PERSPECTIVES ON HEAD INJURY RESEARCH

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ABSTRACT

A review of the current medical and engineering literature provides sufficient understanding of head injury to direct meaningful research. Serious closed head injury typically involves damage to the blood vessels of the brain. Half of the vascular injuries involve subdural hematoma including bridging vein laceration and 30 percent involve focal contusion. Vascular damage is frequently identified with cases of long-duration unconsciousness or coma following closed head injury.

Impact of the front or back of the head results primarily in vascular damage on the inferior surfaces of the frontal and temporal lobes. These are the locations where the brain rests on the skull in the cranium and are different from the speculative sites of neural damage associated with unconsciousness. However, vascular injury of the brain is a specific endpoint for research since the damage is obvious and is generally associated with severe neural damage.

The literature is filled with discussions on the mechanism of head impact injury. Impact produces three-dimensional translation and rotation of the head about its center of gravity. Since the brain is loosely coupled to the skull by the CSF fluid interface, tethering veins, and the membranes covering the brain, abrupt acceleration of the skull generally causes the brain to lag in response. Because of the irregular bony ridges of the frontal and temporal shelves of the skull which support the brain and stiff membranes, relative motion between brain and skull at these locations has a high potential for contusion injury.

Although many mechanisms have been proposed for the injurious effects of head impact, including pressure gradients and cavitation, relative motion between brain and skull, and brain tissue shear, are implicated as the primary mechanisms of severe vascular damage in closed head impact injury. Current methods to evaluate head impact response involve an incomplete evaluation of dummy head dynamics and an inadequate analysis of the brain response during impact.

Fruitful areas of research should emphasize a more complete evaluation of three-dimensional translational and rotational motion of the dummy head during impacts associated with serious head injury. The accurate measurement of head dynamics will allow a more realistic analysis of the potential for brain injury at the known sites of primary vascular damage. The analysis procedure could rely on an advanced finite element

model of the skull and brain; and, it could predict relative motion between brain and skull, and shear strains in the brain. Although the research would emphasize vascular damage as an endpoint, it is important to develop information on the mechanisms of neural injury causing prolonged unconsciousness and coma. The mechanism of neural damage is probably not identical to that for vascular injury, and a clearer understanding of the biological factors involved in neural trauma would eventually enable meaningful biomechanics research.

Although a review of the literature points clearly to the fact that the current head injury criterion (HIC) is based on minimal data over 20 years old, the criterion has been useful for the assessment of automotive safety with anthropomorphic dummies. Yet questions remain unanswered as to whether the criterion is a sufficient procedure for the accurate prediction of the wide range in types and severities of head injury. Due to the significance of head injury as a source of critical, fatal and disabling injury in motor vehicle crashes, attention should be given to research which will evaluate current techniques and develop new procedures to evaluate head injury potential and to assess the benefits of safety technologies.

Incidence and Sources of Head Injury

Based on U.S.A. field accident data (29,31), the head is one of the most frequent body regions injured in motor vehicle crashes. It is also the most severely injured part of the body in half of the cases (Table 1). Head injury occurs with high frequency for each level of injury severity and it is a significant crash injury problem because of the death and disability it causes. When the severity and frequency of injury is considered (18,29), head injury accounts for 30% of the car occupant harm (Table 2). Nearly 80% of the head injury harm is due to interior contacts for the primarily unrestrained U.S.A. occupants. About half of these contacts are with structural members of the interior, such as pillars, rails, windshield, side glass, and steering assembly.

Head injury is a significant societal problem and represents a major fraction of the cost of crash injuries (32). Early estimates of the total societal burden of motor vehicle crash injuries (30) indicate a total U.S.A. cost of approximately $60 billion annually (Table 3). Considering that head injuries account for nearly 30% of the crash injury harm by one estimate, the cost of these injuries approaches $20 billion annually. This estimate of harm was based on a uniform cost for each severity of injury, irrespective of the cost differences for injuries of similar AIS severity (an AIS 5 brain injury has a significantly greater medical and rehabilitation cost than an AIS 5 liver injury). In a more recent estimate of motor vehicle crash harm (4), the significance of individual injuries was included, and head injuries actually approach 50% of the total societal burden of motor vehicle trauma.

In a majority of crashes, vehicle crush occurs with minimal deformation of the occupant compartment, but involves a combination of frontal, lateral, and rotational motions of the vehicle. Because of a distribution in vehicle dynamics, unrestrained occupants interact with vir-

tually all interior components, and head contact occurs over a broad range of velocities. Thus, the occupant's velocity and direction of interior impact and the properties of the object struck are primary factors in the subsequent impact interaction of the head. For the cases of significant intrusion or ridedown, the head contact velocity and impact mechanics are more complicated.

