

REVIEW

Biomechanics and neuropathology of adult and paediatric head injury

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Abstract

The objective of this study was to understand the biomechanics in age-related primary traumatic brain injuries (TBI) causing initial severity and secondary progressive damage and to develop strategy reducing TBI outcome variability using biomechanical reconstruction to identify types of causal mechanisms prior to clinical trials of neuro-protective treatment. The methods included the explanation of TBI biomechanics and physiopathological mechanisms from dual perspectives of neurosurgery and biomechanical engineering. Scaling of tolerances for skull failure and brain injuries in infants, children and adults are developed. Diagnostic assumptions without biomechanical considerations are critiqued. Methods for retrospective TBI reconstruction for prevention are summarized. Mechanisms of TBI are based on the differences between the mechanical properties of the head and neck related to age. Skull fracture levels correlate with increasing cranial bone thickness and in the development of the cranial sutures in infants and in adults. Head injury tolerance levels at three age categories for cerebral concussion, skull fracture and three grades of diffuse axonal injuries (DAI) are presented. Brain mass correlates inversely for TBI caused by angular head motions and locations of injurious stresses are predictable by centripetal theory. Improved quantitative diagnosis of TBI type and severity levels depend primarily on age and biomechanical mechanisms. Reconstruction of the biomechanics is feasible and enables quantitative stratification of TBI severity. Experimental treatment has succeeded in preventing progressive damage in animal TBI models. In humans this has failed, because the animal model received biomechanically controlled TBI and humans did not. Clinical similarities of human TBI patients do not necessarily predict equivalent biomechanics because such trauma can be produced in various ways. We recommend 'reverse engineering' for in-depth reconstruction of the TBI injury mechanism for qualitative diagnoses and reduction of outcome variability.

Key words: *Adults, biomechanics, children, head injuries, mechanisms, reconstruction, youths.*

Mechanisms of traumatic brain injuries

This report addresses causation of traumatic brain injury (TBI) with special reference to critical differences in the biomechanical properties, and responses of the skulls and brains of infants, children and adults.

The incidence of TBI is highest among youths peaking at age 15–24 years. Children aged 5 and under, as well as the elderly aged 65 and over occupy similar secondary peaks suggestive of their vulnerability.⁴⁷ Unintentional injuries are the leading killer of children. Each year, more children, ages 1–14 die from unintentional injuries than from all childhood diseases combined. Under age 1, however, homicide is the leading cause of injury deaths. Head injury is the most frequent cause of death in the paediatric population, comprising between 50 and 80% of all trauma related deaths

each year. Annual paediatric head injury statistics represent an enormous emotional and financial burden of 300,000–400,000 hospitalizations, 6000 to 7000 deaths, and an estimated \$10 billion in annual costs for injury to our children in the USA.⁷⁴

A recent survey by the National Center for Injury Prevention and Control (CDC) of hospitalized and fatal TBI cases in infants 0–4 years old and in youths 15–19 years old in seven states shows the TBI counts and rates per 100,000 for 1994 (Table I). Falls were the major cause of death in infants followed by motor vehicle crashes and then by intentional injuries. Youths died primarily in motor vehicle crashes, followed by falls and then by assaults.¹¹⁷

Our review of the clinical and biomechanical literature regarding the basic mechanisms of the aetiology of TBI in adults is fairly well understood.^{4,23,25,26,93} The biomechanics of paediatric

TABLE I. Causes of traumatic brain injury in infants and youths. Case counts and rates/100,000.

Causes of TBI	Infants (0-4)				Youths (15-19)			
	Case counts hospitalized & fatal	Case counts fatal only	Rate hospitalized & fatal	Rate fatal only	Case counts hospitalized & fatal	Case counts fatal only	Rate hospitalized & fatal	Rate fatal only
Falls	1124	12	42.6893	0.4558	282.5	10	11.7168	0.4148
Motor vehicle occupant	325	48	12.3434	1.823	1786	318	74.0749	13.1891
Assault	196	39	7.444	1.4812	261.5	9	10.8458	0.3735
Pedestrian	133	31	5.0513	1.774	122.5	18	5.0807	0.7466

* Selected data for 1994, reported from seven states and re-analysed by Dr D. J. Thurman, MD, NCIPC. CD3

TBI, as compared with adult TBI are not as clearly defined in biomechanical or diagnostic terms. The clarification of these diagnostic issues is important because children are very vulnerable to TBI below the ages of 3-5. They also suffer intentional, as well as unintentional injuries. Injuries remain the leading cause of childhood mortality, morbidity and disability. In order to clarify the biomechanical aetiology of primary adult and paediatric TBI, special emphasis of these important issues of the physical basis of TBI biomechanics will be given in Appendix I.

Experimental biomechanics of the skull and brain in traumatic brain injuries

Two sets of our earlier tests on experimental whiplash in adult anaesthetized Rhesus monkeys are summarized as follows: the first set was an exposure to a severe extension/flexion 'whiplash' type of head acceleration associated with a terminal minor head impact at the level of 10% probability of cerebral concussion.⁸⁰ The mechanism that was modelled in these experiments mimicked a severe rear-end collision on a torso-restrained occupant. This type of a single severe inertial load caused acute subdural haematomas (SDH), gliding contusions of the brain and spinal cord injuries in these experiments at angular velocity in the range of 500 radians/s and accelerations of 10,000 radians/s² for 5-6 m duration.⁸⁶ A second set of impact experiments on anaesthetized Rhesus monkeys enabled us to develop the data for impact as well as impulsive tolerance levels.^{90,91} The whiplash experiments were further validated in 1968 after clinical analysis on two patients who suffered whiplash injuries in 30 mph rear end collisions to their automobiles while belted, resulting in subdural haematomas and one fatality.⁸⁶ It should be noted, however, that the magnitude of the angular acceleration and the short duration of the pulse measured in our first set of experiments are typical of head angulations occurring in impact loads and not seen in even the most violent shaking as modelled by Duhaime *et al.* until a terminal impact was given and produced the level of head acceleration as predicted for TBI in our previous experiments.²¹

In our impact and whiplash animal models similar injuries of SDH and contusions are produced, but at half the level of angular velocity by impact alone and twice that required by whiplash.^{83,90,91} Scaling of injury thresholds for brains of different mass were performed in three sets of animal models and extrapolated to the brain mass of an average human.⁸⁹ Our experiments tested Holbourn's hypothesis which indicated the critical factor for brain injury as being angular acceleration and linear acceleration as being innocuous. Based on his theory and experiments on physical 2-D models, Holbourn also postulated that smaller brains would require more force than larger brains.³⁹ We tested the vulnerability of three primate species with increasing brain size to various levels of angular acceleration causing brain injury. Our hypothesis predicted increasing risk for brain damage in larger brains according to an inverse 2/3 power of the brain mass, ranging from 30 to 400 g in our three animal models. These experiments confirmed that larger brains were indeed more vulnerable at lower levels of angular velocity and acceleration as predicted by Holbourn. Higher levels of angular acceleration were required for causing similar injury to the smaller brains.^{83,90} It is improbable that the high speed and severity of the single whiplash produced in our animal model could be achieved by a single manual shake or even a short series of manual shaking of an infant in one episode. Our discovery of a lower level of angular velocity (by 50%) in our impact experiments demonstrated the relative ease with which SDH is produced by impact as compared to whiplash alone.⁸⁸⁻⁹¹ Subsequently, however, our experimental results were referenced as providing the experimental basis of the 'shaken baby syndrome' (SBS) by Caffey, Gulthkelch and others by analogy not realizing that the energy level of acceleration in our work related to speeds at motor vehicle crashes at 30 mph.^{10,33} The reality of fatal brain damage by repeated shaking of the larger adult brain and sustained over repetitive significant periods has been well documented recently in an adult Palestinian who died after repeated and sustained violent shaking in 12 sequential sessions by Israeli interrogators of the Shin Bet without evidence of head

impact. Autopsy findings discovered the findings of acute subdural haematoma, retinal haemorrhages and extensive soft tissue bruises in the shoulders and pectoral muscles.⁹⁷ Similar severity and repeated episodes of shaking in children could also result in fatality with similar neuropathologic lesions, e.g. acute SDH and retinal haemorrhage with soft tissue bruises, as well as skeletal injuries at higher levels of shaking force based on the inverse relation to the head mass. Damage to the neck and the spinal cord analogous to our experimental findings would also be expected.

In 1989, Hadley *et al.* published a report on the 'Infant Whiplash Shake Injury Syndrome'. Post-mortem studies did not reveal any definitive evidence of contact phenomena to the head but the authors emphasized the frequency of injuries to the cervical spinal cord similar to that seen in our whiplash experiments.³⁴ Routine examination of the neck in abused children with head injuries should be examined with MRI.

In a separate series of impact experiments in rhesus monkeys, the mechanism of coup and contra-coup contusions with and without skull fracture was defined. The locations of SDH were primarily over the anterior half of the brain and fronto-temporoparietal SDH was the predominant location with no significant correlation with location of skull fracture. The rotational velocity for these experiments causing the brain lesions was measured using high-speed cinematography at 4000 frames/s. These values ranged between 150 to 250 radians, with impact linear velocities of 40–50 feet/s to occipital and frontal sites of impact.⁸⁸

Our second set of impulsive (non-impact) experiments were also done on anaesthetized monkeys in which the head was constrained in a rigid container with the torso fully restrained, with controlled movement applied to the head neck complex. Two types of head acceleration in two groups of subjects received either *translation* of the centre of gravity (CG) of the head by one inch as compared with identical CG translation *plus* angular acceleration at increasing levels of linear acceleration. Pure translation of the head and neck could not produce hemispheric acute SDH, no diffuse petechial haemorrhages in the brain and no injuries to the cervical cord at linear accelerations in the range of up to 1000 *g*. Similar levels of linear accelerations combined with the angular motion of the head on the neck produced classical bilateral acute SDH and diffuse petechial brain haemorrhages in all subjects without impact.^{83,87,91}

The distribution of the acute lesions in the latter group followed the predictions of our centripetal theory for the location of the TBI lesions which had been developed as our experimental paradigm at the onset of our programme in 1965 based on our the clinical observation of the 'one-way' disassocia-

tion of the amnesic and paralytic phenomena in TBI patients at lower levels of TBI severity and the testing of Holbourn's theory.^{83,84,96} Clinical validation of the centripetal theory in adult and paediatric TBI patients was subsequently confirmed by other investigators using CT and MRI and correlations of the brain lesions with neuropsychological evaluations.^{54,55,56,112,125}

A report by Duhaime, Gennarelli and Thibault *et al.* tested the effect of manually induced inertial forces of an impulsive periodic nature. Adult volunteers manually induced repetitive shaking of the head/neck of an anthropomorphic surrogate 'dummy' equivalent to a 6-month baby. The neck of the surrogate baby was replaced by a hinge to allow maximal angular head accelerations. Accelerometers on the head of the surrogate recorded linear accelerations during repeated shaking of the head of the child surrogate by volunteers. The maximum values of the head accelerations related to the angular motions of the surrogate head were found to be well below the requisite threshold for the production of the critical principal strains in the elements of the brain. In contrast to the low levels of accelerations indicative of strains in the brain during repetitive shaking, a single forced rotational motion of the surrogate by a volunteer ending in a contact of the 'dummy' head with a rigid surface at a table height, immediately resulted in higher levels of acceleration associated with the entire spectrum of TBI as found in infants.²¹ Repetitive and prolonged episodic shaking by mechanical means without head impact and over a range of specific time periods has not been experimentally tested. A large animal model is required to correlate the biomechanics and physiopathology of repetitive and prolonged 'whiplash' shaking, particularly to investigate the cumulative effect of repeated shaking.

