Published in final edited form as:
J Head Trauma Rehabil. 2012 ; 27(2): 87–98. doi:10.1097/HTR.0b013e3182114efd.

Association of depressive symptoms with functional outcome after traumatic brain injury

A. M. Hudak[1], **L. S. Hynan**[2], **C. R. Harper**[3], and **R. Diaz-Arrastia**[3]

[1] Department of Physical Medicine and Rehabilitation, University of Texas Southwestern Medical Center, Dallas, TX

[2] Departments of Clinical Sciences (Division of Biostatistics) and Psychiatry, University of Texas Southwestern Medical Center, Dallas, TX

[3] Department of Neurology, University of Texas Southwestern Medical Center, Dallas, TX

Abstract

Objective—To test whether improved functional status correlates with more depressive symptoms following traumatic brain injury (TBI). This is based on the concept that increasing awareness of deficits may exacerbate depression, even while survivors are making functional improvements.

Setting—Discharge from private or public hospital in major metropolitan area.

Participants—471 individuals with TBI (72 % Caucasian; 71% male; median Glasgow Coma Scale score =11; median follow-up period = 6 months).

Main Measure—Beck Depression Inventory-II (BDI-II), Glasgow Outcome Scale-Extended (GOS-E), Functional Status Examination (FSE)

Results—We found significant Spearman's rank order correlations between BDI-II scores and the total FSE as well as all domains of the FSE. Lower functional levels correlated with more depressive symptoms. Modeling of predictive factors, including subject characteristics, injury related characteristics and outcome measures, resulted in 2 models, both containing age and GCS along with other factors.

Conclusion—The relation between depressive symptoms and functional outcomes is complex and a fertile area for further research. The authors would encourage clinicians to monitor patients for depressive symptoms to help prevent the detrimental impact on recovery.

Introduction

During their recovery and for the duration of their lives, traumatic brain injury (TBI) survivors of all severity levels face numerous obstacles. One of the most common challenges, depression, is found throughout the spectrum of TBI severity, but most focus on mild to moderate injuries, reflecting rates of injury severity[1]. During the first 12 months after TBI, rates of major depressive disorder (MDD) determined by diagnostic interview range between 20-30%[2–6]. Most recently, Bombardier[7] found 53.1% of their population to have had MDD at least once during the first 12 months after TBI. By comparison, 12 month rates of MDD in the general population are reported to be 6.6%[8] and 16.6%[9] for lifetime incidence.

Depression after TBI negatively influences cognition[10–12], executive function [12–14], psychosocial function [11, 13], employment [15,16], social and recreational activities [10, 13, 17–20], social roles[7, 11], and functional outcomes[10, 15, 19, 21–23]. Those with post-TBI depression perceive their own injury and outcome to be worse[17]. While the pathophysiology of post-TBI depression is currently unknown, the impact of early depression on functional outcomes may be due, in part, to decreased participation in rehabilitation. In the general population, depression is associated with impairment of executive function[24].

Investigations of the relation between injury severity and development of post-TBI depression have yielded mixed results. Some researchers have found no relation[7, 25], a positive link[2], or an inverse link[3] between injury severity and depressive symptoms. Some authors have theorized that milder injuries produce less cognitive dysfunction and, consequently, more awareness of functional deficits, leading to an increased risk of depressive symptoms[26]. Subjects with greater awareness of deficits are more likely to complain of depression[27–31]. Others have found that poor self-awareness leads to higher levels of psychological distress and worse psychosocial outcome[32]. Some studies indicate that those whose injury occurred greater than 6 months prior to the mood assessment were more aware of their deficits and exhibited greater emotional distress[27, 28, 33, 34]. The relation between the development of post-TBI depression and injury mechanism was explored by Glenn[35] who found a negative relation between violence as a mechanism of injury and depression after mild TBI. Bombardier[7] did not find a significant relation between violent injuries and post-TBI MDD including all levels of injury severity. Others exploring this relation in the context of mass violence have reported relations among TBI, depression and post-traumatic stress disorder (PTSD)[36, 37]. The evidence for the link between educational level and development of post-TBI depression has been mixed, with some associating a lower level of education with depression[31, 38, 39] while others found no impact[2, 7, 13].

