description (13)(15a)	Outcome (1 Month)
1	I	Confusion without amnesia (ding, stunned)	Not known; CT scans usually normal; skull fractures and intracranial bleeding uncommon	Normal except for PCS* and occasional vascular complications
2	II	Amnesia without coma (type A, slow onset; type B, rapid onset)		
3	III	Coma < 6 hours (includes classic cerebral concussion, minor and moderate head injuries)		
4	IV	Coma 6–24 hours (severe head injuries)	Increasing intensity and distribution of diffuse axonal injuries and/or intracranial bleeding (e.g., acute subdural clots) and other visible brain lesions (or tissue tears); CT scans usually abnormal; skull fracture incidence 20–50%	Morbidity increasing to 35%+ and mortality to 50%+
5	V	Coma > 24 hours (severe head injuries)		
6	VI	Coma → death within 24 hours (fatal head injuries)		

*Postconcussive syndrome.

critical amount and therefore when the rostral brain stem shows visible structural damage, the worst subgroup of the grade V case may develop, and the patient never emerges from a vigilant coma state. This type of result is aptly described by the term "persistent vegetative state." Immediate death due to severe concussion may also occur (grade VI). Our hypothesis leads to three critical predictions: (1) When the level of trauma is severe enough to produce what is described as traumatic unconsciousness, the extent of simultaneous primary disruption of functions in the brain is more severe in cortical and subcortical structures, particularly in the critically vulnerable frontotemporal zones, than in the rostral brain stem. (2) It follows that because the mesencephalon appears to be the last of the vulnerable zones to be affected by trauma, primary damage to the rostral brain stem will not occur in isolation in the vast majority of head injuries that are associated with acceleration or deceleration trauma. A truly primary lesion of this part of the brain stem found at postmortem should be always in association with diffuse damage to the brain. If a patient with a lower grade of cerebral concussion dies from other causes, we predict that isolated primary rostral brain stem lesions will not be found. (3) Although confusion and disturbances of memory can occur without loss of consciousness, the reverse should never be seen, i.e., every case of head injury with a grade IV cerebral concussion must have an associated period of traumatic amnesia, the mesencephalon being less vulnerable than the temporal lobes and limbic system.

Traumatic unconsciousness develops after head injury of the usual accelerative type, when the centripetally directed, mechanically induced disconnection isolates the well-protected mesencephalic-diencephalic core from the overlying cerebral mantle. This disconnection is primarily functional, although in more severe cases it is reinforced by structural disconnection, e.g., in the white matter, as shown by the work of Strich and others.^{8,9,15-17} Thus a condition akin to the diaschisis of spinal shock is induced. Monakow's "permanent diaschisis" would, therefore, be analogous to structural reinforcement of the functional disconnections by such lesions as the Strich lesions.¹⁸ The possible mechanism I would propose for such a diaschisis is a modification of that described by Sherrington in 1910,¹⁹ when in his careful analysis of spinal shock he concluded that it was due to "the number and character of the descending nerve paths through which the lesion breaks." Sherrington concluded, "the condition of the spinal reflex arcs in spinal shock appears to resemble a general spinal fatigue rather than an inhibition . . . suggests a loosening of nexus between the links of the neuron chain comprising the arc; a defect of transmission at the synapse."¹⁹ That this conception of the mechanism of diaschisis is useful is supported also by Kempinsky's observations²⁰ that electrical activity in regions remote from a cerebral lesion will be depressed if such regions are in neuronal contact with the damaged zone. Our modification of Sherrington's concept

is that the irreversibility of the diaschisis is directly proportional to the extent of structural transection of axons and persisting abnormalities of membrane potential maintenance associated with synaptic disorganization. In this way, it becomes possible to conceive how trauma could induce the graded syndromes of cerebral concussion and also to consider spinal shock as being functionally similar to grade I-II concussion.²¹ As a result of an extensive series of elegant denervation experiments (posterior root and cord transections) on cat spinal cord and histologic observations on the synaptic organization of the motor neuron surface, Illis suggested that the depression in nerve cell activity after trauma was due to profound alteration of the synaptic zone.²² Thus, denervation of a critical number of boutons terminaux led not only to degeneration of those boutons but also to disorganization of the entire synaptic zone. With time, only the boutons with intact fibers reorganized and recreated a partial mosaic pattern of bouton distribution that was seen in the stage of functional reorganization (after 9 to 15 days). It is interesting to speculate whether such synaptic clearing at the motor neural surface after only partial denervation could be the structural analog of the diaschisis type of phenomenon seen in concussion of the brain or spinal cord. Even when axons are not transected, a sufficiently severe mechanical loading can produce a sufficiently prolonged functional paralysis so as to induce the connecting synaptic zone to undergo the above described disorganization of bouton patterns. In experiments done by Thibault and Gennarelli in our laboratory it has been shown that dynamic mechanical loading of the frog sciatic nerve can modulate the compound action potential between reversible decrements of amplitude to complete abolition of the waveform, which in some severe loadings resulted in a complete inability of the nerve to conduct even though it appeared to be histologically intact (at light microscope levels).