A recent experiment by Smith *et al.* has attempted to develop a model for head shaking TBI in 6-day-old rats subjected to intermittent shaking for 6 s followed by 6-s pauses repeated 60 times daily for 3 days. The authors explain that in the development of this model they were unable to produce subdural haemorrhages or neuronal cell loss by shaking alone. They required an added insult of hypoxia combined with shaking in an inverted position in order to obtain subarachnoid haemorrhages and progressive cortical degeneration.¹⁰⁹ The results showed that in the 6-day-old rat pup brains, only when the *shaken plus hypoxic state* was produced, a moderate to severe *subarachnoid* haemorrhage and progressive degeneration of cortical neurons were found. No subdural haematomas were produced. It is to be noted that at day 7, these rat pups have 50% of their adult brain weight and the inverted position during shaking could also produce intracranial hypertension. Unfortunately, no biomechanical

measurements such as head angular excursion, velocity or acceleration were reported for these experiments. Further experiments with appropriate biomechanical data correlated with neuropathology are required in order to develop a useful model, preferably in a larger animal model under more physiological conditions.

Principles of biomechanics

The mathematical basis of biomechanisms is given in the appendix. The causation of TBI can occur either by impact or by impulsive loading (shaking), which lead to different results. The consequences of an impact that are unique to the infant and young child can be explained on the basis of the compliant nature of the brain-case as compared to the adult, relatively non-compliant skull. In the infant, the skull is not a rigid shell structure (the cranial vault) as is found in the adult; it is better characterized as a segmented structure of loosely associated curved plates where the sutures are incapable of supporting bending loads during the early development of the child. Upon impact this structure undergoes large elastic deformation and, in some cases, even plastic deformation, both of which may result in skull fractures of various types. The shape changes of the skull associated with impact to this unique and developing skull of infants produce large strains throughout the cranium and its contents even without the onset of skull fracture and may be subjected to even greater shape changes in skull and brain when fractures do occur. It is this aspect of the mechanism of infant brain injury that needs to be further clarified.

In contrast, impulsive loading (shaking) will produce minimal if any, deformation of the skull. The biomechanical aspects of the injury mechanisms, the definition of the mechanics terminology, and direct comparison to our knowledge base in adult and pediatric head injury is intended to shed light upon the controversial and confounding interpretations of the literature in this field.^{20,30} It is therefore appropriate at this point to discuss the pertinent concepts of the science of mechanics associated with TBI. These include the biomechanical behaviour of tissues undergoing dynamic loading, kinematics, head motions and, in particular, the fundamentals of dynamics of head injury and tolerance criteria.^{1,23-25,32,96,126}

Biomechanical behaviour of tissues upon load application

The response of any solid structure to specified loading depends critically on its composition, geometry and, in particular, on deformation characteristics. An element may be subjected to uni-axial (one-dimensional) load or forces acting along a

given axes. If several forces act along different axes, an equivalent stress or strain can be computed. Different types of stress or corresponding strain occur depending on how the load is applied. Various modes include: (a) elongation, (b) compression, (c) bending, (d) shear, and (e) torsion (twisting). This terminology is explained and illustrated in the appendix.

The brain is not a solid component, but rather a complex rheological substance, which is permeated by vascular structures and is in contact with cerebro-spinal fluid. This complex exhibits many aspects of fluid behaviour. The brain can be represented at the microscopic (axonal or neuron) level, but this would enormously complicate or possibly inhibit any analysis of regional response to loading. Due to the vast number of neural elements and current lack of knowledge of their interaction in spite of substantial local inhomogeneities and anisotropies, the brain has been approximated as a uniform component whose principal properties consist of its density and viscous characteristics, subject to its extensive and pervasive vasculature. However, recent studies have identified differences in properties of various regions of the brain, e.g. gray and white matter, cortex and brain stem tissues. The stability and shape of the living brain *in vivo* is maintained by the cerebral circulation which enables the blood vessels to function as a 'soft' internal and external scaffold for the brain. The blood vessels can be represented as a series of distributed concentric tubes, each with somewhat different properties, whose overall behaviour is quasi-visco-elastic, or more simply as a single homogeneous vessel for each artery or vein. The prenatal or infant skull can be regarded as a series of nearly elastic/brittle, interlocking plates capable of resisting compression and shear, but incapable of transmitting bending across the sutures or fontanelles. The adult skull, consisting of two solid bone layers sandwiching the diploë is a complex structure that is somewhat flexible; sufficiently large forced deformations of the unit frequently result in fracture at tensile strains of approximately 2%, including crushing of the diploë when radial loads are exerted. Figure 1 depicts average values of skull failure stresses in neonates, young children and adults; the resistance to fracture for an adult is 11 times greater than for the neonate.

Dynamics of head injury

The two separate types of *motion* of the head that can occur as the result of application of a force to the system are: (a) translation, where the total applied force F passes through the center of gravity of the head (exemplified by Fig. B of the Appendix) and (b) rotation, produced by the moment of a force

about the centre of gravity (or else by a 'couple', two equal, parallel and oppositely directed forces whose lines of action are distinct). An example of rotation is shown in Fig. 2. In the real world, both translation and rotation are invariably simultaneously present in varying degrees in head injuries.

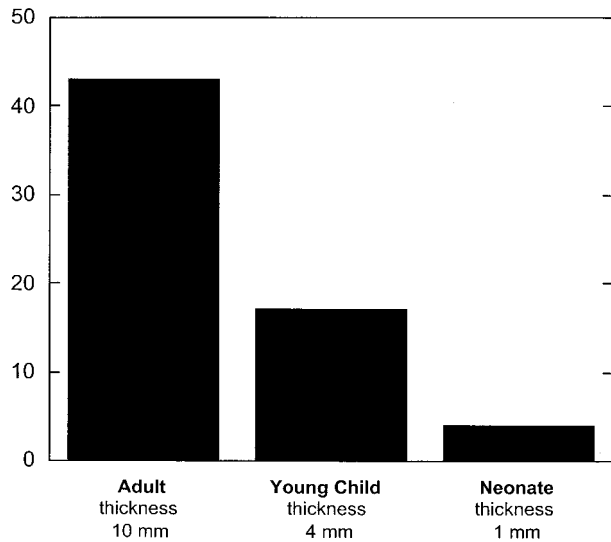


FIG. 1. Skull failure stress in the adult, young child and neonate.

Owing to the uniformity of the kinematic parameters in pure translation throughout a rigid body, which an adult skull approximates in the absence of fracture due to impact, no strain is generated in this object and, hence, no failure will occur. Uniform acceleration for a fluid, on the other hand, generates a pressure gradient in the direction of motion proportional to the acceleration that, in extreme cases, could produce trauma in the brain. Rotation produces differential displacements of adjacent spherical brain layers due to the outwardly increasing translational velocity with respect to the axis of rotation, which is most often located either at the occipital condyles or at the base of the neck. This results in shearing of the tissue, the cause of diffuse axonal injury and various forms of vascular disruptions. The relative motion of the brain with respect to the skull is also responsible for any rupture of vessels such as the bridging veins at the skull/brain interface, which is the most likely cause of subdural haematomas.⁵⁸

Two types of force application, static and dynamic can generate damage to the skull and its contents, that produce different injuries. Static loading, although very rare, occurs slowly as exemplified by the closure of an elevator door in which a head has been imprisoned and subsequent

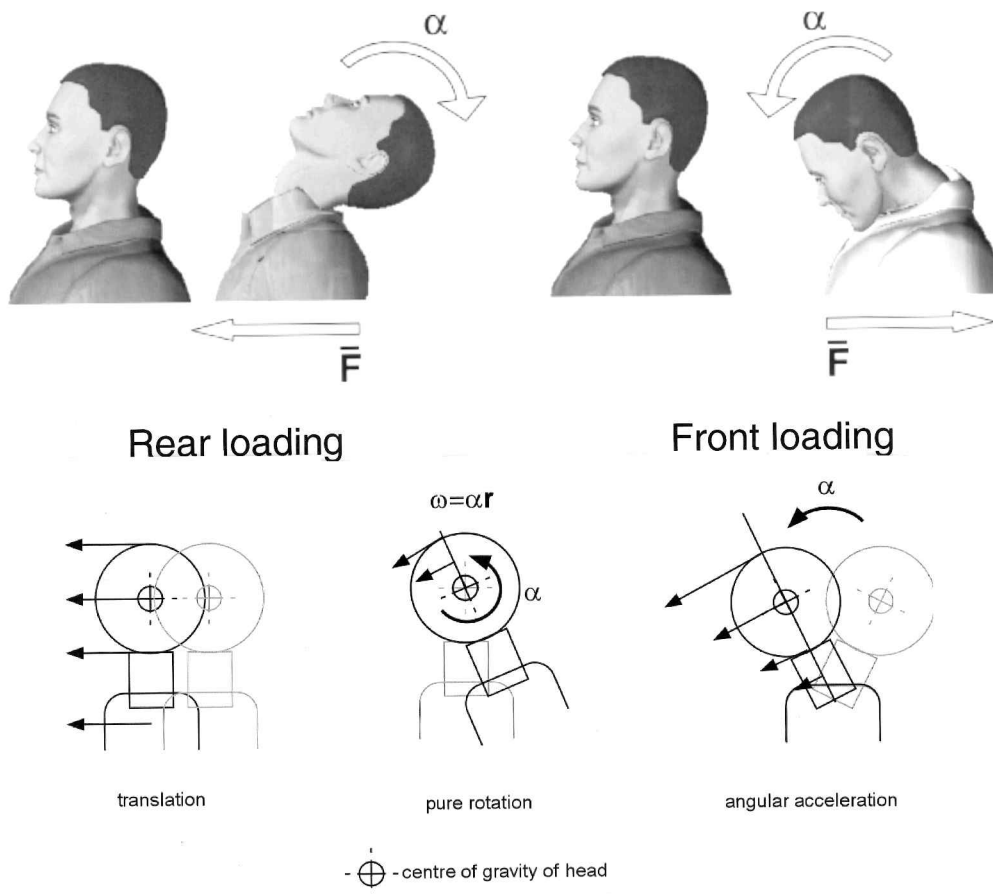


FIG. 2. Impulse loading of head/neck in rear and front loading depicting translation, pure rotation and angular acceleration.

further compression of the cranium. However, by far the two most frequent types of dynamic force application are represented by impulsive loading and by impact loading, two completely different mechanisms with totally different consequences. The first consists of movement of system elements induced by applied motion at an entirely different position of the system (or else by a fluid pressure wave, that is not relevant here). It is exemplified by a vehicular occupant whose car has sustained an impact or in tests involving high speed sleds that are suddenly arrested; the load is applied at the seat, and transmitted through the torso and neck to the head.

