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Skull fractures in car accidents: Types and Causes

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Abstract

Brain injury may be the result of a sudden blow to the head or head impact. The result is a damage at the site of application of force and damage of the remote location. The force acting on the head causes not only local effects through the oppression, but also puts in motion the head, which causes the cranial structures.

The aim of the study was to characterize head injuries with special attention to their types.

After regaining consciousness, and coming out of immediate danger of life reveals a number of problems the nature of neurological, psychological and neuropsychological, the latter reveal the disorder is sometimes quite late due to their discrete nature.

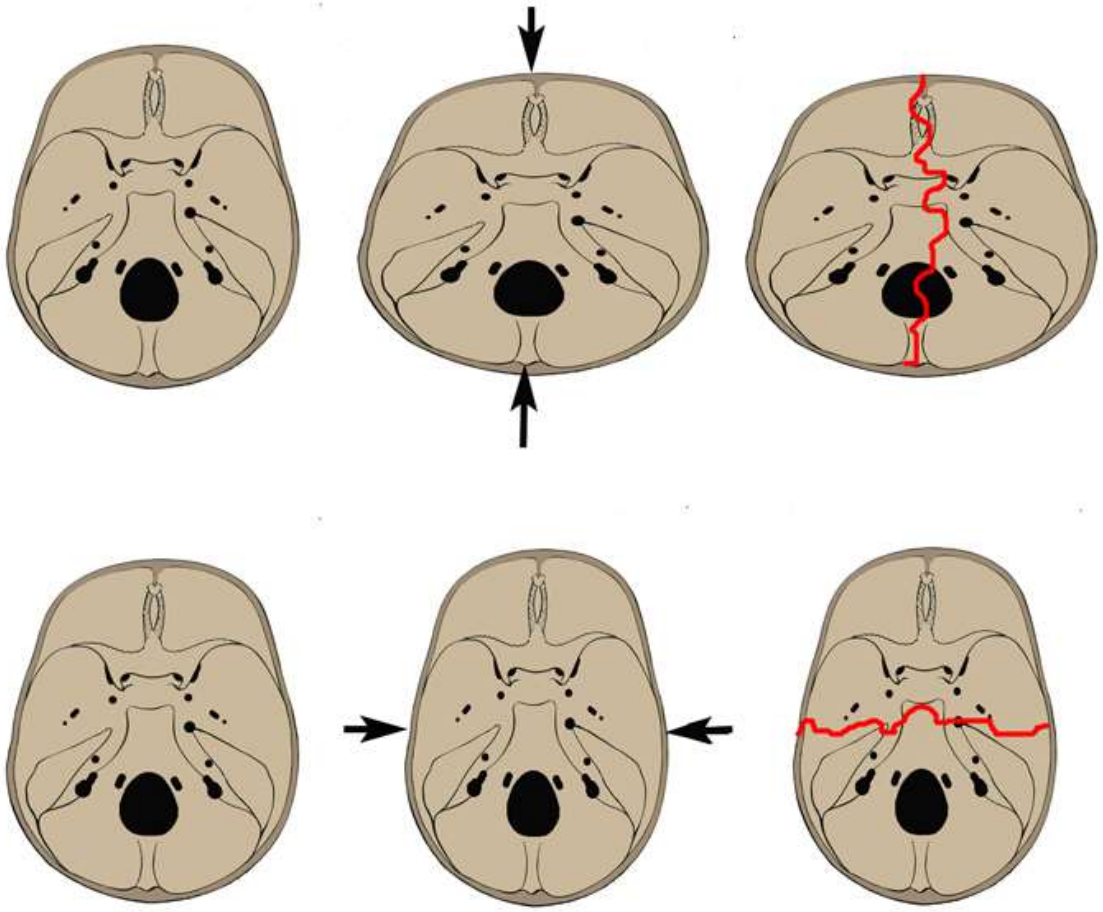
A serious problem is post traumatic stress disorder in the form of fear, helplessness, feelings of constant threat. In extreme cases, they represent a permanent team apical or brain death occurs.

