Correlations Between the Glasgow Score and the Survival Period in Patients with Severe Traumatic Brain Injury

RĂZVAN ȘTEFAN ȚOLESCU¹, MARIAN VALENTIN ZORILĂ², ROXANA EUGENIA ZĂVOI², CRISTINA POPEȘCU³, ILIE DUMITRУ⁴, ALEXANDRU CONSTANTIN OPRICA⁵, LAURENȚIU MOGOANTĂ⁶

¹PhD Student, Department of Histology, University of Medicine and Pharmacy of Craiova, Romania
²Department of Forensic Medicine, University of Medicine and Pharmacy of Craiova, Romania
³Department of Anatomy, University of Medicine and Pharmacy of Craiova, Romania
⁴Department of Road Vehicles, Transportation and Industrial Engineering, Faculty of Mechanics, University of Craiova, Romania
⁵PhD Student, Department of Road Vehicles, Transportation and Industrial Engineering, Faculty of Mechanics, University of Craiova, Romania
⁶Department of Histology, University of Medicine and Pharmacy of Craiova, Romania

ABSTRACT: Traumatic brain injury (TBI) contributes by 30% to the mortality induced by traumatic injuries, also being one of the major causes of invalidity worldwide. The clinical classification of the severity of mild, moderate or severe TBI is made according to the Glasgow scale, according to the patient’s conscious state, motric changes, speech changes and eye opening. In our study, we evaluated the correlation between the Glasgow score at admission and the survival period of patients suffering from TBI, using the data recorded in the Forensic Medicine Institute of Craiova between 2011-2017 on 1005 cases with the diagnosis of death by TBI. We observed that TBI affects persons of all ages, starting from babies up to the elderly aged over 90 years old. Regarding the generation mechanism, most deaths were caused by fallings (438 cases, 43.58%), followed by car accidents (333 cases, representing 33.13%). The number of patients who presented a post-traumatic survival period was 802 (79.80%), of which 779 adults (77.51%) and 23 children (2.29%). Among these, 785 (78.11%-764 adults and 21 children) were hospitalized, while in 64.58% of the TBI patients there was recorded the Glasgow score at admission. 75% of the TBI patients in whom there was recorded the Glasgow score presented a 1st-4th coma degree, with a Glasgow score from 3 to 8 and only 25% had a slightly altered or preserved conscious state, with a Glasgow score=9-15. The survival period varied from less than 24 hours to over 15 days. In the hospitalized patients, there were performed emergency surgeries in 269 (26.76%) cases, the surgical intervention being temporized in 108 (10.74%) patients.

KEYWORDS: Traumatic brain injury, mortality, morbidity, risk factors, structural brain damages.

Introduction

Traumatic brain injury (TBI), defined as changes of the function and structure of the brain, caused by an external physical force [1], represent one of the most frequent causes of morbidity and mortality in young adults aged under 45 years old, all over the world [2,3]. The annual incidence of TBI is estimated to 50 million cases worldwide, still varying a lot from one country to another, the highest rates of morbidity and mortality being recorded in low and middle-income countries [4].

In UK, TBI represents the most frequent cause of death and disability in people under 40 years of age [5], while in the US, it is estimated that 1.7 million people are affected by a traumatic brain injury every year [6,7].

The major causes of TBI are fallings, car accidents, terrorist attacks and sports injuries [8].

There was estimated that, every year, TBI causes losses of global economy of about 400 billion dollars in the US, which represents about 0.5% of the global raw income [6].

Only in the US, the total cost of TBI, including the direct costs of medical treatments and deaths, as well as of a low productivity, was estimated at 60.43 billion dollars [8], while in the European Union the annual costs for medical assistance in TBI exceed 33 billion euros [9].

The global incidence of TBI is about 200 in 100000 inhabitants, with a mortality of about 20 in 100000 inhabitants [10].

