We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

4,800
Open access books available

122,000
International authors and editors

135M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS BOOK CITATION INDEX INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Chapter 2

Head Injury Mechanisms

Esmaeil Fakharian, Saeed Banaee,
Hamed Yazdanpanah and Mahmood Momeny

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/intechopen.75454

Abstract

Head injury is a major cause of death and disability in young, active population. It may introduce energy through the skin to the deepest structures of the brain. The entered energy may cause direct or primary injury, or result in other, secondary, events to the tissues. These are mechanical loads and are classified as static when the duration of loading is more than 200 ms and dynamic when less than this. The dynamic loads are further classified as impact if the injurious agent has contact with the head or impulsive when the load exerted to other body part/s results in damage to the brain by the change in speed of the head motion. Impact loads can either exert their effect with direct contact to the tissue or may cause inertial loads. The direct contact can cause deformation of the skull or induce energy stress waves to the head and brain. All of these events will result in tissue strain due to compression, tension, or shear. The strain will culminate in injury, which may be a scalp abrasion, laceration, skull fracture, or different kinds of intracranial traumatic lesions.

Keywords: TBI, biomechanics, acceleration, primary events, secondary events

1. Introduction

Trauma defined as a physical harm from an external source is probably one of the earliest experiences of the man on the earth. The first evidence of head injury in human was found in Tanzania. It is due to a crocodile bite about 2,000,000–1,800,000 years BC [1]. On the base of Holy Quran and Genesis, the first death is that of Abel happened by a heavy object struck on head by his brother Cain [2]. Along the history these lesions have included all kinds of blunt and penetrating

injuries to the head, more commonly in occupational activities such as those reported in Edwin-Smith papyrus in workers of the Egyptian pyramids [1, 3] or conflicts and quarrels as in Goliath and David confrontation or those gladiators managed by Galen [4]. By the development of the human society and increasing speeds particularly in transportation after industrial revolution, new injurious events appeared so that gradually traffic accidents became one of the most important causes of morbidity and mortality in all parts of the world [2].

Management of head injury has significantly changed in the past few decades with better understanding of the mechanisms of load transfer to the tissues and biophysical, biochemical, and physiological consequences which result in many different clinical presentations from a simple scalp laceration to brief periods of loss of consciousness and extending to persistent vegetative state [5–11].

Considering the mechanisms of load transfer to the head, different kinds of traumatic pathologies, including skull fracture; epidural, subdural, intracerebral, and intraventricular hematoma; as well as different kinds of contusion and finally diffuse brain injuries, could be identified and their behavior and injurious effects on the brain and clinical consequences defined [10].

In this chapter, we are going to discuss about different kinds of head trauma, their classification, and some aspects of biomechanics of these events.

2. Head injury biomechanics

The consequences of trauma as an energy transmitted to the head is dependent on physical characters of the invading substance, including the density of the invading substance, its size, speed, and duration of loading [12].

By the entrance of a damaging energy load or mechanical input to the head, the first delineating factor for the evolving injury will be the duration of the energy loading [13]. This time interval has defined in a range of 50 to 200 ms. Those lasting more than 200 ms are labeled as static loads, and those less than this, and most frequently less than 50 ms, are considered as dynamic loads [14, 15].

The static causes of injury are very rare and are usually seen when the head is entrapped between hard objects, e.g., the ground and the ruined elements of a building in an earthquake. These heavy loads may cause deformation of the skin or bone and their damage (usually a focal injury).

3. Dynamic injuries

The dynamic causes of injury include a wide variety of mechanisms. The first of these is produced by the transmission of energy to the brain tissue through the changes in speed

(as either of acceleration or deceleration) which are known as impulsive loads. Impulsive loading occurs when the head is not directly struck, but set into motion as a result of a force applied to another part of the body [16]. In such instances, usually, there is no direct and gross evidence of injury to head, i.e., the injury is produced by the inertial changes of the head. In the next group, which is known as impact loads, the offending object when strikes the head may result in injury to tissues from the skin level downwardly depending to the surface area, density, size, and speed of object, directly. On the other hand, it may change the speed of the head and cause its acceleration or deceleration. So, there are inertial changes in the head, and the final result may include those produced by the impulsions.