Key words: fracture; head injuries; medic legal autopsy; RTA.

Introduction

The effects of forces on the head depends on the following physical parameters of the object / head: linear velocity, angular velocity, angular and linear acceleration, force, mass and energy. Studies Beninghofa, Bruns Messerer and the second half of the nineteenth century, have shown [1] that the skull, which is apparently a rigid body, subjected to mechanical stresses, is deformed. Under the influence of the static forces, it reduces its dimension along the line of force and extends perpendicularly to this direction, and the moment of exceeding the scope of the strength of bone fracture is formed, the slit is perpendicular to the direction of disruptive forces. The bone acts like a typical non-metallic rigid body, which do not have significant plasticity (ductility, malleability).

The longitudinally running load causes a centrifugally operating the transverse and longitudinal slot formation. In turn, the transversely acting forces produce blunt by analogy with the progress of transverse cracks (Fig. 1).



Dig. 1. Diagram of the mechanisms leading to the formation of linear skull fracture based on the application and the direction of the force. Arrows indicate the directions of compressive forces - in the sagittal plane (top figures) and in the transverse plane (bottom of the figure). Red shows the resulting course of the action of these forces slots fractures. (Figure own according to the literature).

Types of fractures

As to the nature and extent of damage to the bones of the skull, is generally recovered six categories of such pathology. These are: linear fractures, fractures of intussusception (ie. Break-in), rupture of the seams (with a visible gap), basal, and so complex. Rising [3-6].

Linear skull fracture in a bone fractures penetrating the entire depth from the outer to the inner periosteum (dura). They are most common. Usually they run more or less upright, pulling on a limited space and are not associated with displacements factions. Linear fracture typically formed by the action of a blunt instrument, which energy is transferred to a relatively large area of the skull. They do not cause significant intracranial injury, if you do not pass through one of the Dural venous sinuses. A more serious consequence would be the formation of epidural hematoma (fracture line running through the husk of the temporal bone can damage one of the larger arteries dura). In infants and young children the most common fracture is a fracture of the parietal bone, and the type of fracture linear fracture.

Fracture of intussusception is the result of blunt trauma, acting on a limited area of the skull (e.g. blow. Hammer, stone). Faction bone is detached partially/completely and recessed into the cranial cavity. This is due to the risk of intracranial hypertension, brain contusion, intracranial infection (in the case of coatings full perforation of the head), the hematoma/subdural. If the depth to which the fragments were cavity is greater than the thickness of the bone, the risk of damage to the dura mater increases and may be an indication for urgent surgery. In other cases, these fractures do not require immediate surgery. Intussusception bone fractures can be divided into simple and complex. Fractures are the result of simple actuation force on a small area of the skull. Characteristic of a simple fracture of the skull dent - the so-called. fracture ping-pong ball - can occur in infants whose bones are still poorly calcified and present in certain parts of the original structure. the so-called. plexus. In this case, the bones are not damaged, but deformed. Complex fractures of the skull bones are associated with discontinuity of the skin, they are therefore open fractures. Bone defects are then linear, with dents or multi-part fractures. They are therefore open fractures. Bone defects are then linear, with dents or multi-part

fractures. They are therefore open fractures. Bone defects are then linear, with dents or multi-part fractures.

Fractures of rupture seams occur when the line break exceeds one or more of the cranial sutures, causing the relaxation of normal relatively strong syndesmosis fibrosa). These fractures are common injuries to the skull in children 4 years of age and in practice does not occur in older children and adults. Children in this age of the skull sutures are usually very flexible and do not occur there no ossification processes. Cleavage may occur even in the absence of the fracture gap and to be the only symptom skull injury (in addition to damage to the soft parts).