Concussive brain injury (29) occurs in a wide range of interior contacts (Figure 1). For minor, moderate, and severe injury, windshield contacts represent the most common source of injury (Here AIS-76 terminology is used and ascribes cerebral concussion for varying lengths of unconsciousness including coma following head impact. Non-anatomical brain injuries in AIS-80 are ascribed on the length of unconsciousness thus limiting the severity of the term concussion to AIS 2). When the header, pillars, and instrument panel are added as sources of contact injury, these structures account for nearly 3/4 of the concussive injury occurring in motor vehicle crashes. Although there have been significant improvements in occupant protection by the introduction of the high penetration resistant windshield and energy absorbing structures, these vehicle components remain a significant source of brain injury. When critical and fatal brain injuries are considered, the pillars and header account for nearly 50% of the contact injury.

Types of Head Injuries and Their Significance

Based on the data available in the NCSS accident files (18,29), head injuries occur primarily to the face and brain, and over 400,000 occupants experience concussive injury (Table 4). Nearly 80% of these injuries are AIS 1 level concussions which involve confusion, dizziness and amnesia after the crash (Table 5a). AIS 1 and 2 level injuries of the brain do not involve damage apparent on a CT scan, and do not involve skull fracture or intracranial bleeding (21). The more severe injuries of the brain involve contusion and laceration and are generally accompanied by long-duration unconsciousness (Table 5b). Severe injuries involve a significant risk of morbidity and fatality (7), and can involve skull fracture.

Independent estimates (Table 6) indicate that over 200,000 U.S.A. hospital admissions occur annually as the result of brain injury (14,18,31). This represents approximately 16% of the head injured and an important fraction of the total hospital admissions for crash injuries. A majority of the cases involve confusion, amnesia and short duration unconsciousness where admission for overnight observation may be precautionary treatment before release. In the more severe cases, prolonged hospitalization and rehabilitation are required.

The available accident injury and hospital data enable only rough estimates of the frequency of head injuries; and, better data, such as from the NASS and NEISS, are needed to develop a more consistent picture of the epidemiology of head impact injury. However, the area where there is the least data is probably the most important aspect of head impact injury, the disabling effects of brain damage. Only scant information is available on the consequences, treatment and rehabilitation of the

severely brain injured, and even less on the consequences of moderate head injury.

Injury disability is an important emerging problem of crash injury (26,32). With improved medical treatment, there is better survival of the severely brain injured than would have been expected years ago. As the costs for post-traumatic care increase and the number of permanently disabled increase, more attention will focus on the disabling consequences of impact injury. In this regard, safety technologies and injury assessment tools will be needed to protect against disabling injury.

Vascular Injury: Over half of the contusion injury of the brain is subdural hematoma (7,14), frequently attributable to bridging vein rupture (Table 7). Subdural hematoma has the highest risk of mortality, exceeding 60% of the survivors admitted to hospital. Focal injuries occur in about one-third of the cases but typically without skull fracture. In contrast, epidural hematoma frequently occurs with skull fracture. Based on autopsy evaluations of cerebral contusion (5,6,8,9,10,-16,20,22), the most common sites of vascular injury are to the inferior surfaces of the frontal and temporal lobes of the brain (Figure 2). These are the locations where the brain rests on the skull in the cranium and are sites of bony ridges which may be involved in the contusion injury process. Contusion of the frontal and temporal lobes occurs whether the site of head impact is to the front or back of the head (6,8,20). This fact implicates the geometric configuration of the brain and skull at these sites as a significant factor in the injury process.

Neural Injury: Injury of nervous tissue is typically diagnosed by abnormal brain function, either loss of memory and cognitive function, or a loss of consciousness. The more significant concussive injuries involve long-duration unconsciousness and are frequently associated with severe contusion injury (21). In the cases of moderate and minor brain injury, which is more frequent, the injury diagnosis is based on short duration unconsciousness, or confusion and amnesia (Table 5a and 5b). Although such injuries are frequently thought of as benign, follow-up of patients has identified postconcussive sequelae (28), where minor head injuries can have a significant life-altering consequence (17,25).