It is suggested, therefore, that in trying to understand neural disintegration after trauma it would be most profitable to study the organization of the synaptic zone and transmembrane potential states in order to elucidate the structural and functional correlates of the syndromes of cerebral concussion. Correlations of such changes with the resultant sequelae of neural trauma should then provide useful insight into more rational management.

CURRENT RESEARCH APPROACHES AND RESULTS

Our definitive experimental test of Holbourn's hypothesis for the relative contributions of the translational and rotational components of inertial loading clearly showed that *both* contribute to injurious effects on the brain.¹ These experiments utilized an inertial loading apparatus that eliminates contact phenomena, and they also clearly indicated that pure translation did not produce diffuse damage although the focal lesions seen were

dissimilar from those induced by the contact phenomena as seen in previous experiments.¹² In particular, some of the contusions seen under pure translation loads suggested a cavitation mechanism. It was only when rotation was added to translation that all the diffuse damage effects were noted. Thus, cerebral concussion, bilateral diffuse subdural, petechial, and subarachnoid hemorrhages were only seen after the rotational component was introduced (Figs. 2 and 3). The continuation of this work by my colleagues Gennarelli and Thibault in Philadelphia, in collaboration with the neuropathologists Adams and Graham in Glasgow, has resulted in further documentation of the significance of diffuse axonal injuries (DAI) in closed head injuries in Rhesus monkeys subjected to inertial loading with a rotational component.¹⁵ The Glasgow group has clearly demonstrated the similarity of these lesions to those seen in autopsy specimens from human victims of closed head injuries.¹⁶ In these studies, Gennarelli et al. have further defined the kinematics of such rotational effects and demonstrated that the extent of axonal injury, duration of coma, and outcome of this type of experimental head injury correlate better with coronal rather than sagittal plane rotations.¹⁵

In our experimental testing of Holbourn's hypothesis, we also were concerned about the role of contact phenomena. Holbourn implied that the response of the brain within the skull could be modeled by a single degree of freedom springmass system. Thus, short-duration impacts would produce injury proportional to the rotational velocity induced by the blow, while long-duration impacts would display a dependence on the head's rotational acceleration for injury. We were able to confirm the rotational velocity dependence of short-duration pulses producing cerebral concussion in both direct impact head injury and whiplash trauma in Rhesus monkeys. The main facet of Holbourn's hypothesis was, however, *not* proven. Thus, if head rotation were indeed the crucial brain injury mechanism for diffuse injuries, cerebral concussion should be produced at an identical threshold for rotational velocity of the head irrespective of how the head rotation was induced, directly or indirectly, and the local effects of impact should have no influence on the threshold for cerebral concussion. Our experimental data did not support this prediction and indeed showed that about twice the rotational velocity was required to produce cerebral concussion when the animal experienced indirect impact (whiplash).¹² This suggested a significant contribution to brain injury by one or more of three possible factors: a direct contribution of the contact phenomena, a summation of the rotational input with translational effects, or a significant difference in the rate of onset of the acceleration pulse under the two loading conditions.^{1,12}

One of the major problems in extrapolating experimental data from subhuman primate and other animal species to humans is the question of interspecies scaling. We have published one suggestion of how this may be done for rotational velocities and accelerations on the basis of experimental