Basal fractures are the result of very considerable forces. Fractures of the skull base generally include elements anatomical frontal bone, ethmoid, sphenoid, temporal and occipital. Fracture gap extends along the lines connecting the areas of reduced resistance, which are the natural openings, and channels of the skull base: orbital fissure upper channel optic foramen oval, round, spike inner and the outer ear canal, the jugular foramen, the channel internal carotid artery, the opening jagged , oral middle and inner ear and the system of air cells and the auditory tube. They are relatively rare, but their clinical features are typical and always suggest the beginning of serious damage to the skull/intracranial. Depending on the location it is: the presence of blood in the sinuses, rhinorrhea, nasal or auricular hematoma Spectacled hematoma behind the ear (Battle's sign) bleeding from the external auditory canal, pulsating hemorrhage (called. cascading) from the nose, as an effect of bust internal carotid artery in the vicinity of the sphenoid bone, hematoma tympanic cavity. Fractures within the posterior fossa may cause the compression of brain stem and lead to respiratory distress, hypotension and tachycardia / tachycardia. Late complication of fractures of the skull base may be meningitis, due to the opening of the gate by infection of the paranasal sinuses or air spaces of the middle ear and the outside. The so-called. "Fracture rotational" effect of removing skull base portion of the skull base, usually around foramen [7] hematoma tympanic cavity. Fractures within the posterior fossa may cause the compression of brain stem and lead to respiratory distress, hypotension and tachycardia / tachycardia. Late complication of fractures of the skull base may be meningitis, due to the opening of the gate by infection of the paranasal sinuses or air spaces of the middle ear and the outside. The so-called. "Fracture rotational" effect of removing skull base portion of the skull base, usually around foramen [7] hematoma tympanic cavity. Fractures within the posterior fossa may cause the compression of brain stem and lead to respiratory distress, hypotension and tachycardia / tachycardia. Late complication of fractures of the skull base may be meningitis, due to the opening of the gate by infection of the paranasal sinuses or air spaces of the middle ear and the outside. The so-called. "Fracture rotational" effect of removing skull

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Among the special role of the basal fractures occupy those associated with a fracture of the temporal bone, and especially its most constructed and mineralized parts - rocky. Temporal bone has a particularly rigid construction - Part rocky has the most coherent construction of the tissues of the human body. Particularly frequently, because of their necessary to induce a very large force, they occur in the course of traffic accidents at high speed and hit a hard surface. Table I lists the most common causes of injury temporal bone obtained from the literature [8-11].

Table I. The etiology of fractures of the temporal bone obtained from the literature.

Cause	The incidence
Car accident	42-44%
Fall	16-19%
Deduction by vehicle	approx. 10%
sports injury	approx. 19%
Motorcycle accident / bicycle	approx. 13%
Beating	6-17%
Gunshot wound	3-6%
Other	approx. 5%

Fracture of the temporal bone as the first classified Ulrich already in 1926, dividing them into transverse to the axis of the pyramid (parts and rocky above the temporal bone) and longitudinal. It is now widely used by McHough division proposed in 1959. This classification distinguishes fracture longitudinal, transverse, and mixed. The fracture longitudinal dividing line factions extends parallel to the long axis of the pyramid, along its front edge. The slot extends through the top or upper posterior wall of the external auditory canal of bone, and mammary cover cavity drum, then passes the front of hard capsule bony labyrinth, there is a middle cranial fossa, ending its course in the vicinity of the opening of the oval or jagged [12-14]. The cause of these injuries is the most blunt trauma, which power is concentrated in the

parietal or temporal. In the case of transverse fractures is the most common cause of much or very much the force of the surrounding occipital or premier. Fracture gap so classified is perpendicular to the long axis of the pyramid. Transverse fracture typically start in a big hole. Then, the gap extends along the internal auditory canal passes through the screw, guided by the front edge of the pyramid, or extends through the inner ear (screw and vestibule) and along the canal of the facial nerve occurs geniculate. Middle ear, the eardrum and the external auditory canal space remains intact. Sometimes the fracture gap passes on the medial wall of the tympanic cavity, including hillock (promontory) and ending in the area of the round window niche. Mixed temporal bone fractures are also frequently the result of severe trauma, usually hitting with a blunt object in the head with a large or very large force. The damaged structures can be both middle ear. as well as internal. It considered as the most common fractures of oblong, accounting for about 70 to 90% of cases. For transverse fractures include about 10 to 30% of cases, and mixed to 10% [15-17].