Although there is a low risk of mortality and a good prospect for recovery from minor head injury (25), the situation is less favorable for the moderately injured (19,24,26) (Table 8). Only 40% of the patients achieve good recovery. The longer-term effects of the head injury (25) are also a significant factor since many patients with minor and moderate injury experience chronic headaches and memory deficits months after the injury. Based on neuropsychological evaluations, these individuals show a deficit in comparison to a matched normal population of people. There appears to be a psychological change in these patients accompanied by loss of employment in many cases. What might be construed as a minor or moderate injury with insignificant consequences based on the AIS 2 or 3 level injury severity, is actually a more significant problem because it may involve an irreversible alteration of

normal brain function. Even though AIS is a threat-to-life scale, many people interpret an injury severity level as a measure of potential outcome. A complementary "well-being" scale is needed and would assess the long-term effects of crash injuries.

Recent efforts in occupant protection research have focused primarily on technologies to reduce crash injury deaths. This has brought about significant reductions in fatality rates over the years. However, protection from injury disability requires research on technologies and treatments to reduce the incidence of impairment. Many of the disabled are permanently confined to wheelchairs or limited in activity, and the severely disabled are quickly becoming a major health-care issue. The majority of injury disability is from brain and spinal cord injury (26,32) which permanently destroy motor, sensory or cognitive function and require attendant care in nearly half of the cases (Figure 3). Only one in ten of these victims will return to gainful employment and nearly 40% will be unemployable for years after the injury. These individuals face an average life expectancy of 36 years with nearly a quarter living 50 years or more after the injury.

Head Injury Mechanisms

There is extensive literature on the possible mechanisms of head impact injury (33). Unfortunately, the mechanisms underlying vascular and neural injury of brain tissue are complex and many of the published mechanisms are speculative. Some do not adequately account for the underlying physics of head impact. Although, many papers have significantly contributed to the field of head injury mechanisms, some good papers have gone unnoticed in favor of others espousing theories that at first glance appear attractive and thus have become popular in the literature. As fundamental an issue as coup-contrecoup brain injury has become muddled and confused in the numerous papers published on the subject. The definition of a coup and contrecoup injury is not clear. The intent of this overview is not to critically review the substantial literature on mechanisms of head injury, that is a subject for a more comprehensive document, but rather this paper intends to give a perspective on the most likely mechanism underlying closed head contusive injury of the brain and on the needs for research.

The violent acceleration and motion of the skull due to impact produce deformation of brain tissues secondary to skull displacement and can result in both vascular and neural damage. The wide range of direction and location of head impact produces a complex motion of the head and complex deformations of brain tissues. Thus, impact produces a three-dimensional translation and rotation of the head about its center of gravity (Figure 4). Because the brain has inertia and is loosely coupled to the skull, its motion lags the displacement of the skull. The differential displacement causes shear between the brain and skull stretching the vessels that tether the brain (5,11,20), and strain in brain tissue (21,23,28) due to deformations from contact with bony protrusions and membranes (15).

Differential displacements due to tethering, geometric factors and pressure gradients in the brain cause a complex distortion of CNS tissue, which is accentuated at the interfaces between brain and stiff intracranial tissues and structures. Deformation of brain tissue strains the material and can result in brain laceration and contusion. Vascular injuries primarily occur on the inferior surfaces of the frontal and temporal lobes where the ridgy convolutions of the skull accentuate the potential for injury by relative motion between brain and stiffer structures (6,15,21). This is implicated as the primary mechanism of severe vascular injury in closed head impact injury.

Head Dynamics

Since there is minimal risk of brain injury due to non-contact acceleration of the head (18), the accurate measurement of head dynamics during direct head impact is of primary importance. Blunt impact produces translational and rotational acceleration of the head. Because of the wide range of impact types and the lack of measurement technology, the relative significance of translation and rotation to the deformation of brain tissue has not been clarified. Clarification can be achieved only through accurate measurement of the three-dimensional dynamics of the head during severe impact.

A first step is to better understand dummy head dynamics during impacts which have a high probability of brain injury. Field investigations of head injury can provide the impact situations. Although multiple accelerometer techniques have been developed and used in dummy heads, most of the analysis techniques suffer inaccuracies in interpretating the rotational and translational acceleration during violent impacts. A technique to measure dummy head dynamics must be capable of accurately measuring translational accelerations up to 500 g and rotational accelerations up to 50,000 r/s^2 (Table 9). Head impact causing severe brain injury is a short durations event (27), particularly when hard structures such as rails and windshield glass are contacted.