The inertial loads produced by either impulsions or impactions are exerted by different kinds of acceleration/deceleration. These include translational, rotational, and angular ones, which are defined on the base of the changes on the center of gravity of the skull, the pineal gland. In translation, the changes should be along one of the X, Y, or Z planes. In rotation, the process should be around the axes. These two kinds of acceleration/decelerations are not very common due to the articulation of the skull to the spine; however, the former when happened usually is not associated with severe events, while the latter is highly injurious. The most common kind of event is the angular change, which may be a combination of the abovementioned accelerations.

The impaction of an object to the head can result in change in the configuration of the tissue, either the skin, bone, or deep structures. If this change is above the elasticity of the tissue, it

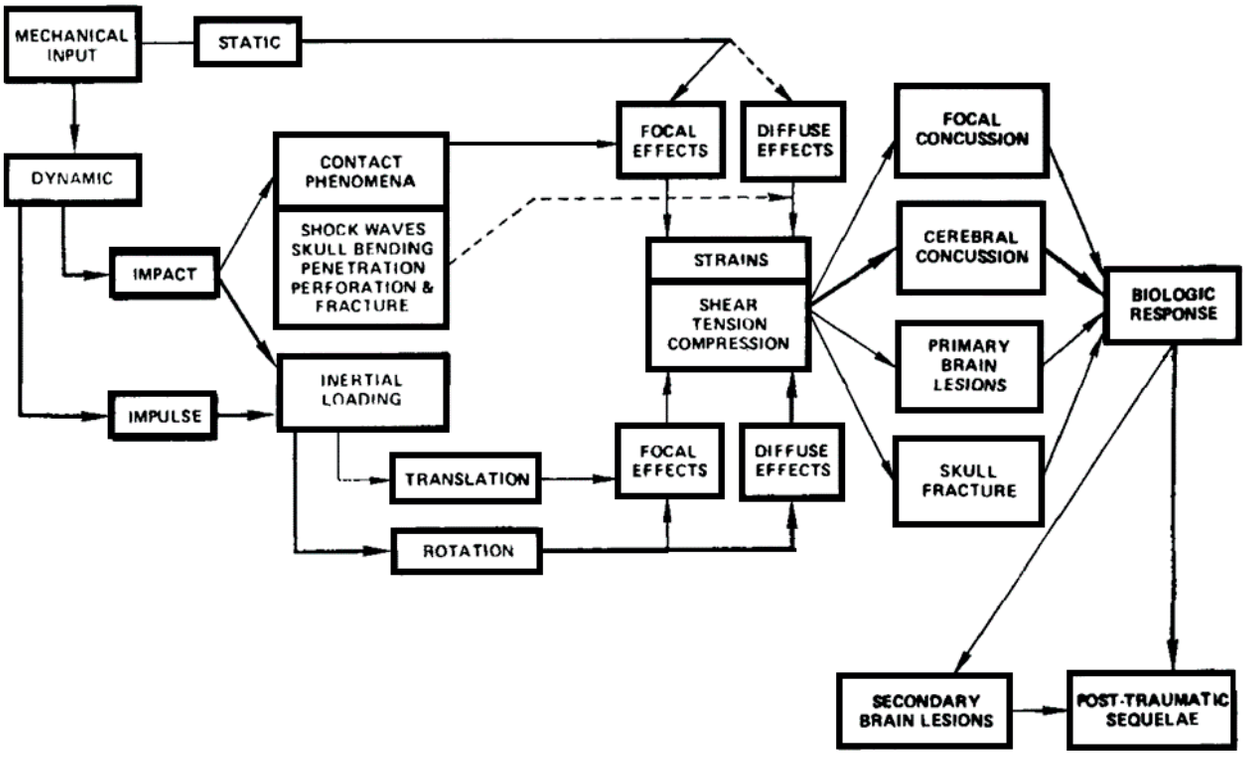

Figure 1. A diagram of head injury mechanisms (from Ommaya and Gennarlli [14]).

will result in its permanent deformity, laceration of the skin, or fracture in the bone. With the greater loads, the offending agent may cause depression of the bone into the intracranial space, namely, depressed skull fracture and laceration of deeper tissues, i.e., dura, brain, and vessels, causing epidural hematoma (EDH), subdural hematoma (SDH), contusion, and intracerebral hematoma (ICH). In more severe cases, especially when the speed is high and the size of the agent is small, perforation and penetration may also happen, e.g., in gunshot wounds. Instead, the impaction may be associated with the passage of a load of energy through the skull and the brain. This energy load causes deformation of the brain and its friction to the surrounding structures including skull base and dural membranes or distortion of the cerebral fiber tracts around each other and finally contusion of the brain tissue (**Figure 1**) [17, 18].