To achieve a better understanding of the connection between functional abilities and post-TBI depressive symptoms, the relatively novel Functional Status Examination[40] (FSE) was used to examine this relation. Compared to more standard measures of functional outcome such as the Glasgow Outcome Scale-Extended[41] (GOS-E) and the Disability Rating Scale[42] (DRS), the FSE explicitly examines domains of Cognitive Function and Social Integration as well as standard domains of Mobility and Personal Care[40, 43]. It takes into account the scaffold of the World Health Organization[44] of disability, handicap and impairment. Developed with consideration of the reported limitations of existing scales, it was designed to address the lack of sensitivity and low ceiling effects[40] of earlier scales. The FSE explores changes in abilities as a function of health, including TBI, and includes the full spectrum of possible outcomes. The FSE (measured by self-report) has been previously correlated with depressive symptoms measured by the CES-D[45], with a Spearman's rank order coefficient of 0.42 ($p<0.001$).

While multiple factors may influence the development of depression, the hypothesis is that the lower FSE scores, indicating better function, will correlate with higher Beck Depression Inventory-II[46] (BDI-II) scores (increasing symptoms of depression). We theorize that as persons with TBI improve functionally, they become more self-aware, and depression ensues as they struggle to accept the outcome of a life-altering event. We also investigate the impact of injury mechanism on the development of significant depressive symptoms. Specifically, we hypothesize that injuries sustained through violent means will be more associated with depressive symptoms. Potential predictive factors of depression after TBI are examined in an exploratory manner.

Methods

We started with a database of 1279 potential participants with TBI who were part of a prospective study between February 2004 and July 2009, having been recruited from two sites encompassing both private and public assistance patients in a major U.S. metropolitan area. The initial study inclusion criteria required 1) TBI needing hospitalization; 2) greater than 14 years old; 3) informed consent obtained. Demographic and injury related data was collected at enrollment. The Glasgow Coma Score47 (GCS) was the lowest score within 24 hours of admission. Participants were contacted 6 to 12 months after injury for a telephone interview that included the Beck Depression Inventory-II, the Glasgow Outcome Scale-Extended (GOS-E), and the Functional Status Examination (FSE).

For this study, the inclusion criteria were 1) age at injury between 14 to 65 years; 2) subject participation in the structured telephone interview during which the BDI-II was collected. Of the 1279 individuals included in the database, 334 were lost to follow up; 474 had information provided by surrogates; and 471 met the required inclusion criteria. Those who did not have the BDI-II measure were either unable to independently participate in the interview due to poor cognitive function, or had a surrogate complete the phone interview. Interviewers were experienced in communication and interaction with persons with TBI. During completion of the demographic and functional questions posed during the interview, the participant's ability to accurately complete the BDI-II was subjectively determined by interviewers.

The original project (Genetic Factors in Outcome after Traumatic Brain Injury R01 HD48179, Diaz-Arrastia, PI) from which these data are drawn received institutional review board approval. De-identified data were used for this retrospective study; therefore, this study did not require institutional review board approval, but was registered.

Measures

GCS

The Glasgow Coma Scale47 assesses depth of impaired consciousness and coma using an ordinal scale consisting of three components (motor, eyes, and verbal). Total scores are the sum of the components and range from 3 to 15 with lower scores representing lower function. In an article combining data from multiple sources with persons in coma from a variety of causes, Prasad48 reports reliability with trained users to be 0.8-1 (intraclass correlation coefficient).