A recent effort to more accurately measure the fore-and-aft (two-dimensional) rotational and translational acceleration of the dummy head has achieved success (3), even in violent impact exposures. The technique relies upon multiple linear accelerometers aligned in the midsagittal plane and a linear least-squares evaluation to determine rotational acceleration about the center of gravity of the dummy head. The advantage of this technique is that the conventional triaxial accelerometer package at the center of gravity of the dummy head is maintained for computation of HIC and comparison of results.

Acceleration of the head is only the driving force which results in deformations of brain tissue. Brain deformations cause injury. Thus, the accurate measurement of head dynamics is only the first step in a procedure to evaluate risk of brain injury. Acceleration data must be used as input to a "post-processing" procedure to predict engineering responses, such as shear strain or tensile strain, at locations where contusion injury of the brain typically occur. One procedure may involve a finite element model (see (12) for a review of models) which

closely approximates the geometric and interface conditions of brain and skull, and whose response predicts brain deformations due to translational and rotational accelerations. This procedure will require a tolerance criterion for tissue damage but would provide a more global assessment of brain injury risk.

The current head injury assessment technology and criterion use a stiff dummy skull covered by an elastic skin, measure a single point acceleration at the head center of gravity, and use a weighting function based on the resultant acceleration to assess the severity of head impact and potential for brain injury. However, the current weighting function (HIC) is not a decisive correlate with vascular damage observed in cadaver experiments (Figure 5). The criterion is also not a precise predictor of skull fracture. One difficulty of correlating injury with HIC may be due to the single point evaluation of risk. The HIC at the interface between brain and skull can be very different from the HIC at the center of gravity (13), depending on the relative magnitude of the rotational acceleration (Figure 6). It is important to recognize that impact experiments show significant rotational accelerations and the site of injury is typically not at the head center of gravity.

Injury Biomechanics

Because of the high frequency and potential significance of neural damage to central nervous tissues, the study of concussion is an important research topic. However, there is scant information on the basic biological mechanisms of neural trauma, and it may not be an immediate response. Neural trauma is a progressive injury which takes time for the pathophysiology to reach a permanent endpoint. Even though unconsciousness is a diagnosable symptom, it may mask a sequence of biological processes that may actually account for the ultimate severity of brain damage.

In the experimental setting it is difficult to localize and observe neural injury except with histology (1,2,28) and anesthesia is a compounding factor. Because of the infancy of neural trauma research, basic study is needed using well-controlled experimental models, and the research teams must include neural scientists, biomechanics and physiologists, to investigate the response of CNS tissues to trauma (Table 10). In the longer term, neural trauma research will link up with other studies that have advanced our understanding of the biomechanics of vascular brain injuries.

At this time, the most obvious and important objective for brain injury research is to understand the injury biomechanics associated with vascular brain damage. Injury causing long-duration unconsciousness is generally accompanied by contusion injury, and the contusion is easy to localization and diagnosis. Thus, experimental research may use vascular injury as an endpoint to assess the significance of translational and rotational accelerations of the skull, of relative motion between brain and skull, and of deformations of brain tissues.

Although it is likely that the mechanism of neural injury is different from that of vascular injury, the development of knowledge on head dynamics causing brain contusion should provide a better engineering criterion to assess safety technologies. Head injury tolerance research would also benefit from study of skull and facial fracture where the emphasis should be on the biomechanics of injury. Eventually, the head injury criterion must adequately address brain injuries causing functional damage, impairment of cognition, changes in personality and behavior, and altered physical and occupational function. However, our knowledge of the basic mechanisms of neural damage is not complete enough at this time to consider biomechanics studies on closed head neural tolerances. Disability caused by brain damage will be an important problem of the future but progress on reducing head injuries is most likely if the biomechanics of severe brain contusion is clarified first, and new technologies developed and used to evaluate safety technologies.

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Table 1

DISTRIBUTION OF MOTOR VEHICLE
CRASH INJURIES,
(Developed from {31}, Annual Injury Projections
Based on 1982 Crash Injury Data in NASS)

Body Region	Injury Severity Level Minor AIS 1-2	Serious AIS 3-4	Critical AIS 5-6	Most Severe Injury
Head	2,613,000	50,000	17,000	50%
Spine	929,000	13,900	2,000	16%
Chest	350,500	36,500	16,000	3%
Abdomen	257,600	55,400	12,000	5%
Extremities	3,245,000	105,000	--	26%
Whole Body	97,000	--	--	--
Total	7,491,000	260,000	49,000	100%

Table 2

CAR OCCUPANT INJURY HARM
(Developed from {18,29}, Frequency Distribution of
Contact Harm based on 1977 to 1979 Crash Injury Data in NCSS)