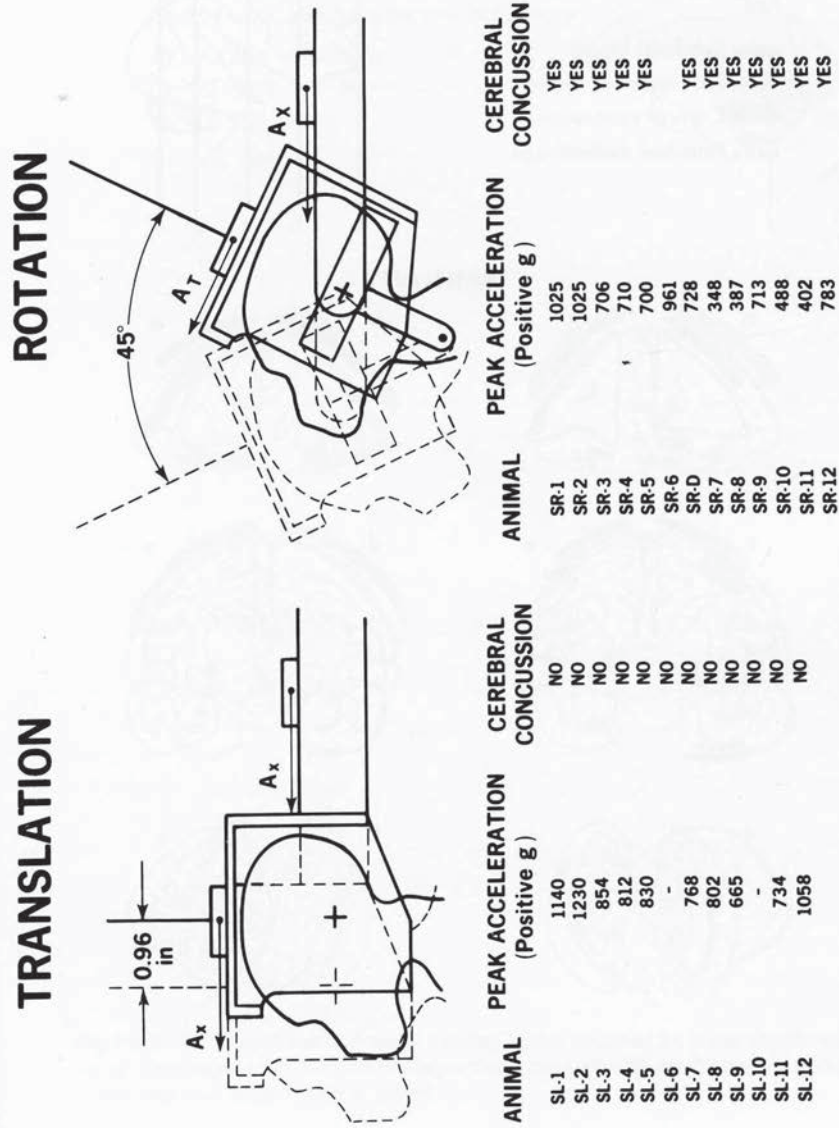


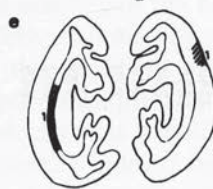
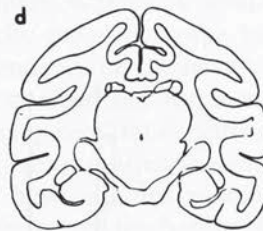
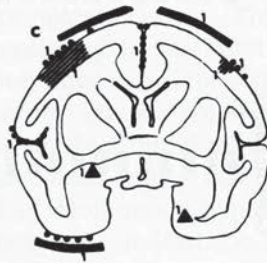
Figure 2. Diagrammatic display of subhuman primate kinematics and results of translational versus rotational (+ translation) acceleration at equivalent inputs. Note absence of concussion under condition of pure translation.

Key

- Subdural blood
- Subarachnoid blood
- Cortical contusion
- ▲▲▲▲ Petechial hemorrhage



TRANSLATED



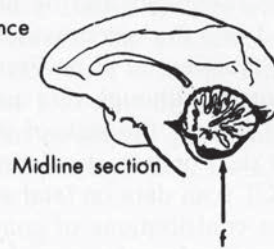
Number indicates frequency of occurrence

④ 90-100% of animals

③ 75-90%

② 50-75%

① Less than 50%



ROTATED

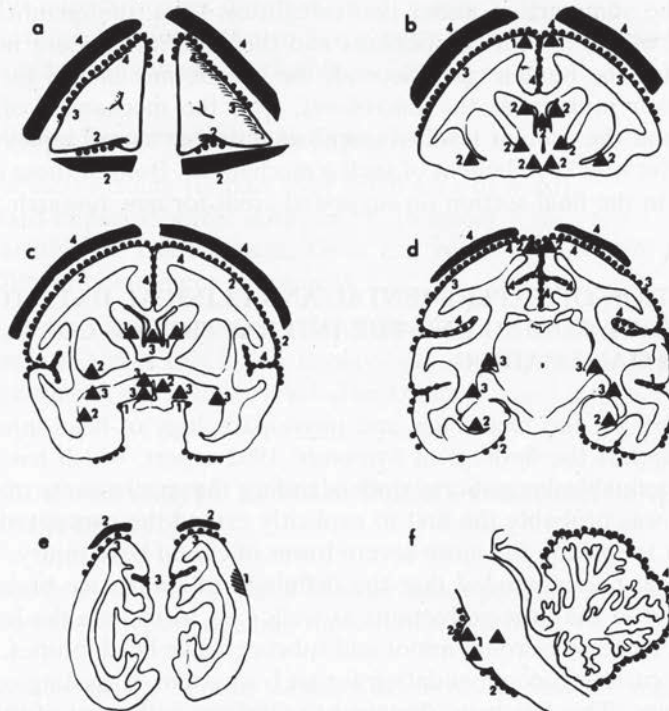


Figure 3. Cross-sections of squirrel monkey brains obtained 24 hours after head injury. Note legends in top part of diagram and markedly diffuse nature of lesions when rotational acceleration is added.