Continued impulsive forced motion reversal, such as would occur in the 'shaken baby syndrome' (SBS), also constitutes impulsive action where traumatic effects to the head and neck are generated by the inertial loads. The resulting differential motion of the brain and the skull is amplified by the behaviour of the neck, which is weak in infants and much stronger in adults. The resultant significant relative displacement of the brain, skull, spinal cord and neck results in failure of the para-sagittal bridging veins, contusion and haemorrhages of the

brain upon striking portions of the skull, and diffuse axonal injuries if significant rotation of the head is involved; most importantly, severe shaking will produce damage to the soft tissues of the neck and its contents. Although a single shake may not induce any noticeable trauma, it is possible that continued repetitive shaking over a period of time, or shaking repeated at intervals can produce significant cerebral as well as cervical spinal cord trauma in an infant.^{10,34,86} Repetitive shaking may also, in some cases display additional stigmata, e.g. bruises to the extremities and body with fractures of the skeletal structures.

Impact, on the other hand, requires the rapid contact of two solid objects, such as a skull and a rigid surface (for example, a table or the floor). This is illustrated for a fall and for a blow by a blunt object on the head in Fig 3. A similar situation prevails if the head, in line with the body, were to strike a rigid surface at an angle to its sagittal plane at speeds of the order of 2–5 m/s. The duration of such contact is very short; in collisions of unbelted vehicular occupants, the contact time with the car component struck was found to be about 3/1000 s or 3 ms. Independent tests by

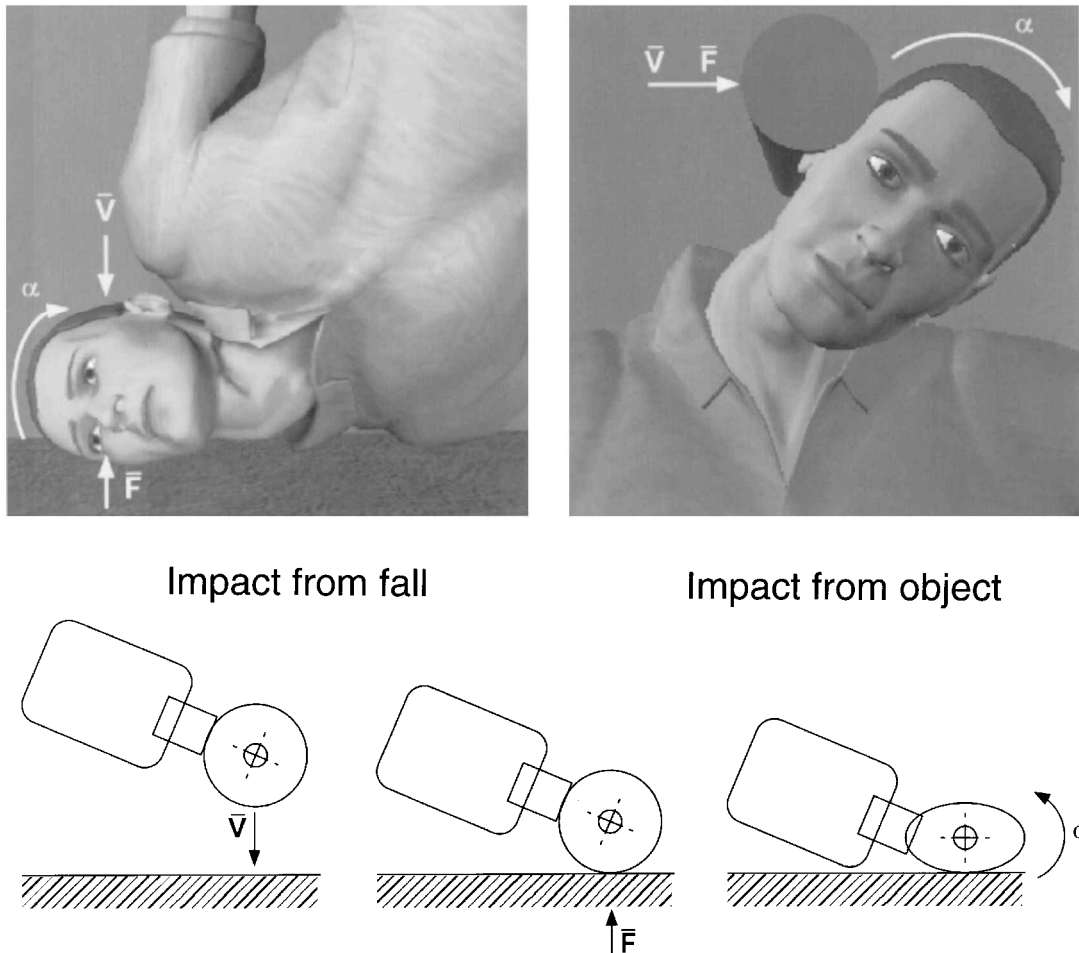


FIG. 3. Impact loading head/neck depicting impact from fall and impact from object.

Goldsmith involving the drop of an adult cadaver skull covered by a replicated scalp and filled with a brain stimulant on a wooden edge at low velocities without skull fracture indicated a contact duration ranging from 3 to 7 ms; a higher impact velocity under these conditions would reduce the contact time slightly. The degree of potential trauma is often gauged by the change in linear and angular velocity produced by the impact, although this does not relate, at least directly, to the stresses generated that are true indices of damage. Gurdjian indicated a limit of 4480 N (1000 lb) as the maximum load that can be applied to an adult head without extreme danger of serious or fatal trauma.³² We have learned since that this is a reasonable approximation for skull fracture in adults and as an index of brain injury without additional description of the loading conditions.

The two consequences of an impact, not present in impulsive loading, are: (a) some type of contact phenomenon, such as indentation, large local deformations, a wide variety of fractures, bruising, etc., and (b) the transmission of loading effects to distal parts of the system, labelled 'wave propagation' whose manifestations often produce damaging effects at locations removed from the contact point, as exemplified by the contre-coup phenomenon. This characteristic includes the generation and release of skull deformations at positions remote from the impact location.^{1,23,32} The specific structure of the interior of the skull plays a significant role in the localization of the coup and contre-coup contusion with and without skull fracture. This has been addressed experimentally in the rhesus model.^{88,90}

The application and consequences of impulsive and impact loading are totally different, and they should not be linked in a single appellation, such as a 'shaken baby/impact syndrome' or 'shaken/impact syndrome' unless evidence for both types of causations are present simultaneously. The use of this dualistic term is a misinterpretation of the Duhaim, Gennarelli, Thibault study because the surrogate 'baby' was first shaken (repeatedly) and did not register loads sufficient to produce injury to the brain. Next, the head of the surrogate was impacted and exceeded the known injury criteria.²¹ The levels of impact loading are from 50 to 100 times greater than the result produced by shaking alone. The impact aspect should be clearly demonstrated by a visible and/or palpable manifestation of the head injury unless the applied force is distributed by a load distributing padding, in which case impact effects may or may not be mitigated depending on the thickness and structure of the pad. Impact, including those due to falls from heights of no greater than 3–4 ft, produce forces an order of magnitude larger than can be generated by impulsive action. A possible *cumulative* effect of

pure repetitive impulsive loading has not been tested in suitable animal models similar to those experiments carried out for impulsive loading analysis.^{90,95}

To demonstrate this point, consider the fall of a toddler through a distance of 0.91 m (3 ft), perhaps representative of his height, by rotation about the bottom of his feet. This results in an impact velocity of 4.24 m/s (13.9 ft/s); a fall of 1.82 m (6 ft) down a set of steps produces a contact speed of 6 m/s (19.7 ft/s). These may be characterized by some as low velocity impacts, but the consequences are potentially highly traumatic: for an extremely conservative value of the contact duration of 0.01 s (10 ms), the peak force generated will be 5970 N for an adult with a head mass of 5 kg falling 1.82 m and 2280 N for a child with a head mass of 2.7 kg falling 0.9 m, respectively. This range of impact velocity can also be considered to be the reasonable levels of maximum impact produced when an infant is manually thrown on to a surface. On rigid surfaces, speeds falls from such heights produce forces that exceed skull fracture tolerance in children and adults.

By contrast, consider the case of shaken baby syndrome (SBS) where a brain mass of 800 g is involved and shaking occurs at the rate of 3 Hz with a total displacement of the infant of 0.6 m (2 ft); this is close to the maximum physically feasible conditions. This pendular motion produces a maximum acceleration of 11 *g*; use of where is Eq (1) eqn 1, which then provides a maximum corresponding force of 86.3 N (19.3 lb); this can be converted to an angular acceleration of about 1000 rad/s². All values are well below thresholds for cerebral concussion, SDH, subarachnoid haemorrhage, deep brain haemorrhages and cortical contusions.²¹ Hence, the fall generates a load about 10 times greater than can be achieved by SBS and in substance can clearly produce these injuries. Thus, while it is possible to produce trauma in an infant by shaking, e.g. a SDH, particularly when shaking is prolonged and repeated at intervals, the injuries would include the cervical cord and spine, but not the brain case, nor contusions in the cerebrum or cerebellum if no impact was also imposed. It is far more likely that impacts due to falls and other causes are more probable in producing TBI by short duration impulsive loading.

Mechanisms of head injury in the adult and child

In a report by McClelland *et al*, the following statement is significant: 'Most severe TBI in children under one year of age is the result of child abuse. Some 10% of all traumatic injuries under five years of age will have a non-accidental cause. Head injury is the leading cause of death in child abuse and of

the 5% of abused children who die, the mortality of the cranial component approaches 30%.⁶⁶ These data also emphasize the non-cranial injuries present in such cases. Understanding the fundamental biomechanics of paediatric TBI compared with adult TBI provides an insight that serves to resolve current controversies, particularly for the diagnosis of intentional versus non-intentional TBI. Our review of historical and current reports on paediatric TBI has also identified five assumptions in the literature concerning the mechanisms of retinal and brain injuries. A critique of these ideas will precede the review of the basic biomechanics and differential data in adults and children. These assumptions are listed below.

Intentional TBI

Child abuse is an increasing problem; between ages 0–4, 2.6 per 100 children are abused and these crimes cost an estimated 22.8 billion US dollars yearly.⁷⁴ The lack of a clear history not fitting the clinical findings is usually the starting point to analyse the available data and fit the most probable mechanism to the injuries. C. J. Hobbs has provided an important caveat stated as follows: ‘The diagnosis of abuse demands absolute certainty to avoid conviction of innocent parents and unwarranted removal of children from their homes. The evidence must satisfy both the needs of clinical diagnosis and the process of law. The two most important corroborative facts in the diagnosis of abuse were the presence of multiple injuries and an inadequate explanation for the injuries.’³⁸ With due Hobbsian caution therefore we cite a set of assumptions in the literature concerning the mechanisms of TBI in paediatric patients that require validation.