FSE

Administered via a structured interview, the Functional Status Examination40 is a 10 domain measure of outcome designed specifically to evaluate changes in daily activities after traumatic brain injury. The ten domains are: Social Integration, Leisure and Recreation, Home Management, Travel, Executive Function, Ambulation, Major Activity Involving Work or School, Financial Independence, Personal Care, and Standard of Living. Comparing current functional activities to pre-injury function, scores range from 9 to 41, with lower scores indicating better function. A score of 41 means the subject has expired. When administered by trained personnel40, 43, 49, the validity (between FSE and GOS rho = -0.72; FSE and GOS-E rho = -0.83) and test-retest reproducibility (rho = 0.8) have been established in the TBI population. One of the FSE's advantages over the GOS-E is the instrument's ability to provide a more descriptive functional picture.

The FSE asks participants to rate their functional abilities framed in reference to pre-injury function. The questions are usually read: "Due to your injury, is anyone helping you more now with (functional activity)," followed by a series of yes/no questions that ultimately results in a score for each domain. Scores are ordinal with four levels (1-4), except for standard of living which adds a level indicating post-injury improvement (5 levels: 0-4). A score of 1 represents no change in function from pre-injury level. When a subject is not working or going to school (retired, working in the home or unemployed both before and after the injury), the scores for the other nine domains are averaged. This averaged score is then used to represent the value for work activities. It is added into the scores for the other nine domains to obtain the total FSE score.

GOS-E

The Glasgow Outcome Scale-Extended[41] is an 8-level ordinal scale measuring outcome after injury, with 1 meaning death and 8 meaning "upper good recovery". It is administered via a structured interview[41, 50] and has been validated for use via telephone[51]. In a population of TBI survivors, the inter-rater reliability kappa was reported to be 0.84; test-retest reliability was reported to be 0.92[51].

BDI-II

The Beck Depression Inventory-II[46] is a commonly used 21-question measure of depression symptom severity. Each item is scored 0 to 3, with lower scores representing lesser symptoms of depression. Total scores range from 0 to 61. Cut offs used in this study are consistent with those given in the BDI-II Manual[46]; 0–13 corresponds to "none to minimal" depression and scores greater than 13 correspond to "clinically symptomatic" depression. The internal consistency was reported to be 0.89-0.91[52-54] in separate populations of undergraduate students. Using an ROC analysis, Homaifar[55] reported on the sensitivity and specificity of the BDI-II in the TBI population. They recommend using different dividing points for depression in the TBI population based on severity of injury. For those with mild TBI, a BDI-II score greater than 18 is recommended to be considered depressed; for moderate to severe TBI, score greater than 34 was considered depressed. These dividing points provide sensitivity of 0.87 and specificity of 0.79. The factor structure of the BDI-II in the TBI population has been analyzed. Using a relatively small sample size and the traditional cut-off of ≥14 for depression, Rowland[56] reported a 3 factor structure. There is no information available regarding the validity of telephone administration of the BDI-II.

Statistical Methods

Medians and inter-quartile ranges (IQR) are reported for non-normally distributed measures. Spearman rank order correlations (rho) were performed between BDI-II scores and participant characteristics, and between BDI-II and outcomes. Where indicated, BDI-II scores were dichotomized at the clinically significant cutoff (> 13 according to BDI-II manual[46], or using divisions proposed by Homaifar[55] : mild TBI >18 or moderate-severe >34); groups are referred to as "not depressed" for those scores lower than the cut point and "depressed" for those scores higher than the cut point. Severity divisions of the BDI-II per the manual (minimal = 0-13; mild = 14-18; moderate = 19-28; severe >28) were compared using Kruskal-Wallis. A Mann Whitney U test was used to evaluate the difference of outcome measures in the not depressed and depressed groups. For any variable that does not have the equivalent number of subjects as the group, the missing number is attributed to missing data.

The relation between mechanism of injury and depressive symptoms was explored. Kruskal Wallis was performed to analyze the impact of injury mechanism on outcome measures. The mechanism of injury was categorized into four groups. The violence group included the following injury mechanisms: aggravated assault, penetrating and non-penetrating violence, and gunshot wounds. Motorized vehicle category included collisions of motor vehicles, motor cycles, all-terrain vehicles and other vehicles. Sports category included track and field, water, and bicycle related injuries. The other category includes falls, hit by falling objects, motor pedestrian injuries and other unknown causes. The nonparametric multiple comparisons with unequal sample sizes (Dunn Method) method was used to evaluate the group interactions for those measures that were significant.