data in three subhuman primate species of gradually increasing brain mass.²³ In an attempt to bypass the problems of scaling, a number of workers have attempted to use cadavers as models for obtaining head injury biomechanical data. Injury responses have been measured in terms of intracranial pressure measurements and by noting the extent of extravasation of pigments perfused into the cerebrovascular system. The work of Nahum et al. in San Diego,²⁴ Tarriere et al. in France,²⁵ and Schmidt et al. in West Germany²⁶ is to be noted. Although very useful for the development of theoretical models of head injury, the lack of physiopathologic response cautions one to use cadaver data only in close correlation with animal and human accident data, where CT scan data on fatal as well as surviving cases are most useful.^{7,25,26,27}

The contributions of contact phenomena and inertial loading to head injury mechanisms has thus been identified separately on the basis of these observations in subhuman primate species. What is not clearly known at this time can be summarized under two categories. First, the quantitative biomechanics of the impact components and their interactions are not yet established for the human case. Second, the precise mechanism for the concussive brain injuries at the microlevel, i.e., the mechanism of cerebral concussion at the cellular level, is unknown although axonal injury is a good candidate for one key element of such a mechanism. Both of these issues are addressed in the final section on suggested areas for new research.

APPLICATION OF EXPERIMENTAL AND CLINICAL DATA TO HEAD INJURY CODING AND THE INTERACTION OF CONTACT AND INERTIAL LOADING

Current data on the mechanics and physiopathology of head injuries continue to support the findings of Symonds' 1962 report, which has provided us with a valuable heuristic in understanding the mechanisms of head injuries. He was probably the first to explicitly extend the concept of cerebral concussion to include the more severe forms of closed head injury.¹⁰ Indeed, we have also recommended that the definition of concussive brain injuries be extended in the other directions as well, i.e., to include the less severe (but much more numerous) minor and subconcussive head injuries.¹ Table 2 shows our current recommendation for such a system of grading concussive brain injuries. This has been designed to conform with most of the current clinical and neuropathologic data, as well as to correspond numerically with the AIS scale levels. The pioneering neuropsychologic outcome studies of Gronwall, Wrightson, and others have shown very clearly that the postconcussive sequelae of even the most minor head injuries can be of major significance in terms of ability to return to work and competence in previous occupations.^{28,29} These data fit very well with our classification of concussive brain injuries (Table 2) as well as with the neuropathologic observations of Oppenheimer and others on neuropathologic lesions present in mild to mod-

erate head injuries.^{9,30} More recent work confirming and extending the observations on the disabilities of the victims of minor head injury has been reported by Rimel and her colleagues at Charlottesville.³¹

The correlation of lesion types with the mechanics in head injuries is an ongoing task. Thus, the lesions shown in Table 1 have for the most part been well described in qualitative terms. Review of the available data on so-called DAI (diffuse axonal injuries) and non-DAI cases of fatal head injuries was carried out in order to propose the following hypothesis, which seeks to explain how the relative contributions of contact phenomena and inertial loading components favor or reduce the production of DAI.

Conditions Favoring DAI Type of Case

1. Distributed loading (impact over large area of head)
2. Soft impact (longer duration, > 10 msec)
3. Partition of kinematics to favor more rotation and less translation
4. Contact phenomena negligible

Conditions Favoring Non-DAI Type of Case

1. Focused loading (impact over small area of head)
2. Hard impact (shorter duration, < 10 msec)
3. Partition of kinematics to favor less rotation and more translation
4. Contact phenomena prominent

It should be emphasized that DAI cases form a minority of fatal head injury cases, and thus this lesion is only one of the possible array of structural alterations at the cellular level after trauma.

HEAD INJURY SEVERITY LEVELS, INJURY CRITERIA, AND TOLERANCE LEVELS

The European Economic Council Biomechanics program has recently produced a consensus report defining these terms as follows:

Injury Level or Injury Severity Level. This term denotes the magnitude of changes in terms of physiologic changes and/or structural failure that occurs in a living body as a consequence of mechanical violence. The AIS scale is widely used for this purpose, but other scales have also been proposed.