4. Tissue strain and tissue injury

All of these elements, tissue deformation, shock wave, and acceleration/deceleration, will exert energy to the tissue and result in tissue strain as compression, tension, or shear. These may result in injury to the tissues, which in the skull are either neural components, vessels, or bone. It must be reiterated that tissue injury will appear when the load entered to the tissue is above the tolerance and elasticity of the tissue so that the changes appeared on that result in an irreversible event. The tolerance of tissues is dependent on their physical characteristics, the amount of energy, duration of energy loading, and the size of the load, and so it is different for different tissues and even different ages for the same tissue. Most of our experiences in usual daily activity are within the physiological tolerance of our tissues and so are harmless, while more aggressive activities such as some of the professional sports, although still within the range, are at the upper limit of physiologic tolerance and if happened repeatedly will result in gradual or even acute appearance of brain dysfunction. What is happening in different accidents, either vehicles or falling from heights, is above the physical tolerance of the tissues and results in different sequels depending on the involved component.

5. Primary and secondary injuries

These are the mechanisms involved in the condition known as primary injury [19, 20], i.e., the direct result of the entered energy to the head. They may in themselves result in other consequences with further injurious effects either as complications of the first phenomenon or exaggerating it. These are known as secondary injuries, the most common of which are hypoxia and hypotension. Secondary injury may also involve mitochondrial dysfunction, excitotoxicity, free radical production, activation of injurious intracellular enzymes, and other mechanisms within the injured nervous tissues which may result in further dysfunctions of the system [13, 20]. Some of the secondary events are similar to the primary phenomenon which will be dealt here, soon. There are also tertiary injuries, which are usually later effects of the energy loading of the head resulting in other system dysfunctions such as electrolyte

imbalance due to kidney problems, different kinds of heart disturbances, liver insufficiency, and so on, which are not under the scope of this section.

Considering the abovementioned components in production of an injury to the head, different kinds of the clinical cases can be identified. It can be started with the injury to the bone. In a static loading, the long duration of the time of the entered load results in change in the normal configuration of the skull. When this is above the elasticity of the bone for toleration of the entering energy which is usually a compression at the entrance point (outer table of the skull) and tension in either just below the load inner table or the periphery of the entered load, it will result in tissue failure as fracture of the skull. The severity of fracture is dependent on the amount of load and timing. If it is not so big and lasts for brief periods, there will be no further damage to the deeper structures, and usually the victim will be conscious with a single line or stellate pattern of fracture. On occasions with a great load, the whole skull is severely broken into fragments and the brain tissue disrupted, so that it may ooze from the lacerated scalp or nose and ear canals. In such instances, the victim is in deep coma with severe impairment of the brain and brain stem function, resulting in death.

Skull fracture may result from impaction of the head by an object and its contact resulting in change in configuration of the skull. The consequence of this contact if the surface area of the object is more than five square centimeters may be fracture in the skull. If the surface area is smaller, the object denser with a higher speed, it may penetrate the skull or even perforate it and pass through the brain tissue, as mentioned previously. If the event is in an eloquent region, there may be neurological deficit dependent on the brain function. These are the direct or primary sequel of the injury. There are other events which may appear as a complication of the mentioned events, secondary traumatic effects. Different kinds of intracranial hematomas, including EDH, SDH, ICH, and even intraventricular hematoma (IVH), as well as contusion of the brain tissue (admixture of vascular and brain tissue injury), may result from injury to the vessels in the related places. These lesions may result in mass effect in intracranial space, increase in intracranial pressure, and herniation of the brain. Brain laceration as a primary lesion may predispose the patient to convulsion and epilepsy. Another important complication of this kind of injury is infection of the bone and intracranial content, if the overlying skin is lacerated and prepares access for the microorganisms to the deeper structures. These latter events are other examples of secondary effects, although except EDH, which is always a complication of skull deformation (with or without fracture) and always a secondary phenomenon; all other events may happen as a primary event, as discussed in Section 3.