Kelly in his research tried to further simplify the division of the temporal bone fractures and offered only two types: wall-saving bone fractures and fractures of the labyrinth of its violation. The most important element of this classification is whether there has been a complete and irreversible hearing loss or not, it is therefore of high clinical relevance [13-18].

Young children may be atypical form of fracture of the skull called a growing fracture. Such fractures are found only in children under 4 years of age. They are the result of some linear fracture of the parietal bone. This change is produced by tearing the dura mater to form a soft bag of the tire, which is the principal mechanism leading to the propagation of edges of the joint fracture. Late stage of such a fracture, if not already provided, it may be meningo cerebral visible externally. Fractures growing usually develop 3-6 months after injury with skull fracture creation of linear.

Types of intracranial injury

Intracranial damage can be divided into early (primary) and late (secondary). The former include: wounds and bruises, to another, edema, hemorrhage, ischemia, thrombosis, resulting in hypoxic brain tissue. No later than they appear: tissue hypoxia and acidosis and pulmonary vascular intracranial. It should be remembered that the functional neurological deficits can occur even though no obvious structural damage: a large transient vasoconstriction occurs in the course of subarachnoid hemorrhage may lead to a significant and extensive damage to the brain tissue [18].

Brain injury may be the result of a sudden blow to the head or head impact. The result is damage at the site of application of force and damage of the remote location. The force acting on the head causes not only local effects through the oppression, but also puts in motion the head, which causes the cranial cavity structures [19].

The main types of cranio-cerebral trauma this impact and the acceleration-driven. Trauma impact occurs by direct application of force. Its effect can be: contusion, intussusception, perforation of the cranial vault, skull fracture, epidural hematoma, subdural hematoma, cerebral contusion/bleeding in the area of the cortex.

Trauma acceleration-driven, produce a result of the pressure gradient and intracranial inertial forces, without contact with the head and may result in subdural hematoma, hematoma, subarachnoid hemorrhage, intracerebral, trauma displacement, diffuse axonal injury, cortical hemorrhage/cavitation.

The mechanisms operating in these types of injuries can be described as a static load: the head is slowly deformed in the compression mechanism, resulting in breaking the cover/base of the skull, often preserved unconscious victim. In extreme cases, when significant deformation of the brain occurs fatal outcome; shock load or contact: object hitting the head or the head of an object. If this is a massive, flat and slowly moving his head puts energy in motion; if it is small, fast, sharp, his energy is concentrated in one place (hitting contact point) and leads to localized damage to the skull closely with perforation of the skull cap and adjacent areas of trauma cranial cavity; impulsive load or without contact: head is driven in a sudden movement with no apparent contact with it, eg. when the force acts on the neck (typical here is trauma type whiplash - shock whip), causing the brain in a sudden movement, initially in the opposite direction, and then in accordance with the direction of the force; Linear acceleration: a sudden change in the direction of movement of the head, yet moving, causes the tension stress and strain on the structure intracranial because of the action of inertia forces. Various mechanical properties of the different structures result in their damage, to varying degrees and scope. The perforations are formed, break up, burst tires, contusion brain tissue and/or diffuse axonal injury; acceleration/and angular position: the head rotates about the center of gravity. It is usually a combination of linear and angular acceleration, a movement always takes place along a curve, because of the fixation of the head on the neck, so the largest amplitude of movement it refers to structures under the vault of the skull. It is the most common type of injury; there arise forces stretching or compression type, acting on connective tissue, nerve, blood vessels, meninges; the mildest effect is extensive damage to the brain (diffuse axonal injury leading to

loss of consciousness), but it can also lead to brain contusions of the pyramid of the temporal bone and sphenoid bone protruding parts.