Injury Criterion, Injury Criteria. This term denotes a physical parameter that correlates well with the injury severity of the body region under consideration. Currently, the HIC is the accepted criterion for head injury, but the precision of its correlation is being questioned.⁷ This term is often used in other ways, thus causing much confusion in the literature.

Tolerance Level. This term denotes the magnitude of loading of the living body or body part that produces a specific type of injury and injury severity level. When used, this term must be specified by defining the following aspects: the physical parameter expressing the magnitude of loading, the type of living body (animal or human, sex and age), the type of body part, what kind of injury, and what injury severity level is being considered. Extrapolations of tolerance levels that do not follow these procedures are not recommended.

By inference, an injury criterion may represent a legal specification and as such may be used to form part of a standard that protective devices or crash environments must satisfy in order to permit their recommendation and distribution to the general public. However, in such cases, the standard or criterion must not be confused with the tolerance level for the reasons given above. Moreover, it is almost inevitable that the specific prescription required for such a standard requires the use of an arbitrary mechanical system, having only a distant and partial correlation with its human counterpart. Such test systems properly permit only comparative evaluation of the relative merits of injury-mitigating components, e.g., helmets, but do not permit their use to duplicate a degree of trauma that would be experienced by a human under corresponding circumstances—nor should they be used to predict human tolerance levels. The following discussion on head injury criteria is based on an earlier report by Goldsmith and Ommaya.³²

Injury Criteria for the Head

As stated, the terms “injury criteria” and “tolerance levels” should not be confused, and the distinction made in this report as suggested by the EEC Biomechanics program is recommended. Clearly, a distinction should be made for the skull and brain and possibly also for the vascular components. The stress and strain fracture characteristics of the skull can be quantified by a mean value and a standard deviation, which may be only slightly smaller than the 50 percentile magnitude but which is preferable to the specification of a breaking load or energy. On the other hand, brain failure cannot be so conveniently delineated because (1) physiologic dysfunction, including death, can occur at levels well below that producing mechanical disruption of neural tissues, and (2) the tissues comprising the brain and blood vessels are so complex and inhomogeneous that neither functional nor structural failure limits for either the entire system or for specified regions have been adequately established. In consequence, an injury level for the brain is normally specified in terms of the magnitude or history of some mechanical parameter considered to be a major indicator of cerebral trauma. Peak and average linear and angular accelerations, their duration and rate of onset, intracranial pressure, volume changes of the skull, and force and energy applied to the cranium have been utilized for this purpose, but it is not yet conclusively proven that any of these parameters are optimal for trauma

correlation. Whether kinematic parameters can be employed at all for this purpose has been questioned, but a recent interesting paper by Ono et al. concludes that it is possible to model head injury assessments from head motions derived from animal and cadaver data.³³ Current practice divides the brain injury generation mechanisms as those resulting from linear or angular motion, respectively.

Linear Acceleration Limits

The first of these demarcations is the Wayne State Tolerance Curve (WSTC), in which it is claimed that the dividing line represents the onset of concussion. This tolerance curve is based on the hypothesis that the dominant head injury mechanism is linear acceleration. Its initial enunciation was based on six experiments on embalmed cadavers striking rigid surfaces at the forehead in the A/P direction in the duration range from 1 to 6 msec. The results were correlated with concussive effects generated in animals and were later supplemented by additional experiments on primates and cadavers and employment of long-duration acceleration tolerance information from human volunteers.

Within a few years, a determined effort had been made to represent the WSTC in analytic form. The resulting expression for a severity index (SI) is:

$$SI = \int [a(t)]^{2.5} dt \quad (1)$$

This is a straight line on a logarithmic plot and did not quite match the curved transposition of the WSTC, but the difference was ignored. A tolerance level of $SI \leq 1000$ was stipulated as acceptable.

Serious objections to the use of this criterion were raised on the basis of the arbitrary value of the exponent and inconsistencies due to failure to distinguish between the approximation to tolerance data, the scaling of severity, and the definition of effective acceleration. It was suggested that the latter term might be replaced by a time-averaged, weighted acceleration, although such a representation was still inadequate in view of insufficient biomechanical validation data and lack of a specific severity designation. However, NHTSA mandated the use of a modified version of this idea, expressed in the form of the head injury criterion (HIC):

$$HIC = \text{Max} \left[\frac{1}{(t_2 - t_1)} \int_{t_1}^{t_2} a dt \right]^{2.5} (t_2 - t_1) \quad (2)$$

where the times t_2 and t_1 are two arbitrary instances of the pulse history chosen so as to obtain the supremum (maximum) of the function. Equation (2) was supposedly an improvement over the SI by concentration on the most dangerous portion of the history, which was bound to take into account the rate of load application. The legal limit of 1000 was placed on the permissible HIC value, although diverse opinions have been voiced that this

magnitude was either too high or too low relative to sustaining intolerable levels of injury. The HIC representation has also generated considerable controversy between its detractors and advocates. These arguments pro and con can be found in the recent consensus workshop proceedings and in the paper by Goldsmith and Ommaya.^{7,32} One of the major drawbacks of the HIC is that it is a go-no go type of criterion; what is needed is a continuous scale.