It is considered that TBI contributes by 30% to the mortality caused by traumatic injuries, being one of the major causes of invalidity all over the world [11].
The clinical classification of the severity of mild, moderate and severe TBI is made according to the Glasgow scale (Glasgow Coma Scale-GCS) according to the patient’s conscious state, motric changes, speech changes and eye opening [3,12,13].

Used ever since 1974, GCS is an important instrument that evaluates the severity of neurological deficits, it allows the performance of a triage of patients with severe brain injuries who require hospitalization in specialized intensive care units or neurosurgery units, also providing important information on the prognosis of TBI patients [14].

Severe TBI not only causes permanent neurological deficits (in about 20% of adults), but it has been shown that up to 14% of the patients remain in a vegetative state, while 20-40% of the patients die as a result of brain injuries or secondary complications [3].

In the US, fallings are the main cause of TBI, with over half a million visits to the emergency room and over 60000 hospitalizations every year [15].

Car accidents are responsible for most deaths of TBI in young adults.

In the European countries, fallings and car accidents are the first and the second most common cause of TBI [16], while in Asia, car accidents exceed the incidence and frequency of fallings [17].

In our study, we proposed to analyze severe TBI followed by death of the injured persons and the relation between the Glasgow score and the survival period.

Material and Methods

The study was performed on a number of 1005 cases of TBI, which underwent autopsy within the Forensic Medicine Institute of Craiova between 2011-2017 (for 7 years).

In order to perform the study, we utilised the data from the medical and forensics patient transcripts regarding deaths caused by TBI in adult persons (over 18 years of age) and in children (persons aged between 0 and 18 years old), respectively.

We analyzed the reports of forensic autopsy, general clinical transcripts from the hospital, with the medical imaging data, laboratory test results, the records made in the emergency room and the data provided by the authorities.

Most of the data were tabulated in Excel sheets and processed for descriptive analysis, including suggestive graphing and charts plotting.

The study was performed after all the protocols have been approved by the management and Ethics Committee of the Institute of Forensic Medicine of Craiova.

According to the present legislation, the measures complied ensured complete protection of personal data information.

Results

In the Institute of Forensic Medicine of Craiova, between January 1st 2011-December 31st 2017 there were performed a total number of 4626 autopsies, of which 1005 deaths (representing 21.72%) occurred as a result of a TBI; among these, 971 (96.62%) deaths occurred in adult persons (aged over 18 years old) and 34 (3.38%) in children (aged 0-18 years old).

Our study highlighted that TBI affected people of all ages, starting from babies up to elderly aged over 90 years old.

Following the age histogram of the patients included in the study group (Figure 1), it can be observed that most TBI-caused deaths, namely 752, representing 74.82% were recorded in the age group 40-79 years of age, with an average of 94 cases/ 5-year interval in this period; also in this age group, more precisely in the age group 60-64 years of age, there was also recorded the “peak” of TBI-caused deaths (109 cases, representing 10.84%).

If in children and young adults, in the age group 0-39 years of age, the number of TBI-caused deaths maintained at a relatively low scale, between 5 and 38 cases on a 5-year interval, which represents a percentage of 0.5-3.78%, the number of cases doubled in the age group 40-44 years of age, 77 cases representing 7.66%.

In the age group 45-79 years of age, the number of TBI-caused deaths was between 76 and 109 cases, a percentage representing between 7.56% and 10.84% for every age group of a 5 years interval.

In the age group 45-79 years of age there were recorded 675 cases, representing 67.16% of the TBI-caused deaths; in the age group 80-94 years of age there were recorded 83 cases (8.25% of the total group of TBI-caused deaths).

Regarding the death of injured persons, the forensics reports highlighted that some persons deceased immediately after the impact, while others survived for a longer period of time.

The number of patients deceased immediately after the impact was of
203 (20.20% of the TBI cases), of which 192 adults (19.10%) and 11 children (1.10%).