1. That infantile head injury mechanisms, in some undefined mathematical manner, could be scaled from animal and adult injury tolerance data, based upon inertial impulsive loading, in the absence of impact.^{10,33}
2. That ‘low’ falls in infants, e.g. less than 4 ft, are not likely to cause skull fractures, acute subdural haematoma (ASDH) and brain injury.^{18–20}
3. That traumatic retinoschisis in abused infants is caused directly by repetitive shaking of the head causing brain motions, which directly produce disruption of bridging veins and ASDH. It is claimed that *similar shaking forces act on the eyes causing the relatively dense lens to move forward and back within the ocular fluids producing retinal haemorrhages by ‘abrupt tugging on the retina’*.^{30,118}
4. That the syndrome of ‘head injured patients who talk and deteriorate or die (the TADD syndrome)’ is not caused by an acute and relatively asymptomatic SDH with minimal initial

sequelae and a lucid interval which can progress to late sequelae such as brain damage (the ‘big black brain’) or to re-bleed in the sub-acute or chronic stage following what retrospectively is found to be a trivial trauma^{16–18,20,40} (personal communication from Dr Duhaime).

5. That the time interval between onset of the cause for intentional TBI injury resulting in a ‘shaken baby syndrome (SBS)’ is always of short duration, i.e. the time for onset of symptoms and signs of severe TBI is always brief and in the absence of a good history, timing of the injury must be extrapolated from data on accidental trauma.^{20,76}

These assumptions individually and in concert are ambiguous or incorrect although they have been used as the bases for differential diagnosis of the mechanisms of paediatric head injuries usually without reference to available biomechanical analysis using reconstruction methods.

In order to discuss the mechanisms of injury to the skull, its coverings and contents there are at least two concepts that must be understood; first, we need to know the behaviour of these materials when quasi-static or dynamic forces are applied and, secondly, we must understand how external forces applied to the system translate into the stresses and strains that will challenge the functional and/or structural integrity of the components of the system. To that end the following concepts are presented:

1. To cause scalp contusion or laceration the stress must exceed the ultimate stress for the capillary network or the composite materials that constitute the matrix and vascular elements of the skin.^{1,23,24,67,126}
2. To fracture the skull the ultimate strength of the bone must be exceeded.^{23,31,32,71,103,114,122–126}
3. To produce subarachnoid, subdural and deep intra-cerebral haemorrhages the vascular elements at various topographic locations must be stretched beyond their ultimate strength.^{1,58,116,120,126}
4. To cause an acute subdural haematoma the parasagittal bridging veins must either stretch beyond their ultimate strength in tension or fail in shear at the cortical surface or at their insertions into the venous sinuses.⁵⁸ Bridging veins can stretch to 30–35% before they tear and bleed. However, pre-loading of such veins at a lower stretch level may result in increased stiffness producing venospasm of varying degrees. This state can result in vein rupture with less force. This could explain why TADD cases with ASDH are often described as having only a ‘trivial trauma’.^{84,93,105,106}
5. To cause cerebral concussion the stresses must reach into the brain stem to produce loss of consciousness; lesser levels of stress will

produce amnesia without loss of consciousness but with a period of amnesic automatic behaviour.^{80,83,85,87,93,94}

- To produce moderate to severe diffuse axonal injury the axons must be stretched beyond their physiological limit or to the point of structural failure. This is most prevalent in the deep white matter such as the corpus callosum, internal capsule, rostral brain stem and basal ganglia. The basic paradigm is that external forces acting upon the head, a complex system of composite materials, will produce stresses and strains that are dictated by the nature, direction and magnitude of the applied loads and the properties of the structure. The overall distribution of stresses and strains resulting in acute lesions imaged by MRI has been documented and fits the centripetal theory discussed earlier.^{26,55-57,83,87,95,112}

Two distinctly different biomechanical systems exist when one views the skull-brain structure of the adult and that of the developing child. The variants in the child, when compared with the average adult, are the constitutive properties of the materials, the geometry of the structures, the age dependent physiological response to mechanical stress and the structural properties of the head. Numerous investigations of the biomechanics of the brain injury in the adult have been conducted. Extensive bibliographical information regarding these studies has been published of which the following references are only representative. Specific injury mechanisms and tolerance criteria for scalp contusion and laceration, skull fracture, diffuse axonal injury, acute subdural haematoma, and deep intracerebral haemorrhage have been advanced.^{23,25,26,32,48,58,64,65,83,84,93,94,96,103,114,116,122-124}

At this point it would be useful to discuss the role of impulsive and impact loading in their association with the various forms of head injury. An impulsive force, resulting in acceleration or deceleration of the head without head contact, is typically applied through the torso and the neck structure, thereby causing the head to be accelerated and decelerated. An example of such a situation would be a restrained occupant of a vehicle that experienced a collision. If the vehicle was stationary and struck from the rear the torso would move with the seat and frame of the vehicle, the head would lag behind and initially rotate backwards due to its inertia and would eventually follow the torso as the neck loads began to produce the necessary forces to accelerate the head. If the vehicle was in motion and impacted an object the torso would be decelerated by the restraint and the head would continue to move forward until arrested by the forces exerted by the neck. Under these conditions there would be no injury to the scalp or skull. This event is similar to the process described as shaking a baby, where the force is applied through the torso, the neck couples

the torso and the head and acceleration and deceleration of the head results. The first structure to experience injury under these conditions is the neck; this is commonly known as whiplash. However, under these conditions the brain is also free to move within the cranial vault and this distortion of its shape can produce stresses within the brain that lead to injury as a function of the magnitude and direction of the induced rotational acceleration, as well as the repetitive nature of this type of whiplash that may or may not be combined with an impact.

Figure 4 compares the magnitude of the rotational acceleration or deceleration necessary to produce the level of shear stress within the brain to cause varying degrees of axonal injury. The figure also demonstrates the theoretical scaling relationship advanced by Holbourn which relates the stress to the mass of the brain and which was experimentally validated in animal models and extended to the human.^{37,39,50,64,65,71,80,81,84,90,96,114} For the adult brain the rotational acceleration required to produce concussion is approximately 4500 radians/s squared and for the infant this value approaches 10,000 radians/s squared. Severe DAI in the adult brain is achieved at rotational accelerations at about 18,000 rad/s, whereas, the neonate tolerance is breached at just below 40,000 rad/s². These data illustrate the inverse 2/3 brain mass rule as explained earlier. Similar arguments are made for acute subdural haematoma and deep intracerebral haemorrhage, but the magnitude of the acceleration for these haemorrhagic lesions is significantly greater than that for mild diffuse axonal injury. At these levels of inertial loading, induced impulsively without contact, the neck torque in the infant would cause severe injury to the high cervical cord and spine long before the onset of cerebral

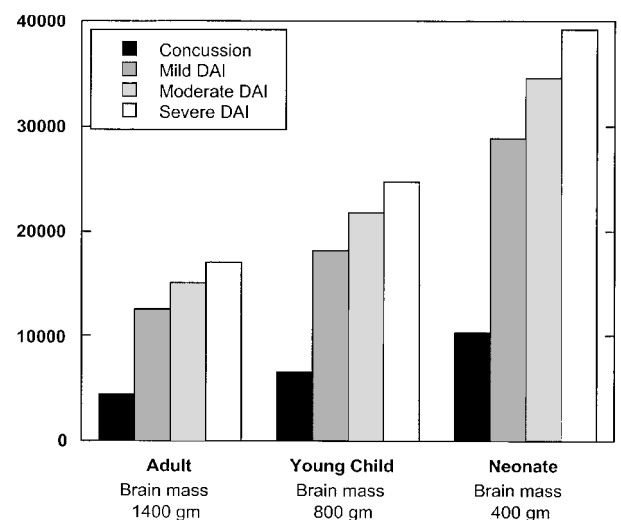


FIG. 4. Brain injury tolerance scaling in the adult, young child and neonate.

concussion.^{25,31} Absence of cervical spinal cord injury would indicate a component of impact in the presence of haemorrhagic brain lesions.

In contrast to impulsive loading, impact contains the elements of inertial loading together with the contact events. Similarly, examples include the head contacting the ground as the result of a fall where the scalp compresses, the skull bends inward, stress waves propagate around the surface of the skull and through the brain material (Fig. 3 — impact from fall). These events occur with a rise time constant of the order of 100 microseconds and before the head has accelerated or decelerated to its maximum value; each event can produce failure of the materials that constitute the head structure. As the scalp is compressed by the impact, the stresses which are related to the magnitude of the force or the impact velocity, the direction of the blow, and the contact area and properties of the impacting surface all play a significant role. (Fig. 4 — impact from object) Following the contact phase the head is accelerated or decelerated giving rise to differential motion of the skull and brain. During the impact associated with a fall the skull will stop and the brain will continue to move within the cranium. This relative motion can lead to stresses which associate with the kinematical conditions. In pure translation tissue experiences compression and tension while in the angular deceleration mode the shear stresses can be very high compared with the failure criteria. The brain is a virtually incompressible material (bulk modulus = 300,000 pounds per square inch); in contrast the shear modulus is of the order of 10 pounds per square inch.⁸¹ Therefore, velocity gradients within the brain will result in shear stresses, which can exceed failure criteria for DAI, ASDH, ASH and deep intracerebral haemorrhage.

The contact loads can produce focal injury in the absence of inertial effects. For example, skull fracture will result when the ultimate strength of the bone is exceeded. Figure 1 demonstrates the stresses necessary to fracture the skull of the adult, young child and the infant. The requisite forces are reduced as a function of the skull thickness and the moduli of the bone. Weber¹²²⁻¹²⁴ provided experimental confirmation of the presence of skull fracture in each of 15 (average age 8.2 months) perfused infant cadavers. Three groups of five cadavers were dropped on stone or tile, carpeted and linoleum covered floor. The fall heights were consistently 82 cm with the cadaver in a horizontal position with head strike at the parieto-occipital zone. This produced an impact velocity of approximately 4 m/s (13 ft/s). Fractures were found in every case.

Weber also investigated whether a skull fracture could occur in unobserved falls and not be recognized by lay care takers. On the basis of his forensic

pathology expertise in fatal cases and his review of clinical reports of falls from parent's arms, tables, strollers, beds and sofas, Weber reports that linear skull fracture does occur in low falls without specific symptoms. He concluded that such asymptomatic linear skull fractures can occur and not be recognized by lay caretakers, as well as by professional staff. It should be noted that prior to Weber's work, no systematic study of low level falls and fracture mechanism in infant cadavers had been carried out. Weber also emphasizes that his experimental results should not be used to exclude wrong doing by parents and guardians. His results only demonstrate the possibility that such an injury (specifically a linear fracture) from low heights are possible. Weber states, 'In all these cases one has to consider the whole situation. The so far suspected vulnerability of the infants' skull when falls occur from low heights have been confirmed as being possible by our experimental results.' In a second paper Weber also reports that only *one* out of 10 cadavers dropped on 2 cm thick rubber foam covered floor and four out of 25 drops on folded (8 cm thickness) camel hair blanket which produced skull fractures. The bone thickness in these cadavers was 0.1–0.4 mm adjacent to the fracture fissures.¹²³ Weber's third report was an attempt to predict the risk of skull fracture in infants (neonates to 14-month-old infants) using diaphanoscopy to locate skull transparency indicating loci of paper thin vulnerable zones in such skulls.¹²⁴

Impact loading, as previously described, can result in stresses and associated deformations and strains that exceed adult human tolerance levels, as well as those in the child, but the weaker infant and child skulls fail at much lower levels as shown in Fig. 2 This failure is preceded by deformation of the brain with or without skull fractures. Impulsive loading on the other hand, resulting in purely inertial forces without contact, are also implicated in several important forms of brain injury in the adult. In contrast, the level of the forces necessary to produce equivalent stresses in the child are far in excess of those that can be reasonably achieved without significant injury to the cervical spinal cord and neck structures. Repetitive and prolonged episodes of shaking of the infants, as well as the adult head/neck system may produce cumulative stresses in the brain resulting in subdural haematomas and abnormally elevated intracerebral pressure. This hypothesis has not been adequately tested in animal models for TBI mechanisms as noted above. Figure 4 depicts brain injury tolerance in adults, young children and neonates.