Angular Motion Limits

There is a substantial difference between the mechanical processes involved in direct head impact and sudden torso motion inducing whiplash type of head motions, although both cause linear and angular acceleration of the head. Head impact requires consideration of both potential skull fracture and trauma due to load transmission to the brain, whereas whiplash need not address skull fracture but will be additionally concerned with neck response.

Extensive experiments testing the rotational or angular motion hypothesis involving subhuman primates were done not only to assess the effects of restraining collars on concussion but also to determine dosage limits in these animals that could be extrapolated to man.^{1,12} Although both direct head impact and torso acceleration (whiplash) tests were performed, quantification and, particularly, scaling of the contact phenomenon between species was not considered to be sufficiently reliable, so that only whiplash experiments were scaled and extrapolated to the human case.²³ Damaging rotational velocities and accelerations for the brain were related by the assumption of a linear, elastic undamped single-degree-of-freedom system. Concussion was induced in the squirrel monkey at about 1200 to 1600 rad/second, in the Rhesus monkey at 300 to 600 rad/second, and in the chimpanzee at about 70 to 90 rad/second, as determined from physiologic responses and high-speed camera measurements of the motion. With the established brain frequency value for the Rhesus monkey (from calvarium measurements) of 5 to 10 Hz and an observed value of 4 to 5 Hz in a human determined from fluorocinematography of radiopaque clips on the brain surface, it is reasonable to delineate the natural frequency of both squirrel monkey and chimpanzee in the 5 to 10 Hz range. The value for the former was chosen as 10 and the latter as 5 from size consideration. The threshold for cerebral concussion in terms of head angular accelerations for the squirrel monkey, Rhesus monkey, and chimpanzee were then calculated as 20 krad/second,² 10 krad/second,² and 2.2 krad/second,² respectively. Employment of the scaling relation (inversely proportional to the two-thirds power of the brain mass) and the above data results in a rotational acceleration and velocity tolerance for man of 1.8 krad/second² and 20 to 30 rad/second, respectively; these limits are plotted in Figure 4. It should be reemphasized that this information shown as a band is considered to be reliable for the Rhesus, sketchy for the chimpanzee, and completely speculative for man.¹² It is expected, therefore, that when data from other sources and in particu-

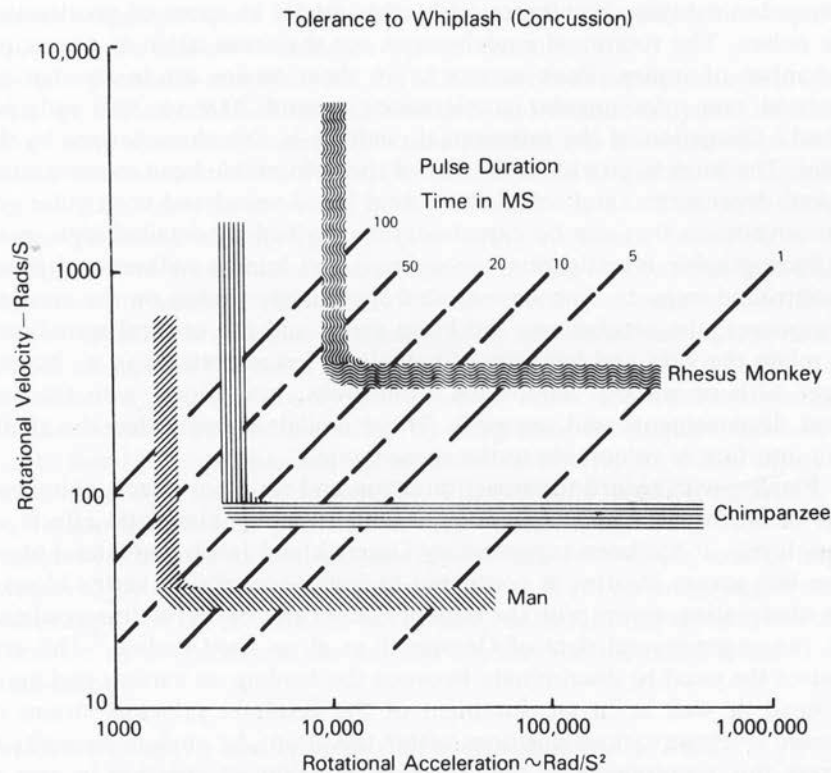


Figure 4. Theoretical scaling of rotational velocity and acceleration for 50 percent probability of onset of cerebral concussion from subhuman primates to humans.

lar from accident reconstructions in humans become available, these approximate bands of concussion thresholds for rotational motions will have to be revised.