Principal Contact	Head	Neck	Trunk	Extremities	All
Steering Assembly	3.1	0.4	21.3	2.0	26.9
Instrument Panel	1.9	0.4	6.0	6.4	14.6
Side Interior	0.8	0.1	9.3	2.6	13.0
Pillars-Rails	7.7	0.6	0.5	0.1	8.9
Windshield-Glass	5.5	0.2	-	0.4	6.1
Other	4.9	7.3	1.7	4.9	18.7
Interior Contacts	23.9	9.0	38.8	16.4	88.2
Exterior Contacts	5.8	1.6	3.2	1.2	11.8
TOTAL	29.7	10.6	42.0	17.6	100.0

Table 3

SOCIETAL COSTS OF MOTOR VEHICLE ACCIDENTS
IN BILLIONS OF $ 1980
(Developed from data in {30}, Annual Societal Costs of
Crash Injury Based on the Average Accident Data from
the 1979 to 1980 NASS)

	Uninvolved	Property Damage Only	Injury (AIS) 1	2	3	4	5	Fatality	Total
Medical Costs	-	-	.54	.62	.63	.34	1.13	.07	3.33
Productivity Losses	-	-	.32	.25	.31	.45	.80	12.10	14.24
Property Loss	-	16.98	2.66	.61	.42	.10	.03	.17	20.98
Legal-Court Costs	-	.36	1.74	.26	.53	.18	.09	.68	3.84
Insurance Expenses	7.05	3.45	1.75	.24	.11	.44	.15	.64	13.83
Other (EMS, Coroner, etc)	-	.32	.44	.09	.04	.02	.01	.06	.98
	7.05	21.11	7.45	2.08	2.05	1.53	2.20	13.73	57.20

Table 4

HEAD INJURY ESTIMATES
(Developed from data in {18,29}, Annual Injury Projections
Based on Data from the 1977 to 1979 NCSS)

	AIS 1	AIS 2	AIS 3	AIS 4	AIS 5	AIS 6	
Head Injury	1,186,900	193,400	17,400	9,000	10,900	6,400	1,420,000
Face	817,800	104,400	11,300	1,800	-	-	935,000
Brain	341,800	83,200	5,200	5,200	10,700	6,300	453,000
Brain Injury							
Concussion	341,800	83,200	5,200	2,900	4,400	500	437,000
Contusion/Laceration	-	-	-	2,300	6,300	5,800	14,400

Table 5

NEURAL INJURY
Derived from (21)
(a)

CONFUSION

AMNESIA only with confusion

KNOCKOUT (< 15 m) only with amnesia
(Lancet 1962)

UNCONSCIOUSNESS (1 < hr) only with focal damage
(Lancet 1973)

DEEP COMA (> 6 hrs) only with brainstem damage
(Brain 1974)

(b)

Injury Severity		Symptom	Brain Damage	Outcome
-	AIS 1	Confusion/Amnesia	Negative CT Scan No Skull Fracture	Post Concussive Sequelae
Minor	AIS 2	Knockout (< 15 m)	No Intracranial Bleeding	
Moderate	AIS 3	Unconsciousness (< 1 hr)	Positive CT Scan < 50% Skull Fracture	> 50% Mortality
Severe	AIS 4-6	Coma (> 1 hr) - Death	Focal Contusions and Bleeding	> 35% Morbidity

Table 6

SIGNIFICANCE OF HEAD INJURY,
(Developed from {14,18,29,31}, Annual Injury Projections
Based on Data from the 1977 to 1979 NCSS, 1982 NASS,
and 1980 Clinical Data)

	NCSS/NASS	NIH
Injured	1,424,000	
Hospitalized	223,000	207,000
Fatalities	13,790	

Table 7

VASCULAR INJURY,
(Developed from data in {7,14}, Frequency Distribution
Based on Clinical Injury Data from 1980)

Hematoma	Frequency (%)	Mortality (%)	Skull Fracture (%)
Epidural	15	20	90
Subdural (Bridging Veins)	51	62	50
Focal	33	40	-
	100%		

Table 8

HEAD INJURY OUTCOME,
(Developed from data in {7,24,25}, Frequency Distribution
Based on Clinical Injury Data from 1980)

Severity	Frequency (%)	Mortality (%)	Recovery (% Good)
Minor	70	0	75
Moderate	20	17	38
Severe	10	41	26

Followup at 3 Months

Severity	Chronic Headache (%)	Memory Deficit (%)	Neuropsychological Deficit	Unemployment (%)
Minor	78	59	+	34
Moderate	93	90	+++	66
Severe	na	na	na	>75