The number of patients presenting a period of survival after the injury occurred was of

| Survival Period | Number of Cases |
|-----------------|-----------------|
| <1 day          | 11              |
| 1-3 days        | 8               |
| 4-7 days        | 7               |
| 8-15 days       | 5               |
| >15 days        | 1               |

Most of the patients with TBI (785, representing 78.11%), who survived the initial impact with the vulnerating agent, were hospitalized. Among these, 764 were adults and 21 were children.

The survival period varied from less than 24 hours, up to over 10 days. In the first day of the injury, there were recorded 177 deceased patients (22.07%) of the initially surviving patients, of which 169 (21.07%) were adults and 8 (1.00%) were children; between 1 and 3 days survived 150 patients (18.70%), of which 148 (18.45%) were adults and 2 (0.25%) were children; between 4 and 7 days survived 202 (25.19%) patients, of which 195 (24.32%) were adults and 7 (0.87%) were children; between 8 and 15 days survived 182 (22.69%) patients, of which 177 (22.07%) were adults and 5 (0.62%) were children; over 15 days survived 91 (11.35%) patients, of which 90 (11.22%) were adults and 1 (0.13%) was a child (Figure 2, 3).

| Survival Period | Number of Cases |
|-----------------|-----------------|
| <1 day          | 192             |
| 1-3 days        | 169             |
| 4-7 days        | 148             |
| 8-15 days       | 195             |
| >15 days        | 177             |

Figure 1. Histogram of deceased TBI patients grouped on age groups (in years).

Figure 2. Distribution of children according to the survival period (WSP=without survival period).

Figure 3. Distribution of adults according to the survival period (WSP=without survival period).
Regarding the TBI causes, in our study we observed that their greatest number occurred as a result of fallings (438 cases, representing 43.58% of the total of deaths), followed by car accidents (333 cases representing 33.13%).

In the group of children cases, the death caused by falling was extremely rare (one case in 438 falling deaths), car accidents represent a more frequent cause of death in children, being recorded 26 deaths in this age group of the 333 cases of car accident deaths.

For evaluating the severity of the brain injuries in 649 of the patients hospitalized (82.67%) we used the Glasgow coma scale (GCS) or the Glasgow score, a score that was found in the clinical observation sheets of the patients. Of the patients evaluated on the Glasgow scale, 629 were adults and 20 were children, aged under 18 years old.

The Glasgow scale is an accurate and objective neurological method, frequently used in the neurotrauma hospitals in Romania, providing valuable data regarding the consciousness state of a person, the eye, speech and motric responses to various stimuli. It is used both for an initial evaluation of the TBI patient and for subsequent evaluations, in order to establish the neurological recovery and the efficiency of the applied treatment.

In our study, the clinical examination at admission, using the Glasgow scale, allowed a rapid evaluation of the severity of brain lesions and the choice of the best treatment methods.

Thus, 172 patients (133 men and 39 women) presented profound coma (GCS=3); other 171 patients (136 men and 35 women) presented 3rd degree coma (GCS=4-5), and 85 patients (60 men and 25 women) presented 2nd degree coma (GCS=6-7). The superficial coma (1st degree, GCS=8) was observed in 39 patients (31 men and 8 women).

Other 128 patients (107 men and 21 women) presented a slightly altered consciousness state (Glasgow score=9-14), while 34 patients (30 men and 4 women) preserved their consciousness from admission (Glasgow score=15) (Figure 4).

Under these circumstances, we may state that of the 629 adult patients admitted to hospital, 467 (74.24%) presented a 4th to 1st degree coma (with a Glasgow score from 3 to 8), while 162 (25.76%) were admitted with a preserved consciousness state.

Regarding the mortality of the adult patients who presented profound coma at admission, 80 (8.24%) deceased in the first day since the injury, 34 (3.5%) survived for 1 to 3 days, 30 (3.09%) survived between 4 and 7 days, 22 (2.27%) survived between 8 and 15 days and 6 (0.62%) patients had a longer survival period of 15 days.