Angular head acceleration has been proposed by Lowenheim as the cause of gliding contusions resulting from excessive strains in cerebral blood vessels.³⁴ The limiting dosage that will initiate such contusions can be quantified as a maximum angular acceleration of $4.5 \text{ krad/second}^2$ and an angular velocity change of 70 rad/second .³⁴

A detailed consideration of the kinematics of head injury including the relevant biomechanics of translational and rotational acceleration responses has recently been published in collaboration with Advani.³⁵ The translational and rotational models described in this article provide an engineering link for quantifying critical head injury parameters. For the translational model these parameters are (1) the skull tangential stresses, (2) brain displacements and contrecoup pressures, and (3) brain shear strains at the surface and midbrain region. This model primarily describes inertial blood volume, cerebrospinal fluid, brain volume, and associated pressures. Con-

trecoup brain injury is interpreted by this model in terms of acceleration time pulses. The rotational model singles out the shear strain as a principal mechanism of injury. Peak surface brain shear strains are in the damage threshold range for angular accelerations around 2000 to 3000 rads per second.² Distortion of the intracranial contents is also characterized by the model. The models provide estimates of the deformable head motions, such as skull depressions, and selected cerebral blood vessel and ventricular system movements that can be experimentally verified by detailed high-speed cinefluorographic investigations on animals and human cadavers subjected to controlled impacts. Subsequent neuropathologic studies on the cerebral hemispheres, the cerebellum, the brain stem, and the cervical spinal cord can relate the sites and intensity of pathologic lesion patterns (e.g., hemorrhages with or without hemostasis, contusions, lacerations, with the predicted displacements and stresses). These models indicate that the skull-brain interface is vulnerable to the shear mode.

Finally, with regard to impact direction and resultant effects of interactions between the contact phenomena and resultant kinematic effects on injury levels, it has been suggested by Grecivik and Jacob that lateral blows cause less severe injuries as compared to fronto-occipital or vertex blows.¹⁷ This observation agrees with the data of Ono et al.³³ but is not in accordance with the experimental data of Gennarelli et al. as cited earlier.¹⁵ This emphasizes the need to discriminate between the loading on various regions of the head as well as in measurement of the resultant principal strains or pressure levels at various positions within the brain. An obvious necessity to address this requirement is the inclusion of a contact criterion in case of impacts that can take into account local variations in skull architecture and curvature.

A SUGGESTION FOR THE USE OF TWO HEAD INJURY CRITERIA

Starting with the Wayne State Tolerance Curve, a variety of empirical equations and lumped parameter models, such as the Gadd Severity Index, the Vienna Institute Index, the Revised Brain Model, the Effective Displacement Index, and the Head Injury Criteria, have tried to fit the Wayne State Curve. Experimental and/or analytic models of the head, such as the Michigan Strain Model, and several continuum and finite element models of the head have been suggested. Of these, the Mean Strain Criterion (MSC) would appear to lend itself well to use as a continuous criterion to address the contact and translation components of the impact. The reasons for this are as follows: of the simple analytic models available, only the MSC obtained its parameters directly from experimental efforts. All the others chose parameters that would make them best fit the Wayne State Curve. Moreover, additional experimental studies have allowed the MSC origina-

tors to propose outputs of the model associated with other levels of injury, i.e., to enable a more continuous type of criterion.³⁶

It is generally accepted that the MSC and other similar criteria are most valid for the direct head impact situation and include only the translation component of inertial loading. Because rotation has been shown to be crucial for the concussive brain injuries (Tables 1 and 2) and for the rupture of the bridging veins between the skull and brain, the MSC *alone* is not adequate. The data identifying and quantifying rotational injury have been largely derived from animal experimentation and transformed into human thresholds via scaling techniques.^{12,23} While there have been many studies of rotation-induced head injuries, these studies tend to either not report injury criteria or support the curve shown in Figure 4. Rotational acceleration may be measured in a dummy by using a nine linear accelerometer package. Angular velocity may be measured by integrating the angular acceleration.