Types of damage to the brain arising as a result of the above-listed and briefly described mechanisms can be grouped into four categories, characterized by increasing severity of the damage are: injury of the compression or tension mechanism: pulling, pushing, stretching in excess of the tolerance limits of tissue; changes caused by these phenomena are usually minor and can usually be seen only in pictures electron microscopy; injuries of the cutting mechanism, resulting in situations where the portion of the brain is moving at a different speed than the whole or rotates at a different speed; changes can be detected by MRI; injuries of the steering mechanism: when the portion of the brain is twisted one way and the other rotates in the opposite or remaining stationary. They arise if serious damage occurs and parenchymal bleeding. The lesions can also be seen on the CT image; injuries of the vacuum device: the sudden stopping of the rapidly head causes a temporary increase in the pressure in the cranial cavity on the side in contact with the object and the reduced pressure on the opposite side; vacuum liberates free gases from fluids (blood, cerebrospinal fluid) which produces a cavitation – presence of small air bubbles in the tissue, leading to local vascular damage to the brain and

Discussed mechanisms, the generated result of an action of various forces, eventually leading to the development of specific morphological changes, which are a common denominator in the form of clinical presentation. These changes, as mentioned above, are dynamic, which means in accordance with the known concept of chain pathogenic, the spread, severity and appending new phenomena, and then quenching and recovery processes or death. Pathologies include, deformation / breakage of the skull. Depending on the speed, the mass, the size of the contact, and the hardness of an object impinging on the skull may perforation, collapse, fracture or deformation of the linear skull fracture with no visible gap, wherein the head is injured dislodge- ment deeper than the head which is in motion. If the contact area is less than 13 cm2 due to impact can be a perforation of the skull, if the field is increased, the result can be a linear fracture. Linear fractures tend to be usually the result of falls. Applying a force to the area temporomandibular joint, or occipital front may cause fracture of the skull base; Cortical bleeding. These are local bleeding as a result of local damage to the surface of the brain. May also extend to the white matter, but there is no cracks or soft tires spider. They cause local damage neurons and cause gradually growing, but limited swelling; These lesions are caused by short-term acting external stimuli (explosion, collapse) and also are caused by the deformation of the skull or skull of sudden rotation. Typically localized to the frontal and parietal, frontal cerebral pole, surrounding orbit front, temporal pole, the side surface of the

temporal lobe. Posterior fossa hematomas are rather the result of force directly to the occipital countryside than by applying a force from the other side of the skull; tear, cut parenchymal. These phenomena concern not only damage the bark, but white matter. Usually it comes to them during shearing forces when the medial part of the temporal lobe is displaced medially and toward the back and pushes the dura, the cerebellum and the foramen magnum. The biggest damage to relate to bend Postcentral gyrus parietal lobe; Diffuse axonal damage occurring as a result of acceleration of action, esp. arc. Manifested clinically loss of consciousness lasting more than 6 hours, while morphologically tangible are only minor local swelling and bleeding. Microscopic changes can be seen esp. the amygdala, hippocampus and front part of the stem of the brain. Often coexists subarachnoid hemorrhage; serious damage to white matter, including the corpus callosum, vault, stem, cerebellum, diencephalon, They occur at appreciable injuries; subsequent inflammatory response as a result of tissue damage and the release of inflammatory mediators. This leads to the migration of leukocytes and development of phagocytic reaction, but usually short-lived[18-26].

Table II. It summarizes the most common pathological changes in specific types of injuries.

Table II. Pathological changes in traumatic brain injuries depending on the mechanism of injury (by Oechman modified)

type of injury	**contact strength**	**noncontact force**	
	+ small ++ average +++ high	linear acceleration	angular acceleration
fracture of skull	+++		
epidural hematoma	+++	+	+
subdural hematoma	++	+++	+
subarachnoid hemorrhage	+	+	+++
bleeding cortical	homolateral	contralateral	
intracerebral hemorrhage		+++	+++
intraventricular hemorrhage			+++
contusion			+++
Diffuse axonal		+	+++
concussion			+++

Secondary changes which occur after hours of injury are: intracranial pressure, neurogenic shock, vascular, as well as biochemical changes (increase in the concentration of ACTH, steroids, ADH, IL-1, TNF, catecholamine's, acute phase proteins, NO synthase, increase of free radicals), caused by hormonal deregulation, generally leading to increased catabolic processes, posing significant problems in terms of maintaining vital functions in these patients [27].