The adult patients who presented a preserved or slightly altered consciousness state (non-comatose, Glasgow score >8) deceased in a number of 16 (1.65%) in the first day, 25 (2.57%) survived between 1 and 3 days, 42 (4.33%) patients survived between 4 and 7 days, 54 patients (5.56%) survived between 8 and 15 days and 25 patients (2.57%) survived more than 15 days.

In the hospitalized adult patients, emergency surgery was performed in 268 (27.60%) cases, the surgical intervention being delayed in 108 (11.12%) patients (Figure 5).
Of the 21 hospitalized children with TBI, in 20 (58.82% of all the children with TBI) of the patients the Glasgow scale in the clinical patient transcripts showed as follows: 17 patients (50%) presented profound coma at admission (GCS=3 points), while 3 patients (8.82%) presented 3rd degree coma (GCS=4-5).

In children, there were not diagnosed 2nd or 1st degree comas or the preservation of consciousness state, all the children having a deeply altered state of consciousness ever since hospital admission.

Regarding the survival period of children in a profound coma (GCS=3 points), 6 children (17.64%) deceased in the first day since the injury, a child (2.94%) survived for 3 days, 5 children (14.70%) had a survival period between 4 and 7 days and 5 children (14.70%) between 8 and 15 days.

In the case of children in a 3rd degree coma (GCS=4-5), a patient (2.94%) deceased after 3 days since the injury and 2 patients (5.88%) between 4 and 7 days after.

Regarding the surgical interventions in children, a single patient underwent emergency surgery, being performed the evacuation of the acute subdural haematoma over the left brain hemisphere.

**Discussion**

TBI lesions, according to their intensity, may cause the death of the patient or his survival with more or less disabilities, on a longer or shorter period of time.

Most often, TBI may lead to loss of consciousness, deterioration of the cognitive and motric ability, behavioral and/or emotional disorders, neurological deficiencies that may be temporary or permanent and may lead to a physical and psycho-social handicap [18,19].

Multiple clinical and statistical studies showed that the incidence, morbidity and mortality of TBI are quite varied from one geographical area to another and even from one country to another, being higher in low and moderate-income countries [4,20].

According to some studies, the TBI incidence increased during the decade 2001-2010; if between 2001 and 2005 the rates of TBI increased from 521 up to 616 in 100000 persons, between 2006 and 2010 they increased up to 824 in 100000 persons [21-22].

Among the patients hospitalized with severe TBI, approximately 43% will present a TBI secondary disability one year later [23, 24].

In our study, performed within the Forensic Medicine Institute of Craiova during a period of 7 years (2011-2017), we found that TBI were responsible for 21.72% of violent death cases.

A high mortality by TBI is recorded all over the world. In the US, between 1995 and 2009, there were recorded about 50000 deaths every year [25,26], while in 2013 there were recorded about 56000 deaths [27].

A tendency of increase in the mortality by TBI is also recorded all over the world as a result of the increase of work and car accidents.

In our study, we observed that severe TBI, followed by death, affected all age groups, being found from babies to 90-year-old people.

Our study, similar to other studies [6,28,29], observed that most severe TBI were found in adult persons and less in children.

Still, there should be mentioned that, every year, in the US only, over 500000 children suffer from traumatic brain injuries, of which more than 35000 require hospitalization and more than 2000 die [6,30].

TBI are produced by various mechanisms.

In our study, in adults, most deaths were caused by falling (43.58%), followed by car
accidents (33.13%); in contrast, in children most TBI ending in death of the child were caused by car accidents.

Our data is in accordance with other studies in the UE and US that showed most TBI are caused by falling, followed by car accidents [27,31,32].

TBI distribution as result of car accidents was quite varied, still they were more common in young people who have a lower driving experience, in general, they have a tendency to drive more aggressively and do not anticipate possible dangers, they drive under the influence of drugs or alcohol, etc. [33,34].

In our study, the highest number of deaths was observed in adults aged over 60 years old, namely 495 persons, representing 49.75% of the entire study group.