It should be emphasized again that *two* criteria for brain injury are recommended to be used in tandem. Thus, if MSC predicts an AIS 1 and the rotational criterion predicts an AIS 4, the injury is an AIS 4. Most real-world accidents as well as our tests involve both direct contact impact (measured by the MSC) and rotational injury. Until an integrated criterion addressing both components is developed, we would recommend the combined use of the MSC and the angular acceleration tolerance levels as indicated below.³⁷

For Contact Impacts and Translational Accelerations—Use MSC

AIS 1	MSC .00256	Values of mean strain
AIS 2	MSC .00433	
AIS 3	MSC .00610	
AIS 4	MSC .00787	
AIS 5	MSC .00964	
AIS 6	MSC .00964	

For Rotational Accelerations—Measure rotational acceleration ($\ddot{\theta}$) about the center of gravity (cg) of the head, and the rotational velocity ($\dot{\theta}$) about the head cg.

If $\dot{\theta} \geq 30$ rad/second and:		
$\ddot{\theta} < 1700$ rad/second ²		AIS 2
$\ddot{\theta} < 3000$ rad/second ²		AIS 3
$\ddot{\theta} < 3900$ rad/second ²		AIS 4
$\ddot{\theta} < 4500$ rad/second ²		AIS 5

If $\dot{\theta} < 30$ rad/second and:		
$\ddot{\theta} < 4500$ rad/second ²		AIS 0 or 1
$\ddot{\theta} \geq 4500$ rad/second ²		AIS 5

SUGGESTED AREAS OF RESEARCH

The following is an eclectic listing of what I consider to be some significant problems that need to be tackled within four methodologic categories for the study of closed head injuries.

I. Animal Models (including cellular and tissue studies)

- A. Inertial loading. This technique has provided very good data on combined rotational and translational acceleration-induced trauma but has not yet been used to validate scaling predictions for head injury tolerances based on other techniques. Only one report of pure translational loading in the squirrel monkey is available,¹ and these data require validation and extension in larger brains.
- B. Direct impact. Use of impact techniques to examine more closely the interaction between the contact phenomena and inertial loading effects is required.¹²
- C. Neuropathologic and neurophysiologic studies. The excellent studies of Adams and Graham require extension to the electron microscopy level now that the DAI lesion locations can be reproduced reliably in the coronal angulation mode.^{15,16} Dynamic and functional studies of the morphology and neurophysiology of axonal transport in suitable invertebrate preparations, such as the squid axon, under controlled loading conditions may well provide valuable insights into the field strain parameters that correlate with reversible and irreversible disruption of axonal functions by mechanical loads. Analog studies on neuronal cell body synaptic physiopathology and cerebral vascular physiology are also needed.
- D. Measurement of material and structural properties. Although some data on the mechanical properties of cranial tissues are available, much more are needed.³⁸

II. Physical Models

There are currently three groups working with physical assemblies modeling the head and neck, at Goteborg in Sweden (Dr. B. Aldman et al.), at Berkeley (Dr. W. Goldsmith), and at Philadelphia (Dr. L. Thibault). Improvements in these models in conjunction with work on the animal and tissue studies will be extremely valuable.

III. Human Studies

- A. Accident data banks. Information from such sources as well as from in-depth longitudinal studies of selected cases is invaluable in forming useful hypotheses for further investigation as well as to validate predictions made on the basis of experimental or theoretical work. In a recent consensus workshop organized by the National Highway Traffic Safety Administration (NHTSA) it was noted, however, that there is a significant mismatch of such data in terms of the biomechanical and medical data sets.⁷ Thus, NIH-supported data banks

are rich in clinical and physiopathologic information but poor in biomechanical input data for these accidents. The opposite seems to hold for the NHTSA-supported data banks, such as the National Crash Severity Study and National Accident Sampling System files.⁷ In collaboration with Dr. Howard Champion at the Washington Hospital Center, we are currently developing a new methodology seeking to correct this mismatch in a pilot study of 100 motor vehicle accident cases where the accident investigation and biomechanical data gathering are triggered by the arrival of the suitably injured patient at the trauma center. The data obtained will be used in improved accident reconstruction using physical and mathematical techniques.

IV. Theoretical Models

A large number of mathematical models have been developed, all of which have used linear theories involving only small strains. Nonlinearities associated with large strains, large rotations, or nonlinear material responses have not been modeled. Although it is true that the formalization of such complex properties can be computationally formidable it is necessary if we are not to remain saddled with trivial solutions. Thus, brain tissue is most probably anisotropic, inhomogeneous, and of a multiphase nature. Neuroanatomic data would indicate that the brain is certainly a composite material with variable orientation of the local principal directions of material response throughout its substance.³²

The finite element method for mathematical modeling is a powerful tool that will undoubtedly prove to be even more useful than hitherto.³⁹ However, it would appear that the availability of better brain material property data and development of a model able to handle nonlinearities and large strains are essential.⁴⁰

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