Summary

After regaining consciousness, and coming out of immediate danger of life reveals a number of problems the nature of neurological, psychological and neuropsychological, the latter reveal the disorder is sometimes quite late due to their discreet character. A typical phenomenon is also retrograde amnesia, the longer, the more severe injury. Often there is also long-lasting differently anterograde amnesia. Sometimes serious consequence of post-traumatic epilepsy. All this makes the man after severe mechanical brain trauma very often for a long period of time is disabled and loses its ability to function normally. A serious problem is post traumatic stress disorder in the form of fear, helplessness, feelings of constant threat [28,29].

The survival of the victim is highly dependent on the severity of the brain injury. Light damage is characterized by a period of unconsciousness lasting up to 20 minutes, with the Glasgow outcome scale at the level of 13-15 points. Rather, there is no focal neurological symptoms and cannot be diagnosed brain contusion or hematoma. Despite the lack of perceptible macroscopically visible pathological changes are often the type of neuropsychological disorders. Within a few weeks of the injury is followed by headache (71%), fatigue (60%), the type of imbalance dizziness. Usually, symptoms disappear within three months, some residual symptoms may persist longer.

Patients with moderately severe brain trauma achieve the result in Glasgow scale in the range of 9-13 pkt00. About 30% of them have tangible CT intracranial pathology. Of these, 1.3 shows the progression of changes and changes in the nature of late, 4-10% during hospitalization requires a craniotomy. Mortality in this group is 0,9-2,5%, a further 15% require intensive care, and only 60% present a good clinical condition after 6 months of injury. Over 70% of victims are not able to return to work within three months.

For severe brain damage indicates the result of the scale of Glasgow below 9 points. The risk of a fatality in this group is from a dozen to several dozen percent. Cured patients present a significant or very significant sphere of cognitive disorders, memory deficits, behavioral disorders, neurological deficits.

In extreme cases, they represent a permanent team vegetative state or brain death occurs.

References:

1. Gardner, Raquel C., and Kristine Yaffe. "Epidemiology of mild traumatic brain injury and neurodegenerative disease." Molecular and Cellular Neuroscience 66 (2015): 75-80.
2. Harvey, Lara A., and Jacqueline CT Close. "Traumatic brain injury in older adults: characteristics, causes and consequences." Injury 43.11 (2012): 1821-1826.
3. Allareddy, Veerasathpurush, Veerajalandhar Allareddy, and Romesh P. Nalliah. "Epidemiology of facial fracture injuries." Journal of Oral and Maxillofacial Surgery 69.10 (2011): 2613-2618.
4. Faul, Mark, et al. "Traumatic brain injury in the United States; emergency department visits, hospitalizations, and deaths, 2002-2006." (2010).
5. Crooks C.Y., Zumsteg J.M., Bell K.R.: Traumatic brain injury: A review of practice management and recent advances. Phys. Med. Rehab. Clin. N. Am. 2007, 18, 681–710.
6. Corrigan, John D., Anbesaw W. Selassie, and Jean A. Langlois Orman. "The epidemiology of traumatic brain injury." The Journal of head trauma rehabilitation 25.2 (2010): 72-80.
7. Roozenbeek, Bob, Andrew IR Maas, and David K. Menon. "Changing patterns in the epidemiology of traumatic brain injury." Nature Reviews Neurology 9.4 (2013): 231.
8. De Boer J., Dubouloz M.: Handbook of disaster medicine. Van der Wees, Utrecht 2000.
9. Osmond, Martin H., et al. "CATCH: a clinical decision rule for the use of computed tomography in children with minor head injury." Canadian Medical Association Journal 182.4 (2010): 341-348.
10. Streck Jr, Christian J., et al. "Evaluation for intra-abdominal injury in children following blunt torso trauma. Can we reduce unnecessary abdominal CT by utilizing a clinical prediction model?." The journal of trauma and acute care surgery 73.2 (2012).
11. Babcock, Lynn, et al. "Predicting postconcussion syndrome after mild traumatic brain injury in children and adolescents who present to the emergency department." JAMA pediatrics 167.2 (2013): 156-161.
12. Dayan, Peter S., et al. "Association of traumatic brain injuries with vomiting in children with blunt head trauma." Annals of emergency medicine 63.6 (2014): 657-665.
13. Rabiner, Joni E., et al. "Accuracy of point-of-care ultrasound for diagnosis of skull fractures in children." Pediatrics 131.6 (2013): e1757-e1764.
14. Dunning J., Stratford-Smith P., Lecky F., Batchelor J., Hogg K., Browne J., Sharpin C., Mackway-Jones K.: A meta-analysis of variables that predict significant intracranial injury in minor head trauma. Arch. Dis. Child 2004, 89, 653–659.