Logistic regression, using the stepwise procedure, was performed to identify potential factors predictive of depression (BDI-II). Models included age, educational level, injury severity measure (either GCS or duration of hospital stay), injury cause, gender, GOS-E, and the total score of the FSE, or the domains of FSE as predictors. Given the exploratory nature of these analyses, P was set at 0.1557.

Analyses were performed using SPSS version 1758 and SAS version 9.259. Nonparametric analyses were performed because assumptions for parametric analyses were violated. Statistical significance was set at p < 0.05 using two tailed hypothesis tests.

Results

Total Group

The overall group consisted primarily of Caucasian (72%, N=338) males (71%, N=335) whose median age was 32 years (N= 470, IQR 21-49). The next highest enrolling group was Hispanics at 17% (N=81). The median educational level was high school diploma (N=337, IQR 11-15 yrs of education). The median GCS was 11 (N=450, IQR 3-15). The most common injury mechanism was motorized vehicle (57%, N=267). The median length of hospitalization was 12 days (N=428, IQR 5-22). The median time of follow up was 6 months (N= 466, IQR 6-8 months) at which time the median GOS-E was 7 (N= 466, IQR 5-8); the median FSE was 15 (N=442, IQR 11-23); and the median BDI-II score was 4 (N= 471, IQR 0-11). Of the total group, 19% (N=87) had clinically significant depressive symptoms by the BDI-II manual division. The correlations between BDI-II and FSE (N = 442, rho = 0.510, p < 0.0001) and between BDI-II and GOS-E (N = 466, rho = -0.416, p < 0.0001) were significant, but that between BDI-II and the time of outcome data collection was not significant (N = 466, rho = 0.056, p = 0.237). The correlations of time of data collection with the GOSE (N=459, rho = 0.0182, p=0.6967) and FSE (N= 436, rho = 0.0033, p=0.9452) were not significant. The correlations between the BDI-II and each domain of the FSE were significant (p < 0.0001). Please refer to Table 1. Of the domains, the strongest correlations were for Social Integration and Leisure and Recreation.

Division by TBI severity

Mild Injury

The mild TBI group, defined as those with GCS of 13 to 15 (N= 214), consisted primarily of Caucasian males and is described in Table 2. The mechanisms of injury were motor vehicle related for 41 % (N=87), other category for 42 % (N = 90), violence for 13% (N=27), and sports related for 3% (N= 7). The correlation between BDI-II and duration of hospital stay (N=194, rho=0.178, p=0.0128), GOS-E (N=213, rho = -0.425, p<0.0001) and FSE (N= 204, rho = 0.530, p<0.0001) were found to be significant, whereas age, educational level, time to follow up and GCS were not significant. Using the BDI-II manual divisions, 17% (N=37) of this group would be considered to have clinically significant depressive symptoms.

Moderate-severe Injury

The moderate-severe injury group was defined as subjects with a GCS less than 13 (N=237). The group consisted primarily of Caucasian males who were injured by motorized vehicle (72%, N=170). The next most common injury mechanism was other mechanism (22%, N=51), followed by violence (3%, N=6) and sports (2%, N=4). This group is described in Table 2. The correlation between BDI-II and GCS, duration of hospital stay, educational level, time to follow up, GOS-E, and FSE were examined and only age (N=237, rho = 0.143, p =0.0281), GOS-E (N = 234, rho = -0.391, p <0.0001) and FSE (N = 220, rho = 0.468, p <0.0001) were significantly correlated. Using the traditional boundaries of the BDI-II (BDI-II >13), 21% (N=43) would be considered to have significant depressive symptoms.