We consider that this fact is due to the presence of comorbidities that reduce the capacity of body response to physical external aggression.

The clinical studies performed up to now showed that there is a general consensus according to which the probability of death caused by TBI increases with age, even though the elderly are affected by less severe TBI.

The presence of an associated cardiac pathology, atherosclerosis, diabetes or of a coagulopathy significantly increases general mortality in the older adult population [35-37].

During the evaluation of the TBI patient, the neurological examination should be finalized as quickly as possible, even from the emergency room.

Besides the general clinical examination, the neurological examination should use the Glasgow scale for estimating the severity of brain injury, a method that is based on the TBI patient’s response to certain stimuli [38-40].

The Glasgow scale is considered an essential test in measuring the neurological function and the severity of brain injuries in patients with TBI; it is a simple, practical method that can be performed quite rapidly, it is efficient as far as costs are concerned and it helps in establishing the clinical diagnosis, of treatment conduct and prognosis for every patient.

In our study, we observed that a high number of patients (78.11%) with severe TBI were hospitalized, but the neurological evaluation with the Glasgow score could be performed only in 64.58% of the total number of TBI patients.

Among these, about 75% presented a 1st-4th degree coma, with a Glasgow score from 3 to 8 and only 25% had a slightly altered or preserved consciousness, with a Glasgow score=9-15.

There may be observed that also the patients in the latter TBI group deceased, one of the possible thanatogenerating mechanisms being the post-traumatic development, in a period of time varying from case to case, of hemorrhagic intracranial collections with a subsequent alteration of the consciousness.

In the last 30 years, the Glasgow scale was considered an accurate and specific marker for the clinical evaluation of traumatic brain injuries and for the choice of the most adequate treatment methods (brain surgery, over 24 hours intubation, hospitalization, etc.).

Also, up to now, the studies showed that there is a direct relation between the increase of mortality after a brain injury and the decrease of the GCS from 15 down to 3 [41-43].

Practically, the clinical severity of TBI has been evaluated by the GCS for a long time, as such: mild, with GCS values=14-15; moderate, with GCS values=9-13 and severe, with GCS values=3-8 [44,45].

Severe TBI have a mortality rate of 30-40% and may cause physical, psychosocial and social disabilities in over 60% of the patients who survive [46,47].

In our study, most patients who deceased had a GCS lower than 8, but about 25% of the TBI patients who were hospitalized had a GCS higher than 8 and still deceased.

This aspect makes us consider that mortality in TBI patients is influenced not only by the GCS but also by age and associated diseases.

Conclusions

TBI affects people of all ages, from babies to adults aged over 90 years old and they represent a major cause of mortality.

Most deaths by TBI occurred in adults, especially in the ones over 60 years old, due to present associated diseases in the elderly.

Most cases of TBI were caused by falling, followed by car accidents.

Of the total no 1005 of TBI-caused deaths, in the first 24 hours 380 patients (about 37%) died (of which 361 adults and 19 children), thus showing the severity if TBI on general mortality.

About 75% of the hospitalized patients had a Glasgow score=8 or lower and only 25% had a GCS higher than 8, which shows that brain damage may be serious starting from the impact with the injuring agent.

Although some patients had a GCS higher than 8, they deceased as a result of some
associated diseases or the development over time of intracranial blood collections.

That is why we consider that GCS is not an exact prediction factor if there are not taken into account the patient’s age and associated diseases.

Conflict of interests
None to declare.