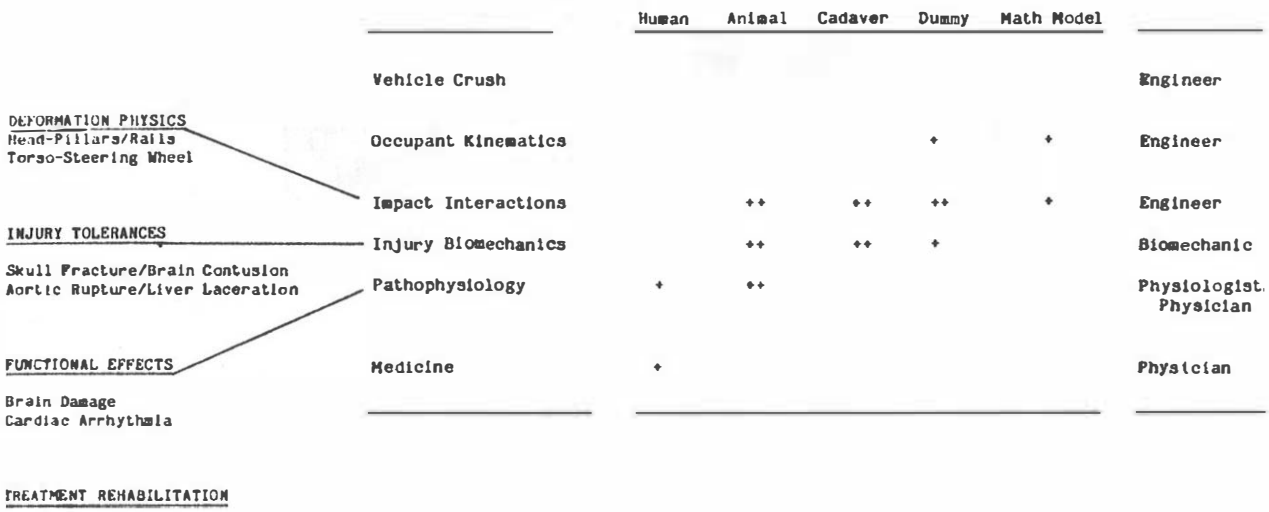
Table 9

CADAVER HEAD IMPACTS,
(Developed from data in {27})

	Force		Acceleration	
	Peak (kN)	Duration (ms)	Translation (HIC (g))	Rotation (r/s^2 (g))
Frontal Impact				
Padded (AIS 4)	6.6	9.4	1,170 (190)	9,570 (66)
Rigid (AIS 2)	14.6	3.8	5,560 (515)	14,620 (102)
Lateral Impact				
Padded (AIS 2)	4.2	10.6	580 (140)	6,650 (46)
Rigid (AIS 5)	9.6	6.9	11,050 (530)	37,550 (260)

Table 10

INJURY BIOMECHANICS RESEARCH TOOLS



	Human	Animal	Cadaver	Dummy	Math Model	
Vehicle Crush						Engineer
Occupant Kinematics				+	+	Engineer
Impact Interactions		++	++	++	+	Engineer
Injury Biomechanics		++	++	+		Biomechanic
Pathophysiology	+	++				Physiologist, Physician
Medicine	+					Physician

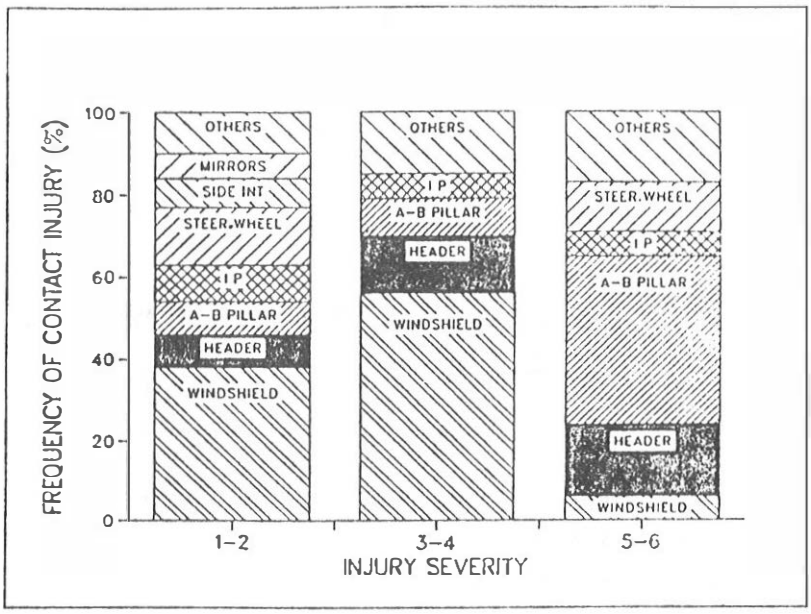


Fig. 1: Distribution of brain concussion injury, developed from data in (29), from crash injury data collected from 1977 to 1979 in the NCSS, courtesy of P. Park and T. Khalil.