Mann Whitney U was used to analyze difference between mild TBI and moderate-severe TBI. There were significant differences between age at injury (p<0.0001), educational level (p=0.0033), days of hospital stay (p<0.0001), BDI-II (p=0.0113), GOS-E (p<0.0001), FSE total (p<0.0001), and 8 of the 10 domains of the FSE (personal care p=0.0005, ambulation p= 0.0019, travel p<0.0001, work p<0.0001, leisure and recreation p=0.0004, home management p= 0.0002, cognition p=0.0001, financial independence p<0.0001).

Subjects Missing GCS

Subjects who were missing GCS (N=20) were transferred from regional acute care hospitals to an inpatient rehabilitation unit. While imaging confirmed the presence of TBI, the initial GCS was not located within the transfer documents. This group primarily contained Caucasian (N=17, 85%) males (70%, N=14) with a median age of 49 (N=19, 23-55). The correlation between BDI-II and FSE (N=18, rho 0.567, p=0.0141) was significant, however, the correlation with GOS-E was not (N=19, rho = -0.214, p=0.3795). The BDI-II manual divisions indicate 5% (N=1) of this group had clinically significant depressive symptoms.

BDI-II Symptom Severity Grouping by the Manual

Using the BDI-II manual divisions, the population was subdivided into depressive symptoms severity: minimal (BDI-II 0-13, N=384), mild (BDI-II 14-18, N=38), moderate (BDI-II 19-28, N=33) and severe group (BDI-II > 28, N=16). Please see Table 3 for group details. For the minimally depressed group, there were significant correlations between the BDI-II and GCS (N= 365, rho=-0.173, p=0.0009), hospital stay (N=348, rho=0.159, p=0.003), GOS-E (N= 381, rho =-0.271, p<0.0001) and FSE (N= 363, rho= 0.353, p <0.0001). For the mildly depressed group, there were significant correlations between the BDI-II and age (N=38, rho=-0.332, p=0.042), hospital stay (N=34, rho=-0.432, p=0.011), and GOS-E (N=37, rho =0.450, p=0.0052). For the moderately depressed and the severely depressed groups, there were no significant correlations between the BDI-II and other variables.

The Kruskal Wallis test was performed to compare the four groups stratified by BDI-II manual divisions. All had 3 degrees of freedom. The educational level, FSE and GOS-E were significantly different (education χ^2 = 9.67 p=0.0216; FSE χ^2 = 85.07, p<0.0001; GOS-E χ^2 = 63.6, p<0.0001). Age and duration of hospital stay were not significant.

When the total group of subjects was dichotomized by depressive symptoms (BDI-II ≤ 13 = not depressed, BDI-II > 13 = depressed), no between group differences in age, GCS, length of hospital stay, or time to follow up were found. There were significant differences between the not depressed and depressed groups for educational level (median 12, IQR 11-15, N=278 vs. 12, IQR 10-12, N=59; Mann-Whitney U p = 0.0059), GOS-E (median 7, IQR 5-8, N=381 vs. median 5, IQR 3-6, N=85; Mann Whitney U p<0.0001) and FSE (median 14, IQR 10-20, N=363, vs. 27 IQR 20-31, N=79; Mann Whitney U p<0.0001). There were significant differences between the depressed and not depressed groups for all domains of the FSE (p<0.0001 for each). The FSE and GOS-E for the depressed group indicated worse functional status when compared to the not depressed group. The correlation between BDI-II and GOS-E (not depressed: N=381, rho = -0.271, p<0.0001; depressed: N=85, rho= 0.185, p=0.0906) and FSE (not depressed: N= 363, rho = 0.353, p<0.0001; depressed: N=79, rho=0.028, p=0.804) were significant for the not depressed group only.

BDI-II Score Divisions based on TBI severity (Homaifar55)

Based on their GCS, subjects were divided into mild (GCS 13-15), and moderate-severe (GCS 3 to 12) injury. Using the Homaifar55 divisions, 12% (25/214) of the mild injury subjects and 1% (3/237) of the moderate to severe injury subjects would be considered to have clinically significant depression. These groups are fully described in Table 4.