References
1. Timofeev I, Santarius T, Kolias AG, Hutchinson PJA. Decompressive craniectomy - operative technique and perioperative care. Adv Tech Stand Neurosurg, 2012, 38:115-136.
2. McIntosh TK, Saatman KE, Raghupathi R. Time of intracranial blood collections. Neurosurgery, 2008, 63(2):382-384.
3. Reis C, Wang Y, Akyol O, Ho WM, Ii RA, Stier G. The Dorothy Russell Memorial Lecture. The molecular and cellular sequelae of experimental traumatic brain injury: pathogenetic mechanisms. Neuropathol Appl Neurobiol, 1998, 24(4):251-267.
4. Maas AIR, Menon DK, Adelson PD, Andelic N, Bell MJ, Belli A, Bragge P, Brazinova A, Büki A, Chesnut RM, Citerio G, Coburn M, Cooper DJ, Crowder AT, Czeteiter E, Czosnyka M, Diaz-Arrastia R, Dreier JP, Duhaime AC, Ercole A, van Essen TA, Feigin VL, Gao G, Giacino J, Gonzalez-Lara LE, Gruen RL, Gupta D, Hartings JA, Hill S, Jiang JY, Ketharanathan N, Kompanje EJO, Lanyon L, Laureys S, Lecky F, Levin H, Lingsma HF, Maeggele M, Majdan M, Manley G, Marsteller J, Mascia L, McFadyen C, Mondello S, Newcombe JA, Polinder S, Peul W, Piercy J, Polinder S, Puybasset L, Rasmussen TE, Rossaint R, Smielewski P, Söderberg J, Stanworth SJ, Stein MB, von Steinbüchel N, Steward W, Steyerberg EW, Stocchetti N, Synnot AJ, Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet, 1996, 348(8975):1044-1056.
5. Bader M, Li Y, Tweedie D, Shlobin NA, Bernstein A, Rubovitch V, Tovar-Y-Romo LB, DiMarchi RD, Hoffer BJ, Greig NH, Pick CG. Neuroprotective Effects and Treatment Potential of Incretin Mimetics in a Murine Model of Mild Traumatic Brain Injury. Front Cell Dev Biol, 2020, 7:35.
6. McIntosh TK, Maxwell WL, Nicoll JA. Recent advances in neurotrauma. J Neuropathol Exp Neurol, 2000, 59(8):641-651.
7. Alarcon JD, Rubiano AM, Okonkwo DO, Alarcon J, Martinez-Zapata MJ, Urrutia G, Bonfill Cosp X. Elevation of the head during intensive care management in people with severe traumatic brain injury. Cochrane Database Syst Rev, 2017, 12(12):CD009886.
8. Mangat HS. Hypertonic saline infusion for treating intracranial hypertension after severe traumatic brain injury. Crit Care, 2018, 22(1):37.
9. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet, 1974, 2(7872):81-84.
10. Marehbhan J, Muehlschlegel S, Edlow BL, Hinson HE, Hwang DY. Medical Management of the Severe Traumatic Brain Injury Patient. Neurocrit Care. 2017, 27(3):430-446.
11. Rau CS, Wu SC, Chen YC, Chien PC, Hsieh HY, Kuo PJ, Hsieh CH. Effect of Age on Glasgow Coma Scale in Patients with Moderate and Severe Traumatic Brain Injury: An Approach with Propensity Score-Matched Population. Int J Environ Res Public Health, 2017, 14(11):1378.
12. Barlow KM. Traumatic brain injury. In: DulacO, Lassonde M, Sarnat HB (Eds): Handbook of Clinical Neurology, Pediatric Neurology Part II, Elsevier, 2013, Amsterdam, 112:891-904.
13. Peeters W, van den Brande R, Polinder S, Brazinova A, Steyerberg EW, Lingsma HF, Maas AI. Epidemiology of traumatic brain injury in Europe. Acta Neurochir (Wien), 2015, 157(10):1683-1696.
14. Li M, Zhao Z, Yu G, Zhang J. Epidemiology of Traumatic Brain Injury over the World: A Systematic Review. Austin Neurol & Neurosci, 2016, 1(2):1007-1020.
15. Barker-Collo SL, Feigin VL. Capturing the spectrum: suggested standards for conducting population-based traumatic brain injury incidence studies. Neuroepidemiology, 2009, 32(1):1-3.
16. Angeloni C, Prata C, Dalla Sega FV, Piperno R, Hrelia S. Traumatic brain injury and NADPH oxidase: a deep relationship. Oxid Med Cell Longev, 2015, 2015:370312.
17. Reilly P. The impact of neurotrauma on society: an international perspective. Prog Brain Res, 2007, 161:3-9.
18. Kheirbek T, Adams CA Jr, Cioffi WG, Thomas KS. Population of Patients With Traumatic Brain Injury in Skilled Nursing Facilities: A Decade of Change. J Head Trauma Rehabil, 2019, 34(1):E39-E45.
19. Lueckel SN, Teno JM, Stephen AH, Benoit E, Kheirbek T, Adams CA Jr, Cioffi WG, Thomas KS. Population of Patients With Traumatic Brain Injury in Skilled Nursing Facilities: A Decade of Change. J Head Trauma Rehabil, 2019, 34(1):E39-E45.
20. Adalbert R, Nogradi A, Babetto E, Janeckova L, Walker SA, Kerschensteiner M, Misgeld T, Coleman MP. Severely dystrophic axons at amyloid plaques remain continuous and connected to viable cell bodies. Brain, 2009, 132( Pt 2):402-416.
23. Adibhatla RM, Hatcher JF. Lipid oxidation and peroxidation in CNS health and disease: from molecular mechanisms to therapeutic opportunities. Antioxid Redox Signal, 2010, 12(1):125-169.