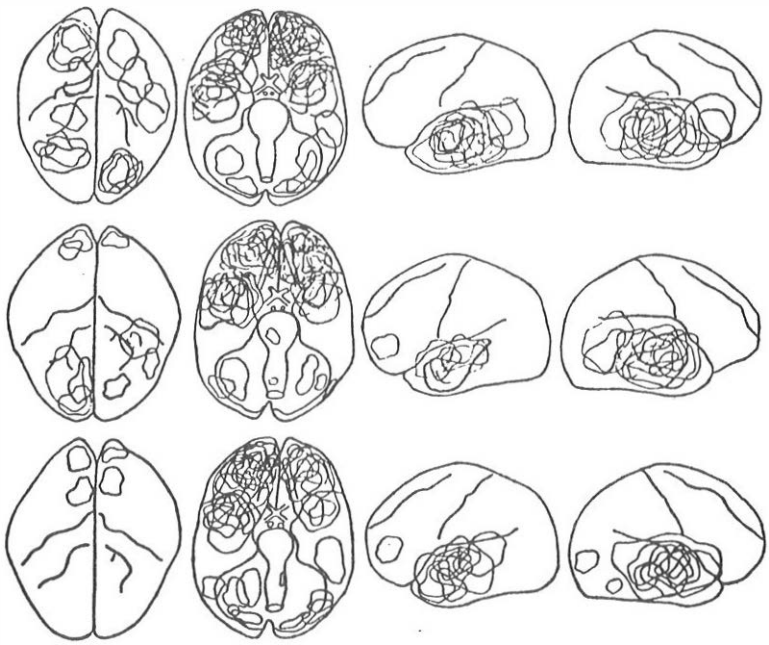


Fig. 2: Composite location of cerebral contusion based on 152 autopsies, from (8,9) with permission.

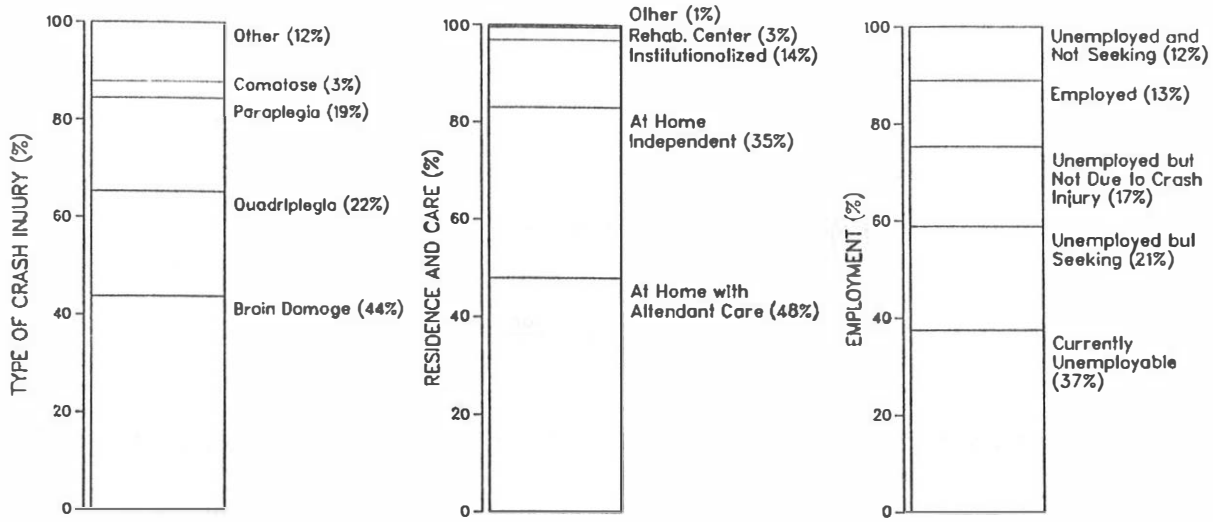


Fig. 3: Types of disabling crash injury and consequences of disability, from (32), based on crash injury followup of victims in 1978 and 1982.