Clinical data on paediatric TBI

The biomechanical findings described above are well validated by clinical data. One of the most extensive

studies on paediatric head trauma was reported by Berney *et al.* in 1994.⁸ This was based on a consecutive, unselected series of 1812 cases of TBI in children less than 15 years old. Three categories were established by age: 0–3, 3–9 and 9–15 years. Their main conclusion was that 184 children in the 0–3-year-old category sustained low energy trauma, suffering more skull fractures, more subdural haematomas and early seizures as compared with the other two groups. The low energy level was described as falling off toboggans and equivalent low level falls, i.e. less than 3–4 ft. Children between 3–9 had rather higher energy incidents, were more often in coma and did not have subdural haematomas, but did have significant brain swelling. Children between 9 and 15 were more like young adults with more severe TBI than the previous two categories. They suffered more with extradural haematomas as compared with subdural bleeding. One of the authors' conclusions was 'the smaller the child, the lower the energy involved in the injury'.⁸ These clinical data on impact injury mechanisms indicate the role of the specific biomechanical properties of baby skulls, which result in significant deformation of the skull and brain shape enabling the injuries to occur at lower levels of impact to the head and therefore the brain. All of the three age categories of children included cases of 'talk and die or deteriorate' (TADD) syndromes for each grade of energy. There were five times more TADD cases with skull fracture as compared with the non-skull fracture cases and they constituted 25% of all the ASDH cases, most of which were in the 0–3-year-old category.⁸

In 1993, Rivara *et al.* published a population based study of fall related injuries in children and adolescents resulting in hospitalization or death based on all hospital discharges for 1989 and 1990. The most common type of fall resulting in hospitalization was a relatively low fall from one level to another, e.g. from beds, tables, chairs and playground equipment (37.5% of all falls). Head injuries associated with 'skull fractures, concussion and intracerebral haemorrhages occurred to more than one-fifth of the children. This was especially common among pre-school age children in whom 42.1% had head trauma compared with only 14.4% of adolescent aged 15 through 19 years.¹⁰⁰

A prospective study by Luersen *et al.* in 1988 reviewed the outcome after TBI related to age (paediatric and adult). The authors had 8814 patients of which 1906 were children younger than 14. In infants less than 2 years old, 65% had non-fatal TBI caused by falls, 20% by motor vehicle crashes and 5% by assaults. Fatal TBI at this age were caused in 20% by assault, 40% by motor vehicle crashes and 40% by falls.⁵⁹

A retrospective study by Howard *et al.* in 1993 reviewed 100 infants who suffered TBI of which 28 also had subdural haematomas. Abuse was initially

suspected in 14 of the 28 cases and investigations led to legal actions in 10 of the 14 cases. An unusual finding in this report was an apparent increased susceptibility to the development of subdural haematomas in non-Caucasian infants. Of the 28 TBI and subdural haematomas, 17 were Caucasians and 10 were non-Caucasians with one mixed-race infant. Given the overwhelmingly Caucasian population base, the number of non-Caucasian ASDH injuries was significant at 62 versus 21% for the Caucasian group ($p < 0.01$). All of the 28 cases with ASDH had well described history and evidence of head impact. Retinal haemorrhages were present in 20% of these infants. The non-Caucasian infants developed ASDH after trivial falls and with normal scalp examination as compared with Caucasian infants. The conclusion of this study was that intentional injuries were less common than unintentional TBI and shaking as the only cause of SDH was not supported.⁴⁰ The unusual vulnerability noted in non-Caucasian head injured children was also related to other published observations in Japan. Falls from sitting or standing positions were described to be the cause of ASDH and retinal haemorrhages in 26 Japanese infants as reported by Aoki & Masuzawa.² All were infants with acute SDH apparently due to minor head trauma without LOC and no contusions of the brain. Presentation was mostly by seizures following other minor TBI. Average age was 5.1 months and 23 were boys. All had retinal and preretinal haemorrhages. Two children died, mild retardation was found in one and epilepsy in one with the remaining 23 showing normal development. Early surgical treatment to remove the SDH clots was standard and in another case (not included in this series) the SDH was not removed because it was considered as trivial. Forty days later severe damage with multicystic encephalomalacia was confirmed.

Greenes & Schutz have recently reported a retrospective review of 101 infants less than 2 years old who were admitted for suspected TBI to a tertiary care paediatric hospital over the course of 6½ years. Nineteen of these infants, all less than 1 year old, were asymptomatic but harbouring 'occult' intracranial injuries. They had no loss of consciousness, no mental or behavioural changes, no seizures, irritability or vomiting, without retinal haemorrhages or a bulging fontanelle, and with a normal neurological examination. No history of trauma was given or witnessed for two infants. Ten suffered falls ranging from 2 to 5 feet, four fell down five to nine stair steps, one suffered from a motor vehicle crash and one from child abuse. All were alert and five were playful. Subdural haematomas were described as 'small' in seven with mild shift of midline and maximal 1-cm width in one of these. Six had epidural haematomas with

1 cm width and shift in two cases. One had both subdural and epidural haematomas. Eighteen of this group had scalp contusion or haematomas and 18 had skull fractures. No intervention other than anticonvulsants were provided in nine infants. All infants experienced no serious neurological deterioration except one subject had a brief self-limited seizure. The finding of 18 cases out of 19 harbouring 'occult' intracranial injuries also had skull fractures supports the experimental data of Weber.¹²³

The authors of the report state that none of these infants required subsequent surgery or a significant degree of medical management. Specifically, they noted that 'the clinical significance of occult TBI in these patients is not entirely clear'. They also note that 'our confidence intervals are consistent with a rate of late clinical complications in these patients that may be as great as 28%'. They also realized that some TBI patients were not admitted and discharged from the ER without a diagnosis and no follow-up. A prospective study of all infants with TBI entering the ER with a uniform protocol would provide improved data to decide which infants required brain imaging and skull radiograph and a standard follow-up letter to the parents after discharge may uncover late complications such as development of acute re-bleeding in an occult subdural haematoma in a case of the TADD syndrome. What is unequivocal in this report is the conclusion that 'physicians cannot depend on the absence of clinical signs of brain injury to exclude intracranial injury in infants younger than one year of age.'²⁹

An extensive review on the epidemiology and a etiology of infant TBI by De Rocco & Velardi is found in the text book titled: 'Head Injuries in the Infant and Newborn' by Raimondi, Choux & Di Rocco.^{15,98} Data on two large series totalling 13,452 infants in the first 2 years of life were analysed. Most of the children who suffered head injuries were at home or in familiar surroundings. Falls were the dominant mechanism and occurred from cots, cribs, prams, high chairs and down steps. Abuse, motor vehicle injuries and falls from heights were the other mechanisms. Compared with non-intentional aetiology, intentional TBI was relatively rare in this age group.¹⁵ In a second series by Canestri & Monzali of 5363 children up to 3 years of age, 3% were found to have suffered TBI. The majority of skull fractures (and evidence for increased intracranial pressure) occurred in infants between 9 and 17 months with the highest peak at 9 months. Falls were found to be a major cause of TBI and were grouped in two categories: falls from cots, cribs, prams, high chairs, often due to insufficient supervision, and falls against or from furniture and down steps (related to independent ambulation). In most cases the fall was from a

height of about 90 cm or less (about 35 inches). Most of these falls did not result in observable injuries.¹¹ Infant 'Walkers' are known to be hazardous; this device caused TBI and skull fractures in 47 of 150 infants (10.6%) as described by Kavanaugh & Banco.⁴⁵

Child abuse

Di Rocco & Velardi in their report mentioned above also reviewed intentional TBI in the context of all causes, confirming the main incidence of abuse in children under 2 years of age. They compared US data from New York and Illinois Central Registration and published data from five other US sources for a total of 705 cases along with 1719 cases observed at the Hôpital de la Timone, Marseille, France. The US data revealed a peak incidence between 9 and 12 months of age. The French data compared the relative incidence of four common aetiologies of infant and child TBI, i.e. falls, road accidents, abuse and other causes, for five categories of age. Between 0 and 2 months of age, falls were the cause in 79% of this group. Between the ages of 2 and 6 months road accidents contributed 8% of causation. From 6 to 12 months, child abuse accounted for 2% of the 1719 cohort. From 12 to 24 months, other non-specified causes were noted in 11% of the cases.¹⁵ These data are generally in agreement with the data shown in Table I and re-analysed by Thurman from the National Center for Injury Prevention and Control (NCIPC) of the CDC.¹¹⁸ The availability of CT and MRI has significantly improved the accuracy of diagnosis in TBI of all types but the biomechanical aspects of causation are not fully integrated into the nosologic skills of medical practitioners. As demonstrated by Weber, if a fall or blow to the head in infants is cushioned by a load distributing material, a range of brain lesions, including subdural and subarachnoid bleeding, intraparenchymal haemorrhages, parenchymal swelling and brain lacerations can occur without apparent external evidence of head injury. This result is due to the infant's easily deformed skull causing deformation and strains in the brain, and its vascular components as discussed in the section on biomechanics. Recent reviews and reports of intentional TBI and the outcome have provided important data on clinical and radiological information of this tragic source of brain injuries and fatalities.^{5,15-19,45,74} Two aspects, however, have not been fully clarified; the mechanism of rebleeding in subdural haematomas and the biomechanics of retinal haemorrhages.