24. Albert-Weissenberger C, Siren AL. Experimental traumatic brain injury. Experimental & translational stroke medicine. 2010, 2:16.

25. Coronado VG, McGuire LC, Sarmiento K, Bell J, Lionbarger MR, Jones CD, Geller AI, Khoury N, Xu L. Trends in Traumatic Brain Injury in the U.S. and the public health response: 1995-2009. J Safety Res, 2012, 43(4):299-307.

26. Fazel S, Wolf A, Pillas D, Lichtenstein P, Långström N. Suicide, fatal injuries, and other causes of premature mortality in patients with traumatic brain injury: a 41-year Swedish population study. JAMA Psychiatry, 2014, 71(3):326-233.

27. Taylor CA, Bell JM, Breiding MJ, Xu L. Traumatic Brain Injury-Related Emergency Department Visits, Hospitalizations, and Deaths-United States, 2007 and 2013. MMWR Surveill Summ, 2017, 66(9):1-16.

28. Koskinen S, Alaransta H. Traumatic brain injury in Finland 1991-2005: a nationwide register study of hospitalized and fatal TBI. Brain Inj, 2008, 22(3):205-214.

29. Andelic N, Anke A, Skandsen T, Sigurdardottir S, Sandhaug M, Ader T, Roe C. Incidence of hospital-admitted severe traumatic brain injury and in-hospital fatality in Norway: a national cohort study. Neuroepidemiology, 2012, 38(4):259-267.

30. Bennett KS, DeWitt PE, Harlaar N, Bennett TD. Seizures in Children With Severe Traumatic Brain Injury. Pediart Crit Care Med, 2017, 18(1):54-63.

31. Tagliaferri F, Compagnone C, Korsic M, Servadei F, Kraus J. A systematic review of brain injury epidemiology in Europe. Acta Neurochir (Wien), 2006, 148(3):255-268.

32. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. J Head Trauma Rehabil, 2006, 21(5):375-378.

33. Washington, DC: National Highway Traffic Safety Administration, 2016, Occupant Restraint Use in 2014: Results from the NOPUS Controlled Intersection Study [online]. Available at: https://crashstats.nhtsa.dot.gov/Ap/Public/Publication/812244 [Accessed 15.09. 2020].

34. Washington, DC: National Center for Statistics and Analysis, 2014, Alcohol-impaired driving: 2013 data (Report No. DOT HS 812 102) [online]. Available at: https://crashstats.nhtsa.dot.gov/Ap/Public/Publication/812102 [Accessed 18.09. 2020].