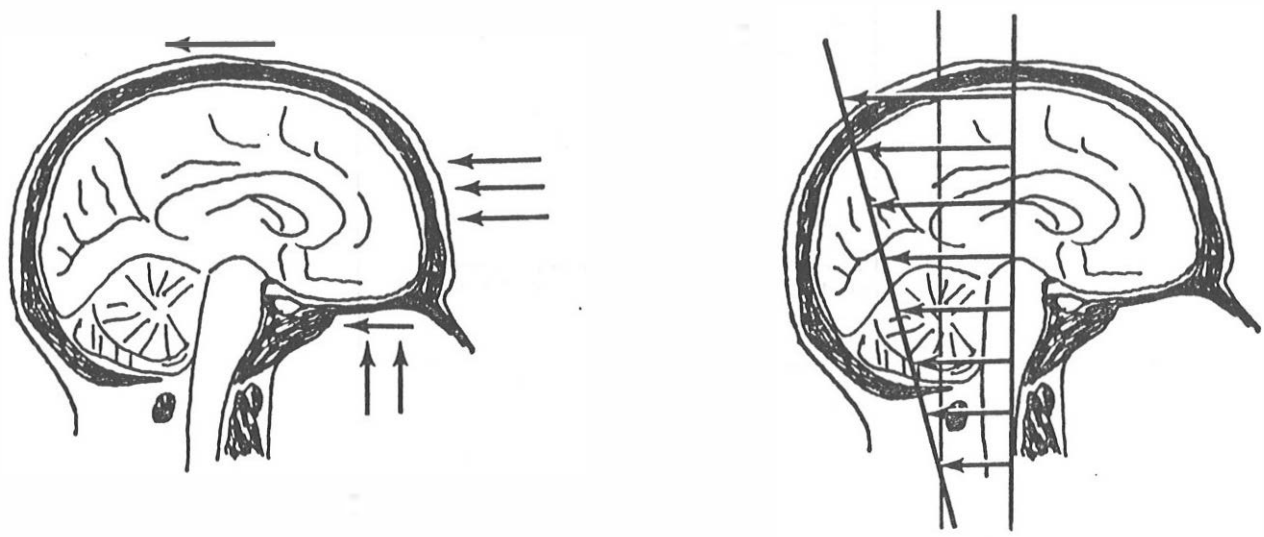


Fig. 4: Head dynamics involve translational and rotational acceleration.

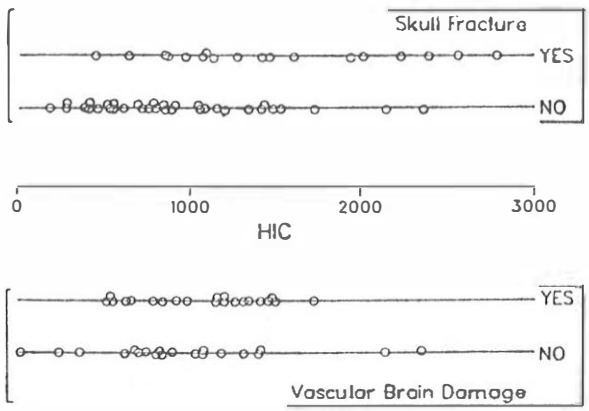


Fig. 5: Cadaver head impact data, developed from data collected by Mertz, 1983.

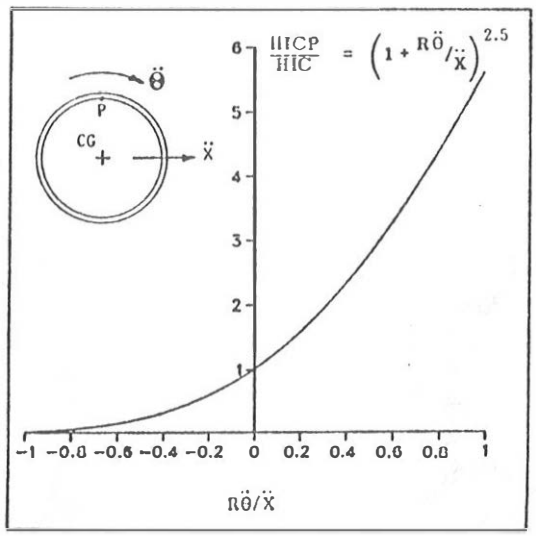


Fig. 6: HIC is not a unique measure of the severity of head acceleration, from (13) with permission.