Rebleeding in the subdural haematoma (SDH)

James Barkovick in his book *Pediatric Neuro-imaging* (1995) provides a definitive statement on paediatric and adult neuroradiology. He states that the radio-

logical images (CT and MRI) of SDH in infants and children in all its states are the same as seen in adult cases with SDH, both in its acute and chronic states.⁵ Three important consequences follow. First, there are no special neuro-imaging phenomena of SDH unique to the patient's age, e.g. in the density changes over time, clot retractions, onset of rebleeding in subacute and chronic SDH, mixing with CSF, and the phenomena of 'disappearing' SDH, which has been described in paediatric cases by Duhaime.¹⁸

A quantitative study by Mats Bergstrom *et al.* from the Karolinska Hospital on the phenomenology of rebleeding in 41 cases of SDH and nine cases of extradural haematomas (EDH) sheds useful light upon what is a somewhat obscure syndrome when autopsy data are not available.⁷ Serial CT scans of the brain were analysed to plot the Hounsfield Units of each scan versus the time lapse from the time of the trauma to 4 weeks post-trauma.^{84,93} Bergstrom *et al.*'s histories of these patients did not provide any evidence of 'new traumas in association with onset of symptoms. Spontaneous rebleeding may well explain the onset of symptoms as well as the attenuation values being so much higher than those of CSF and serum. It is unlikely that a sudden onset of symptoms a long time after trauma would be explained by slow continuous enlargement of a hematoma.' Finally, this report also indicates that a rapidly expanding rebleed within a relatively small sub-acute or chronic SDH caused by low level trauma with no serious neurological disturbances *ab initio* could be the most probable explanation for the sudden onset of more serious signs and symptoms requiring surgery due to a re-bleed in the SDH of a patient who was not initially a candidate for surgery at the first onset.⁷

Confirming the age of the SDH and its rebleeding potential

Apart from the standard requirement to save SDH clots for pathologic reports, recent quantitative investigations have emphasized the importance of saving the SDH during surgical removal of the clot in order to correlate the concentration of fibrinogen and fibrin monomer with the preoperative CT findings. Nomura *et al.* have classified 41 SDH cases into five categories based on the preoperative CT scans and correlated these images with the concentration of fibrinogen, which indicates rebleeding and fibrin monomer, which indicates coagulation activity using sodium dodecyl sulphate-polyacrylamide gel electrophoresis and immunoblotting. The five categories of CT images were as follows: *Layering type* SDH hyperfibrinolytically active with a highest tendency to rebleed. The *mixed density* type has also a high tendency to rebleed, but with lower hyperfi-

brinolytic activity. The *lower density* and *isodense* SDH had very low tendency to rebleed and low fibrinolytic activity. High density SDH are usually acute and subacute cases. In suspected child abuse and fatal cases, detailed histological studies of the clot and adjacent dura are obviously essential to determine the age of the haematoma. In SDH survivors, the clot is also an important component of the definitive diagnosis of causation when correlated with the CT findings.⁷⁷

Hyperacute subdural haematomas

These very acute haematomas are mixed density subdural haematomas, which develop rapidly under two conditions that were first described by Greenberg *et al.* in 1985.²⁸ These conditions are two-fold as follows:

1. When the haemorrhage is an actively bleeding source, either from a number of bridging veins or, less commonly, from an arterial source.
2. When a coagulopathy is present. The differential diagnosis of this type of SDH by *CT findings alone* can mislead and indeed may suggest a rebleed in a subacute or chronic SDH. Surgical exploration or autopsy is necessary to prove the diagnosis of the hyper-acute nature of this SDH. A recent forensic report on a 15-month-old infant who died after intentional TBI initially presented with CT showing a mixed density SDH. This was interpreted as caused by rebleeding in an older SDH. Surgical exploration, however, discovered active bleeding and postmortem histological findings confirmed this to be a hyperacute SDH caused by child abuse. Confirmation of the nature of an SDH requires careful pathological examination of the haematoma, as well as a careful search for other stigmata, particularly soft tissue injury and fractures associated with the aetiology of intentional TBI. Soft tissue injuries have been found in 92% of 371 children who were abused.¹⁰⁴

An important radiological clue for diagnosis of a rebleed occurs when a clear interface of an SDH is found separating an inner hyperdense and an outer hypodense SDH components on CT. In such a case there is good reason to assume that this appearance is the result of a rebleed in a subacute or chronic SDH. This was documented in a 1983 report in the *Journal of Neurosurgery* by Kao who reviewed 140 patients with 'chronic' SDH. Only seven patients presented with the CT findings of two components as described above. All but one of these seven cases had a definite history of head trauma more than 1 month before they developed acute disturbances and profound hemiparesis. This group was compared with seven chronic subdural haematoma patients who had only mild clinical signs and

symptoms. They were confined in bed for a period before CT and craniotomy. The CT scans in this group did not show the interface, and the layering of low and high density SDH as described in the first group. The parsimonious conclusion was that a rebleeding into a chronic SDH which was sufficiently old so as to be seen as homogeneous prior to the rebleed is real. The author suggests that a sufficient period of being in bed with the brow up before CT examination causes sedimentation in the SDH, which is seen in a small proportion of patients but may be specific for a significant amount of rebleeding to produce acute deterioration.⁴⁴

Biomechanics of retinal haemorrhages

Although retinal haemorrhages are found in children and adults with severe CNS trauma, they are not pathognomonic for intentional TBI. The haemorrhage can be unilateral or bilateral, and may involve all the layers of the retina, as well as the choroid and vitreous. Deeper haemorrhages may be well circumscribed and have a dome shape giving rise to the appearance of retinoschisis.^{27,30,51} Between 1964 and 1967 there were at least 13 publications concerning the hypotheses for the pathogenesis of retinal and optic sheath haemorrhages associated with intracranial bleeding.⁷⁵ The consensus of 10 out of the 13 concluded that the mechanism was either increased venous pressure, sudden rise of intracranial pressure (acting on orbital veins) or passage of blood through intracranial subarachnoid space with such force that intradural haemorrhages occur. The central retinal vein and its choroidal branches collapse due to the rise of pressure (Manschott). Walsh & Cogan also favoured this hypothesis.^{12,121} The alternative hypothesis is *retinoschisis* caused by the whiplash effects on the head moving the brain back and forth, tearing veins and causing SDH, while the eyes similarly 'shake' within the orbits causing the dense lens to move back and forth, stressing the connections between the lens, vitreous gel and the retina, and especially the macular region resulting in retinoschisis.³⁰ Two recent publications are pertinent to the validity of the 'rapid rise of ICP' hypothesis. First, the human optic nerve sheath has sufficient elasticity to allow dilation in response to a rapid rise of intracranial pressure (ICP), particularly when the TBI is severe.³⁵ A second report on 'Head injury: cause of both brain edema and retinal hemorrhages' was presented at the 1995 meeting of the National Association of Medical Examiners. This study was a prospective, post mortem study of 169 child deaths. Brain oedema was found in 106 cases and 63 cases had no oedema. Retinal haemorrhages were found in 68 of the 106 cases with oedema and only in two cases in the 63 which had no oedema. TBI was the confounding variable, i.e. 61 of the 68 cases with retinal haemorrhages died from TBI with oedema as

the cause of death. The conclusion was that TBI caused both the brain oedema and retinal haemorrhages and the increased ICP related to increased brain volume and therefore is most probably the cause of retinal haemorrhages.²² Moreover, Dr Andrea Cibis Tongue in a critique of the Greenwald *et al.*'s retinoschisis hypothesis stated that it was 'unlikely that the basic lesion is one of retinoschisis rather than haemorrhage secondary to increased venous pressure.' This author also stated that the vascular system in infants may be more fragile and susceptible to abrupt pressure changes.¹¹⁸ Green *et al.* have more recently also supported the hypothesis of intra-ocular mechanically-induced retinoschisis. Noting the direct relation of retinal haemorrhages to the presence of severe intracerebral and ocular injuries these authors conclude that increasing trauma severity 'invokes a mechanism similar to that causing subdural hematoma' by the effect of 'inertia/momentum of the vitreous body within the eye during shaking'. They further conclude that 'the mass of the vitreous (which forms a large proportion of the mass of the globe) acts upon the points at which it is attached during both acceleration, with up/down, left/right, axial and rotational movements, producing first small haemorrhages in the retina (intra-retinal and subhyaloid haemorrhage), then retinal detachment (directly, with haemorrhage having adaptive effect. Optic nerve sheath haemorrhage is also explained by this type of mechanics, i.e. 'the result of angular, rotational or axial movement about a point in the most anterior part of the optic nerve, posterior to the sclera'.²⁷

The hypothesis of 'intra-ocular' retinal haemorrhages caused by orbital shaking has not been tested experimentally. The eye has a relatively very small mass compared to the brain. Biomechanical calculations based on Holbourn's inverse 2/3 rule, previously discussed, predict that, smaller masses require higher levels of force to cause damage and larger masses require less; the levels of force required for retinal bleeding by shaking to damage the eye directly is biomechanically improbable. The work of Hansen & Helmke also indicates that the role of sudden rise of ICP is more likely to cause bleeding than the 'shaken eye' hypothesis. Most probably the latter is not a testable hypothesis. By exclusion therefore, the sudden rise of ICP secondary to TBI is a valid (and testable) hypothesis.^{12,35,75,118}

Principles of biomechanical reconstruction of human TBI

Application of existing biomechanical methods for in-depth reconstruction of adult and paediatric TBI are currently available and have been applied for at least two decades, primarily in the field of protection and prevention of humans in motor vehicle crashes and preventing injuries in hazardous environments,

such as playgrounds. The overwhelming bulk of this literature is published in governmental reports and journals in the fields of engineering and biomechanics. The best of these reports are developed by multidisciplinary teams including physicians, engineers and pathologists. Injuries to all parts of the body, including the head and neck are related to the causative forces utilizing the Trauma score, Abbreviated Injury Scale and the Glasgow Coma Score. For motor vehicle crashes, sources of injuries are correlated with the vehicle damage and crash circumstances. *Post hoc* reconstructions based on accident reports and medical records (even with inspection of the crash vehicles) are less informative than in-depth reconstructions closer to the time of the injury onset.

A clinical trauma nurse in the Trauma Unit and an injury reconstruction team are on standby which provide more accurate data. With the support of the National Highway Traffic Safety Administration and in collaboration with H. Champion we were able to develop for the first time an ongoing, in-depth re-construction programme for head and neck injuries. A trained nurse clinician notified the reconstruction team when the patient was admitted and the reconstruction team immediately proceeds to the crash site to gather the initial injury source data.