An important point regarding static and impact contact loads to the head is that they usually cause focal lesions in the brain, and these kinds of lesions are not accompanied by change in level of consciousness, primarily. This can be used as a hallmark for those injuries which are not produced by the inertial loads to the brain. It should be reiterated that changing level of consciousness in the above discussed lesions may happen as a complication of either enlargement of the produced hematoma or contusion or the mass effect produced by other secondary effects of injury like edema around the lesions. However, the mechanism of disturbed consciousness in these lesions, usually, is not injury to the brain as the main source of consciousness, because

it is a wholistic function and focal damages cannot produce it, but it is mainly produced by the displacement of the brain tissue from its connecting hiatuses and compression/ischemia of the brain stem sources of the condition. These are well known as cerebral herniations, as another example of secondary injury.

Concussion, diffuse axonal injury (DAI), SDH, ICH, and IVH as primary lesions should be discussed with the mechanism of change in speed of movement of tissues in the head or inertial loads [21]. These can be viewed as a wide spectrum of injuries with very mild cases as brief period of confusion and memory disturbance to short interval of loss of consciousness or concussion [16], to long-standing deep coma or persistent vegetative state (PVS) due to diffuse injury to neurons and axons of the brain or DAI. In normal circumstances, axons are compliant and readily return to their original length after loading. However, with rapid application of tissue strain, such as at the time of head impact, with the anisotropic and complex arrangement, axons behave differently, essentially becoming brittle and vulnerable to injury [22].

In between there are injury to vascular components either in the surface of the brain (SDH), due to the difference in the elasticity and ability of the brain movement and the bridging veins connecting the brain to the venous sinuses placed in the dural layers, or in deeper parts from the cortex and subcortical layers (ICH) to the ventricle (IVH). As was stated previously, the common presentation of all of these events is loss of consciousness (LOC) of the patient from the time of event. The duration of LOC is dependent on the energy load, its effect on the specific parts of the brain, and severity of the injury in the brain.

A key clinical point is that when these lesions are produced by non-inertial loads, as discussed in previous paragraphs, and cause disturbance of level of consciousness due to their secondary effects, appropriate and in time decompression may result in recovery of the consciousness, while in those with inertial loads, decompression may not be followed by recovery of consciousness just after operation or even in longer durations. So, restrict consideration on the clinical course of the patient at the time of admission and focusing on the possible unconsciousness will help the surgeon to predict probable surgical findings and the early post-op outcome.

6. Conclusion

We suggest that application of the discussed algorithm for assessment of the injured patients may help clinicians for predictions of the sequelae outcomes. If used appropriately it even can be used for clinical evaluation of the injured patients and decision-making for a rational paraclinical study. Although increasing availability of computed tomographic (CT) scanners in most hospitals has supplanted the need for skull X-ray study as one of the primary steps in patients with head injury, however whenever inertial loads are considered as the main mechanism of trauma, even in the absence of CT scanners, the use of skull X-ray will not be helpful for the diagnosis of the probable injuries.

Finally, it must be kept in mind that classifications and delineations are used for better understanding of the events on the base of current knowledge and so may occasionally not comply

with all of the events in reality. While managing head injury patients, one of these pitfalls is the definition of dynamic and static loadings of the brain on the base of duration of the event which is a small fraction of a second for both. This means that it is always possible to have a spectrum of different mechanisms and lesions due to both of the mechanisms. The algorithm should be used for better prediction, understanding, and explanation of the events on the base of detailed clinical evaluation and not as a restrict rule.

Author details

Esmaeil Fakharian[1,2]*, Saeed Banaee[1], Hamed Yazdanpanah[1] and Mahmood Momeny[1]

*Address all correspondence to: efakharian@gmail.com

1 Department of Neurosurgery, Kashan University of Medical Sciences (KAUMS), Kashan, IR Iran

2 Trauma Research Center, KAUMS, Kashan, IR Iran

References

[1] Bertullo G. History of traumatic brain injury (TBI). African Journal of Business Management. 2015;**3**(7):381-409. DOI: 10.18081/2333-5106/015-07/381-409

[2] Fakharian E. Trauma research and its importance. Archives of Trauma Research. 2012;**1** (1):1-2. DOI: 10.5812/atr.5287

[3] Ghannaee Arani M, Fakharian E, Sarbandi F. Ancient legacy of cranial surgery. Archives of Trauma Research. 2012;**1**(2):72-74. DOI: 10.5812/atr.6556