35. Susman M, DiRusso SM, Sullivan T, Risucci D, Nealon P, Cuff S, Haider A, Benzil D. Traumatic brain injury in the elderly: increased mortality and worse functional outcome at discharge despite lower injury severity. J Trauma, 2002, 53(2):219-223.

36. Thompson HJ, McCormick WC, Kagan SH. Traumatic brain injury in older adults: epidemiology, outcomes, and future implications. J Am Geriatr Soc, 2006, 54(10):1590-1595.

37. Mushkudiani NA, Engel DC, Steyercber EW, Butcher I, Lu J, Marmarou A, Slieker F, McHugh GS, Murray GD, Maas AI. Prognostic value of demographic characteristics in traumatic brain injury: results from the IMPACT study. J Neurotrauma, 2007, 24(2):259-269.

38. Davis DP, Vadeboncoeur TF, Ochs M, Poste JC, Vilke GM, Hoyt DB. The association between field Glasgow Coma Scale score and outcome in patients undergoing paramedic rapid sequence intubation. J Emerg Med, 2005, 29(4):391-397.

39. Galgano M, Toshkezi G, Qiu X, Russell T, Chin L, Zhao LR. Traumatic Brain Injury: Current Treatment Strategies and Future Endeavors. Cell Transplant, 2017, 26(7):1118-1130.

40. Nik A, Sheikh Andalib MS, Ehsaai MR, Zarifian A, Ghayoor Karimian E, Bahadoor Khan G. The Efficacy of Glasgow Coma Scale (GCS) Score and Acute Physiology and Chronic Health Evaluation (APACHE) II for Predicting Hospital Mortality of ICU Patients with Acute Traumatic Brain Injury. Bull Emerg Trauma, 2018, 6(2):141-145.

41. Borgiali DA, Mahajan P, Hoyle JD, Powell EC, Nadel FM, Tunik MG, Foerster A, Dong L, Miskin M, Dayan PS, Holmes JF, Kuppermann N, Pediatric Emergency Care Applied Research Network (PECARN). Performance of the Pediatric Glasgow Coma Scale Score in the Evaluation of Children With Blunt Head Trauma. Acad Emerg Med, 2016, 23(8):878-884.

42. Reith FCM, Lingsma HF, Gabbe BJ, Lecky FE, Roberts I, Maas AIR. Differential effects of the Glasgow Coma Scale Score and its Components: An analysis of 54,069 patients with traumatic brain injury. Injury. 2017, 48(9):1932-1943.

43. Steyercber EW, Mushkudiani N, Perel P, Butcher I, Lu J, McHugh GS, Murray GD, Marmarou A, Roberts I, Habbema JD, Maas AI. Predicting outcome after traumatic brain injury: development and international validation of prognostic scores based on admission characteristics. PLoS Med. 2008, 5(8):e165:1251-1261.

44. Teasdale G, Jennett B Assessment of coma and impaired consciousness: a practical scale. Lancet,1974, 304(7872):81- 84.

45. Hartings JA, Bullock MR, Okonkwo DO, Murray LS, Murray GD, Fabricius M, Maas AI, Woltzik J, Sakowitz O, Mathern B, Roozenbeek B, Lingsma H, Dreier JP, Puccio AM, Shutter LA, Pahl C, Strong AJ, Co-Operative Study on Brain Injury Treatment Strategies and Future Endeavors. Cell Transplant. 2017, 26(7):1118-1130.

46. Dikmen SS, Machamer JE, Powell JM, Temkin NR. Outcome 3- 5 years after moderate to severe traumatic brain injury. Arch Phys Med Rehabil, 2003, 84(10):1449-1457.

47. Mena JH, Sanchez AI, Rubiano AM, Peitzman AB, Sperry JL, Gutierrez MI, Puyana JC. Effect of the modified Glasgow Coma Scale score criteria for mild traumatic brain injury on mortality prediction: comparing classic and modified Glasgow Coma Scale score model scores of 13. J Trauma, 2011, 71(5):1185-1192.