This strategy enables maximal data capture for correlation of the mechanics of the vehicle crash with the medical diagnoses and biomechanics of the injuries to the patient. Weekly rounds to review and merge the two data sets, biomechanical and clinical, provided the raw data base for reconstruction of the crash and the injury mechanism, as well as recording the outcome of the patient. Between 1982 and 1986, this methodology was implemented at the Shock Trauma Unit at the Washington Hospital Center. Two-hundred-and-forty-six cases were identified and 101 of these patients with head and/or neck injuries were completely studied including autopsy reports, X-ray and CT reports. Further analysis of cases in which the most complete crash and injury data sets were available, enabled physical and mathematical reconstructions, which were tested using anthropomorphic dummies seated in identical motor vehicles to reproduce the crash sequence at a sled test facility. The anatomically appropriate anthropomorphic dummies reproduced the quantitative data confirming the biomechanics of the specific injuries to the head and neck.⁷⁹ Extensive data of this type continues to be collected for injury prevention and protection purposes.^{43,52,60-63,69,70,72} Biomechanical reconstruction of data in free falls have also been obtained based on the pioneering work of DeHaven and subsequently by the work of Cummins & Potter and of Snyder *et al.*^{14,13,111} More

recently, methods for TBI reconstruction have also become available.^{3,41,60,70,101,108,119}

A research program featuring teams consisting of neurosurgeons, biomechanical engineers and adequate staff would be able to conduct in-depth reconstructions of all types of TBI injuries, including motor vehicle crashes, and falls to establish a more comprehensive data base of reconstructed TBI cases. Categories of severity will be based on a synthesis of the biochemical data, as well as the clinical, radiological and biomechanical data. In addition to CT, early stage MRI would provide more details of the acute TBI injury. This data set of biomechanically driven categorization of TBI cases, as well as the outcomes of these patients would serve as the template for predicting the vulnerability and outcome of subsequent patients of similar age, sex, mechanism and approximately equivalent levels of force under current TBI protocols. A recent report has been presented on the use of serum markers, e.g. neuron-specific enolase, creatine kinase BB, myelin basic protein and S-100 as biochemical indices for outcome predictions in TBI, as well as for stroke and subarachnoid bleeds. S-100 appears to be a promising marker in a study on 30 patients with TBI of high severity (GCS < 9) and 11 patients with minor TBI (GCS > 12). Blood samples were taken 6 h after injury and after defining the outcome at discharge, which was measured by the GOS. All the minor TBI had favourable outcomes (mean S-100 + 0.35 mg/l) at discharge. The severe TBI cases fell into two groups; 31% had an unfavourable outcome with a mean S-100 level of 4.9 mg/l, while the remainder of 69% had a favourable outcome with a mean serum level of S-100 at 1.2 mg/l. All the groups differed significantly ($P < 0.05$). This work indicates a potential biochemical measure, which can be correlated with the biomechanical severity.¹⁰²

A recent comprehensive review on TBI by Teasdale & Graham on the comparison of novel therapeutic agents for controlling the secondary progressive brain damage in animals and human TBI was a major stimulus for writing our report.¹¹³ The authors comment on the paradoxical result of the novel experimental treatments in two categories of these subjects which were indeed puzzling. These new agents were proven to be very effective in the animal models of TBI, but failed to achieve similar benefits in TBI patients. The most probable explanation of this paradox may depend on the fact that there are at least two confounding variables, which are present in human cases of TBI. First, there is the relatively uniform population of the animal subjects compared with the relatively genetically variegated humans as suggested by Teasdale & Graham.¹¹³ Secondly, the levels of force causing the TBI in the animal models is also uniform,

indicative of the controlling role of biomechanics causing the experimental TBI. Two uncontrolled variables exist in the human TBI research. Seeking to control secondary injuries in human TBI is significantly handicapped by biomechanically incomplete stratification whereas both of these variables are well controlled in the animal models of TBI. It follows therefore that establishing a research programme built upon a biomechanically reconstructed stratification of TBI patients receiving equivalent levels of mechanical forces, similar mechanisms, and with approximation for age, sex and other biological variables, could significantly improve the therapeutic effects of novel therapies for controlling the secondary progressive damage to the brain. The use of biological markers such as the S-100 serum factor could also be of additional value in reducing the levels of human variability.

Finally, such a programme based on biomechanics integrated with biomedical data would also serve to solve the outstanding problem of currently inadequate National and International standards for Head Injury Criteria and definition of Head Injury Tolerance. The Federal Government, and several private and industrial agencies have prescribed standards concerning certain protective devices and other modules relating to vehicular impacts, which must be satisfied in order to be accepted. One of the major dictums has involved the stipulation of peak values of *linear* head acceleration and corresponding maximum durations for which these values could be sustained. It must be emphasized that no correlation has been established between these parameters and head trauma; furthermore, such linear acceleration limits have not been related to stresses and strains that can be compared with observed cerebral damage. It should also be mentioned that the known potential damage of angular motion is not considered in these standards. These criteria use unacceptable parameters from an engineering viewpoint to correlate with trauma. It is strongly recommended that head injury criteria be established that follow the standard practice of engineering by relating conditions to levels of stress, strain, or absorbed energy.^{23-25,31,65-67,79,101,107,108,111}

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Appendix: the physical basis of head injury biomechanics

Biomechanical behaviour of tissues upon load application

The deformation characteristics of all materials, including the tissues of the head, are determined experimentally under both slow (static) and dynamic loading. In this presentation, the loads and deformations will be restricted to two-dimensional considerations; this simplifies the description by avoiding the use of vector notation. The descriptions can be readily extended to three dimensions, but this will lead to some complications in concepts and mathematical expressions, particularly in the case of simultaneous rotations.

The application of force F is expressed in terms of engineering stress $\sigma = F/A_0$ where A_0 is the original cross-sectional area; the force is assumed to be distributed uniformly over the section. When compressive stresses are distributed uniformly over an entire body, this object is said to be in hydrostatic compression. Deformation is characterized by an engineering strain $\epsilon = (L - L_0)/L_0 = \Delta L/L_0$ which represents the change of length divided by the original length for extension or compression, and by an angle in case of rotational changes. Two-dimensional deformations are depicted in Fig. A (a-e). The cases of tension (T), where an object

tends to elongation, compression (C), where the element tends to contract, bending, shearing and torsion are depicted in Fig A(a) and A(b) under conditions of uniaxial loading. Bending is the result of a force applied in a direction not coincident with the central axis of the unit, resulting in the application of a moment $M = Fd$ [Fig. A(c)], where d is the perpendicular distance from the line of action of the force to the axis about which bending occurs. Simple shear [Fig. A(d)] is generated when two equal and parallel forces F generate angular deformation, while torsion is produced by a force acting perpendicular to the axis of, say, a circular bar at a distance r from the centerline; the effect of this action is the torque $\bar{T} = r\bar{F}$, which produces a twisting of the elements of the bar [Fig. A(e)]. Several of these actions can occur simultaneously.

The behaviour of a material is mechanically defined by the relation between stress, strain and, if important, by the rate of load application. This relation is called a constitutive equation and its characterization requires experimental measurement of the load-deformation characteristics. Many substances exhibit regions where the deformation occurs instantly upon load application and simultaneously disappears completely (or nearly so) upon force removal, representing reversible behaviour. Such a material is called *elastic* and its fabric is restored to its original condition upon unloading. Some materials stressed beyond the critical value, called the elastic limit, will exhibit permanent (or plastic) deformation resulting from irrevocable arrangement of the microstructure. However, materials permanently deformed may exhibit a measure of residual elasticity and could recover some of the deformation by a process called deformation.

When the response of the material depends significantly on the rate of load, stress or strain application, the solid is labelled as a visco-elastic substance. Furthermore, many substances, particularly biological at components, do not exhibit uniform properties either in various directions at a given location, or in moving from location to location. The first characteristic is called anisotropy, while the second is labelled inhomogeneity. In the presence of either of these complications, proper material description requires the determination of the stress-strain relation both in terms of position and direction to permit appropriate modeling of the loading and unloading response.

Failure of a solid element can occur by fracture or rupture that may take place in a brittle manner, or be preceded by permanent (plastic) changes of shape. It may also take place as the result of excessive deformation; physiological dysfunction most frequently precedes mechanical disruption. Failure limits for most engineering and many stable biological materials have been exhaustively tabulated.^{1, 24}

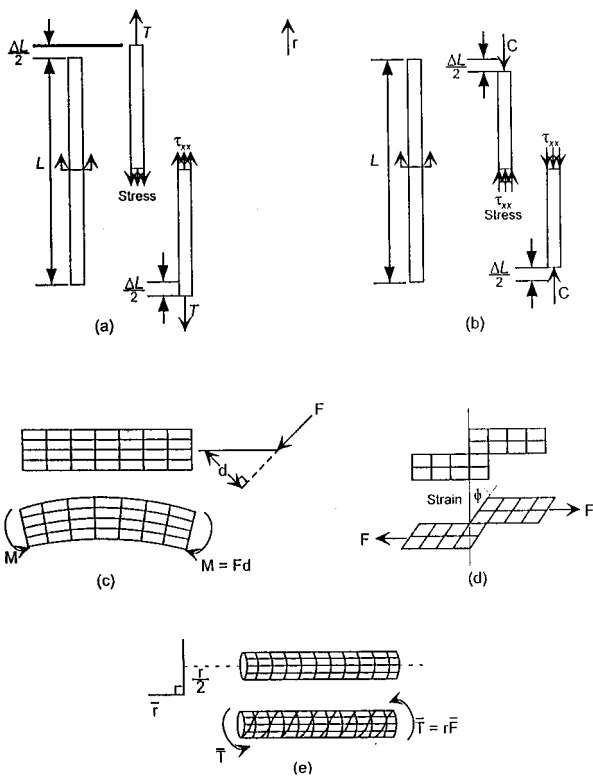


FIG. A. Uniaxial loading, stresses and deformation of a structural member. (a) Tension, (b) Compression, (c) Bending, (d) Simple shear, (e) Torsion.

31, 58, 120, 126 Although some data have been obtained on the variation on mechanical properties with age, as for the skull of a neonate, child and adult,¹¹⁴ there is an urgent need for additional information on this subject.

Motion characteristics of objects (Kinematics)²³

It must be emphasized that biomechanics is an engineering discipline and, as such, employs terminology and concepts compatible with the principles of mechanics. An object may be considered as a particle, an entity of negligible dimensions compared to the path through which it travels. Its resistance to changes in motion is characterized by its mass $m = W/g$, where W is its weight and g the acceleration of gravity, $9.81 \text{ m/s}^2 = 32.2 \text{ ft/s}^2$ on the earth. The kinematics of a particle are quantified by its displacement r , defined as the linear distance and direction from its initial position O to its final location P ; by its velocity, which is the time rate of change of position, i.e. $v = dx/dt$, and by its acceleration $a = dv/dt = d^2r/dt^2$, the time rate of change of its velocity. Movement may occur in three dimensions, characterized, for example, by a rectangular coordinate system with orthogonal axes along x , y and z , and an origin O , as shown in Fig. B(a). When this motion is confined to two dimensions, say x and y , it is called planar motion [Fig. B(b)] and when it is directed along a single line, say the x -axis, it is termed rectilinear motion [Fig. B(c)]. It should be noted that, in general, the displacement r is not the same as the distance travelled; this occurs only in the case of uniaxial motion when there has been no reversal of the movement.

When the dimensions of the object are comparable or large relative to the displacements it executes, the system can no longer be regarded as a particle, but must be described as a rigid or deformable body. Rigidity can be assumed if the deformations resulting from the motion can be neglected, such as either the upper or lower arm when rotated. Even when significant deformations occur, such as brain movement during rapid acceleration of the head, the skull itself may be approximated for certain purposes as a rigid object, whereas the brain must always be regarded as deformable in the analysis of trauma. However, in view of the peculiar nature of the paediatric skull, it should frequently also be considered as non-rigid. Most biomechanical situations, particularly those involving the head/neck complex, require treatment of the anatomical components as bodies of finite dimensions.