[4] Ghannaee Arani M, Fakharian E, Ardjmand A, Mohammadian H, Mohammadzadeh M, Sarbandi F. Ibn Sina's (Avicenna) contributions in the treatment of traumatic injuries. Trauma Monthly. 2012;**17**(2):301-304. DOI: 10.5812/traumamon.4695

[5] Cloots RJH, Gervaise HMT, van Dommelen JAW, Geers MGD. Biomechanics of traumatic brain injury: Influences of the morphologic heterogeneities of the cerebral cortex. Annals of Biomedical Engineering. 2008;**36**(7):1203-1215. DOI: 10.1007/s10439-008-9510-3

[6] Gaetz M. The neurophysiology of brain injury. Clinical Neurophysiology. 2004;**115**:4-18

[7] Cloots RJH, van Dommelen JAW, Kleiven S, Geers MGD. Multi-scale mechanics of traumatic brain injury: Predicting axonal strains from head loads. Biomechanics and Modeling in Mechanobiology. 2013;**12**:137-150. DOI: 10.1007/s10237-012-0387-6

[8] Greve MW, Zink BJ. Pathophysiology of traumatic brain injury. Mount Sinai Journal of Medicine. 2009;**76**:97-104. DOI: 10.1002/msj.20104

[9] Post A et al. The dynamic response characteristics of traumatic brain injury. Accident Analysis and Prevention. 2015;**79**:33-40. DOI: 10.1016/j.aap.2015.03.017

[10] Saatman KE et al. Classification of traumatic brain injury for targeted therapies. Journal of Neurotrauma. 2008;**25**:719-738. DOI: 10.1089/neu.2008.0586

[11] Stein SC. Patrick G, Meghan S, Mizra K, Seema SS. 150 years of treating severe traumatic brain injury: A systematic review of progress in mortality. Journal of Neurotrauma. 2010;**27**:1343-1353. DOI: 10.1089/neu.2009.1206

[12] McLean AJ, Anderson RWG: Biomechanics of closed head injury. In: Reilly P, Bullock R, editors. Head Injury. London: Chapman & Hall; 1997. ISBN 0 412 58540 5

[13] Meythaler JM, Peduzzi JD, Eleftheriou E, Novack TA. Current concepts: Diffuse axonal injury-associated traumatic brain injury. Archives of Physical Medicine and Rehabilitation. 2001;**82**:1461-1471. DOI: 10.1053/apmr.2001.25137

[14] Ommaya AK, Gennarelli TA. Cerebral concussion and traumatic unconsciousness. Brain. 1974;**97**:633-654

[15] Smith DH, Meaney DF, Shull WH. Diffuse axonal injury in head trauma. The Journal of Head Trauma Rehabilitation. 2003;**18**(4):307-316

[16] Poirier MP. Concussions: Assessment, management, and recommendations for return to activity. Clinical Pediatric Emergency Medicine. 2003;**4**:179-185. DOI: 10.1016/S1522-8401(03)00061-2

[17] Bayly PV, Cohen TS, Leister EP, Ajo D, Leuthardt EC, GM: Deformation of the human brain induced by mild acceleration. Journal of Neurotrauma. 2005;**22**(8):845-856. DOI: doi.org/10.1089/neu.2005.22.845

[18] Feng Y, Abney TM, Okamoto RJ, Pless RB, Genin GM, Bayly PV. Relative brain displacement and deformation during constrained mild frontal head impact. Journal of The Royal Society Interface. 2010;**7**:1677-1688. DOI: 10.1098/rsif.2010.0210

[19] Hawryluk GWJ, Manley GT. Classification of traumatic brain injury: past, present, and future. In: Grafman J, Salazar AM, editors. Handbook of Clinical Neurology. Vol. 127 (3rd series). Traumatic Brain Injury, Part I. Waltham, USA: Elsevier B.V.; 2015. pp. 15-21

[20] Werner C, Engelhard K. Pathophysiology of traumatic brain injury. British Journal of Anaesthesia. 2007;**99**(1):4-9. DOI: 10.1093/bja/aem131

[21] Post A, Hoshizaki TB. Mechanisms of brain impact injuries and their prediction: A review. Trauma. 2012;**14**(4):327-349. DOI: 10.1177/1460408612446573

[22] Johnson VE, Stewart W, Smith DH. Axonal pathology in traumatic brain injury. Experimental Neurology. August 2013;**246**:35-43. DOI: 10.1016/j.expneurol.2012.01.013