15. Dunning J., Stratford-Smith P., Lecky F., Batchelor J., Hogg K., Browne J., Sharpin C., Mackway-Jones K.: A meta-analysis of clinical correlates that predict significant intracranial injury in adults with minor head trauma. J. Neurotrauma 2004, 21, 877-885.

16. Ersahin Y., Gülmen V., Palali I., Mutluer S.: Growing skull fractures (craniocerebral erosion). Neurosurg. Rev. 2000, 23, 139-144.

17. Bin, Steven S., Sara A. Schutzman, and David S. Greenes. "Validation of a clinical score to predict skull fracture in head-injured infants." Pediatric emergency care 26.9 (2010): 633-639.

18. Frey L.C.: Epidemiology of posttraumatic epilepsy: A critical review. Epilepsia 2003, 44 (Suppl. 10), 11–17.

19. Garner A., Lee A., Harrison K.: Comparative analysis of multiple-casualty incident triage algorithm. Ann. Emerg. Med. 2001, 38, 541-548.

20. Gavett, Brandon E., Robert A. Stern, and Ann C. McKee. "Chronic traumatic encephalopathy: a potential late effect of sport-related concussive and subconcussive head trauma." Clinics in sports medicine 30.1 (2011): 179-188.

21. Galetta, K. M., et al. "The King-Devick test as a determinant of head trauma and concussion in boxers and MMA fighters." Neurology 76.17 (2011): 1456-1462.

22. Adamsbaum, Catherine, et al. "Abusive head trauma: judicial admissions highlight violent and repetitive shaking." Pediatrics 126.3 (2010): 546-555.

23. Morton, Patricia Gonce, et al. Critical care nursing: a holistic approach. Lippincott Williams & Wilkins, 2017.

24. Piteau, Shalea J., et al. "Clinical and radiographic characteristics associated with abusive and nonabusive head trauma: a systematic review." Pediatrics 130.2 (2012): 315-323.

25. Colbert, Cherie A., et al. "Value of cerebral microhemorrhages detected with susceptibility-weighted MR Imaging for prediction of long-term outcome in children with nonaccidental trauma." Radiology 256.3 (2010): 898-905.

26. Zetterberg, Henrik, Douglas H. Smith, and Kaj Blennow. "Biomarkers of mild traumatic brain injury in cerebrospinal fluid and blood." Nature Reviews Neurology 9.4 (2013): 201.

27. Kemp, A. M., et al. "Neuroimaging: what neuroradiological features distinguish abusive from non-abusive head trauma? A systematic review." Archives of disease in childhood 96.12 (2011): 1103-1112.

28. Goniewicz, Krzysztof, et al. "Epidemiology of road traffic accidents in adults. A systematic review." Journal of Education, Health and Sport 7.7 (2017): 92-100.

29. Goniewicz, K., et al. "Road accident rates: strategies and programmes for improving road traffic safety." European journal of trauma and emergency surgery 42.4 (2016): 433-438.