A rigid body can execute all the movements of a particle in up to three dimensions; this is called the translational component of the object's motion. The displacements, velocities and accelerations of all particles of the object are by definition the same

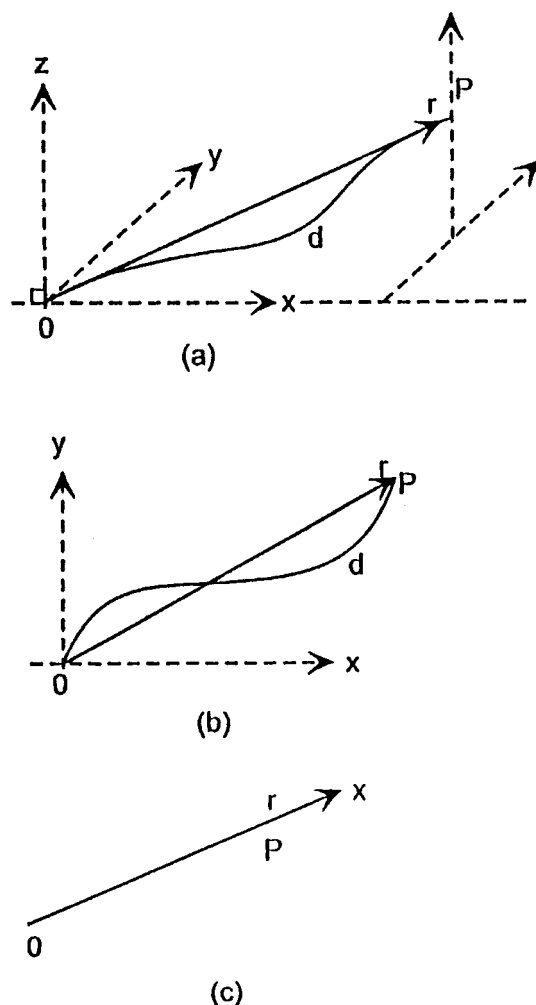


FIG. B. Displacement of an object considered as a particle. (a) In three dimensions, (b) In two dimensions [planar], (c) In one dimension [rectilinear]. In general, the distance travelled, d , differs from displacement r .

in this motion, although the reference point for their quantification is usually chosen as the centre of gravity or centre of mass G . However, in addition, such a body can either independently or simultaneously execute rotation, which consists of instantaneous circular motion of all particles of the object about a given axis, as shown in Fig. C (a-c).

The angular displacement is denoted by θ , the angular velocity by $\omega = d\theta/dt$, and the angular acceleration $\alpha = d\omega/dt = d^2\theta/dt^2$, relations that are completely analogous to the case of linear motion.

It should be emphasized that, at any instant of time, there exists a single angular velocity ω about some axis and a single angular acceleration α , either about the same axis or about a different axis; in general, these axes change with time. No motion due to rotation occur along these axes. There exists also an inertial resistance to rotation similar to the effect mass has on translation. This is characterized by the moment of inertia, I , which represents the sum of all infinitesimal mass elements, dm , of the

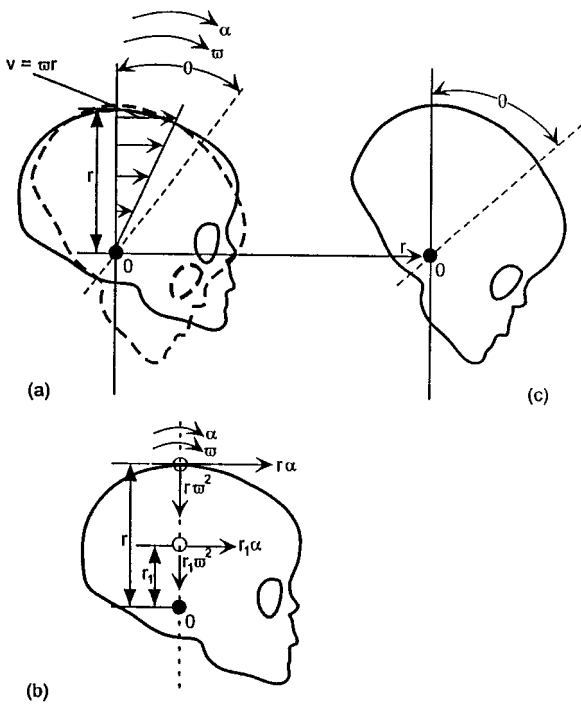


FIG. C. Rotation of the head about the occipital condyles, about an axis passing through O.

objects, multiplied by the square of their respective distances from the axis of rotation r^2 . (This is mathematically defined by the integral $I = \int_{\text{volume}} r^2 dm$.)

In general, when the reference frame is represented by a standard rectangular (Cartesian) $Oxyz$ co-ordinate system, the rotational velocity and acceleration, which are likely to be in a direction not represented by x , y or z are usually broken up so as to represent respective components along the reference axes. For example, as indicated in Fig. C (a-c), head revolution due to the action of the neck muscles can occur in the AP direction about an axis perpendicular to the sagittal plane. Lateral bending may also take arise due to rotation about an axis perpendicular to the midplane of the sternum, as shown in Fig. D(a), whereas simple head rotation, Fig. D(b) takes place about a vertical axis.

If more than one of these rotations occur simultaneously, the magnitude of the combined speeds and accelerations, and the instantaneous axes of rotation must be obtained by the methods of vector analysis. (Note: angular displacement θ is *not* a vector because two such displacements, $\theta_1 + \theta_2 = \theta_2 + \theta_1$, the commutative law of addition, which all vectors must satisfy; however, angular velocity and angular accelerations conform to this requirement). In order to avoid this complication, head rotation will be considered in the sequel to occur only about a single, fixed axis, such as those cited above. However, it must be noted that each element of a rotating body not located on the axis of rotation experiences an instantaneous linear velocity $v = r\omega$,

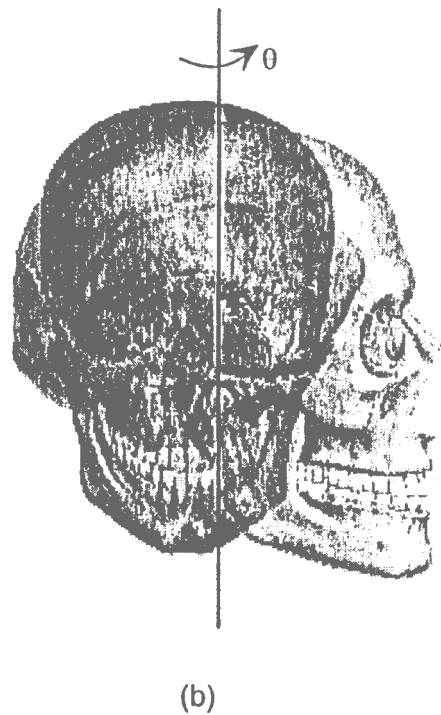
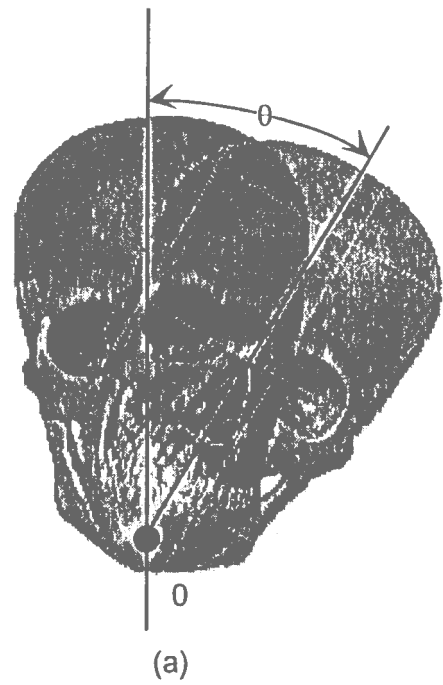


FIG. D. (a) Bending resulting from rotation about an axis perpendicular to an anterior section. (b) Head rotation about a vertical axis.

where r is again the perpendicular distance from the element to the axis (or point) of rotation. Furthermore, each such element is subjected to two simultaneous linear accelerations; one is the normal component $a_n = r\omega^2$, which is directed from the element towards the axis of rotation, and the other is the tangential component $a_t = r\alpha$ perpendicular to a_n and in the direction of the angular acceleration

α , if such acceleration exists. These components are depicted in Fig. C(a) and C(b).

Finally, a rigid body can experience simultaneous translation and rotation which, when confined to a single plane with the axis of rotation perpendicular to that plane, is called 'plane motion'. Such a situation is illustrated in Fig. C(c) for the head/neck complex executing rectilinear translation r and rotation about an axis perpendicular to the AP plane. Here, the linear velocity and acceleration components at any point of the system can be obtained by the arithmetic summation of their components in the x and y directions that are produced by the two separate types of motion. More complicated movements can be readily analysed by vectorial extensions of these methods.

Causation of head injury dynamics

This section deals with the two types of motion already described, translation and rotation, and their causation. As already indicated, pure translation can only exist when the resulting force acts on a particle or passes through the centre of gravity G of a (presumed) rigid body. In this case, the movement is governed by Newton's second law of motion:

$$F = ma = (W/g)a \tag{1}$$

where acceleration can be determined by measurement of time and distance, the mass m can be quantified from Newton's law of universal gravitation (subject too complicated to discuss here). Force F can only be defined by using eqn (1). An example of such force is the weight of an object which, when placed on the ground without any motion, is pushed upwards with the same amount of force as its weight. This action is shown in Fig. E(a); in practice, the movement of the head is restricted by the physical constraints of the neck (and its possible extensions) and by the body.

The law governing rotational motion is given by

$$M = I\alpha \tag{2}$$

where M is the applied moment about the mass centre and I is the moment of inertia about this point. Alternatively, the moment can be taken about

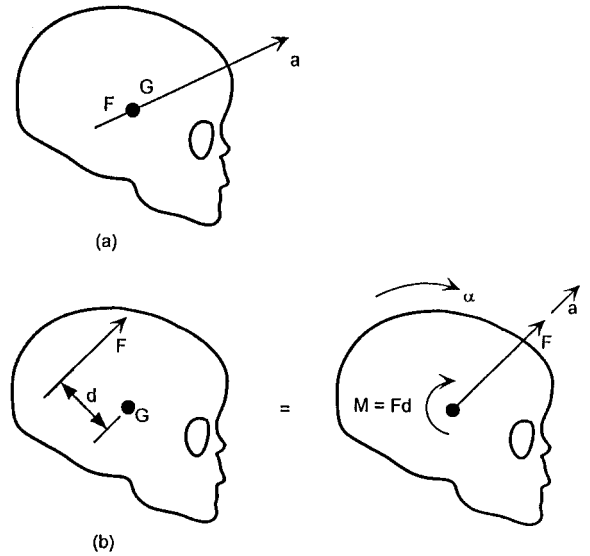


FIG. E. (a) Resulting force passes through mass centre G of a rigid body. (b) Resultant force passes at a distance d from mass centre G and equivalent force system.

a fixed point of rotation in plane motion, provided I is computed about this same position. The effect of a force displaced from the mass centre G by a perpendicular distance d from its line of action is equivalent to a combination of this force acting through the mass centre and a moment $M = Fd$ acting in the appropriate direction to provide the required rotation, as illustrated in Fig. E(b). As stated previously, such rotations produce differential displacements of adjacent spherical layers of the brain due to the outwardly increasing translational velocity of elements with respect to the axis of rotation. This generates shearing of the tissues, resulting in extreme cases in diffuse axonal injury and disruption of the bridging veins due to the difference in the motion of the skull and the brain by virtue of their differing distances from the axis of rotation and their disparate densities, that lead to differing values of the moment of inertia I .

Further descriptions of the application of the laws of motion may be found in any of a plethora of textbooks on the topic of dynamics. A condensed version of these principles is presented in Ref. 24.

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