Mild TBI

For the mild TBI-not depressed group (BDI-II <19 and GCS >12), the significant correlations with the BDI-II were GCS (N=189, rho = -0.143, p =0.0499), duration of hospital stay (N=170, rho = 0.195, p=0.0108), GOS-E (N=188, rho = -0.310, p<0.0001), and FSE (N=180, rho = 0.410, p<0.0001). For the mild TBI-depressed group (BDI-II >18 and GCS >12), no significant correlations were found between the BDI-II and the other variables. For the mild TBI category, a Mann Whitney U test was performed to examine the between group differences (depressed vs. not depressed). The only significant differences were for the GOS-E (p<0.0001) and FSE (p<0.0001).

Moderate-severe TBI

For the moderate-severe TBI-not depressed group (BDI-II < 35 and GCS < 13), the significant correlations were between the BDI-II and the GOS-E (N=231, rho = -0.375, p<0.0001) and FSE (N=217, rho = 0.452, p<0.0001). For the moderate-severe TBI-depressed group (BDI-II > 34 and GCS <13), there were only three subjects, two of whom were female. The median age was 32 (N=3, IQR 23-41) and the median GCS was 6 (N=3, IQR 3-7). The educational level was missing for all. The mechanism of injury for the entire group was other mechanisms. The median GOS-E was 3 (N=3, IQR 3-4); the median FSE was 33 (N=3, IQR 28-37); the median BDI-II was 42 (N=3, IQR 38-59). The small number of subjects in the moderate to severe TBI-depressed did not permit use of correlation analysis or the Mann Whitney U test.

NIH-PA Author Manuscript

Impact of Mechanism of Injury for total sample

We used the Kruskal Wallis test to examine the association of injury mechanism on demographic and outcome variables. We found significant associations with age, GCS, BDI-II, and the FSE domains of Ambulation and Travel. Trends were found for GOS-E, FSE total score, and the FSE domain of social integration (Table 5). Of the above statistically significant variables, the Dunn Method showed significant post hoc differences (p < 0.05) between injury mechanism categories of violence and other mechanism categories and between vehicle and other mechanism categories for age. Significant post hoc differences were found for GCS (p < 0.05) between violence and motorized vehicle as injury causes, and motorized vehicle and other mechanisms. Significant post hoc differences were found for BDI-II (p<0.05) between motorized vehicle and other mechanism and between sports and other mechanisms.

NIH-PA Author Manuscript

Modeling

Four logistic regression models were examined. Variables included in each model were age, educational level, gender, GOS-E, and sports as an injury mechanism. Varied among the models was the use of GCS or hospital length of stay as an indicator of injury severity and the total FSE or the domains of the FSE, as functional indicators. Two models were chosen. The best fitting model using the total FSE as a functional indicator and the best fitting model using the domains of the FSE as a more granular functional indicator are described. Of the models with the total FSE, the best model (model 1) contained total FSE, age, and GCS. The model fit to the data was excellent (Hosmer-Lemeshow p= 0.743). Of the models containing the FSE domains, the best model (model 2) contained the domains of Social Integration, Standard of Living, and Home Management, as well as GOS-E, education, and GCS. The model fit to the data was again found to be excellent (Hosmer-Lemeshow p= 0.792). See Table 6.

Discussion

The purpose of this study was to examine the relation between functional abilities measured by the FSE and post-TBI depressive symptoms measured by BDI-II. The gender distribution of this population is consistent with published reports concerning the ratio of males to females sustaining a TBI. The median age of this population was higher than the national reports of TBI patients who present to the emergency department, where the peak incidence occurs in the 15 to 19 year olds and the 0-4 years[60], but younger than those reported by Pickelsimer's[61] population of hospitalized subjects (54% were younger than 45 years). This population may be skewed due to the availability of a pediatric trauma facility, where patients below the age of 14 years are transported. The most common mechanism of injury was motorized vehicle-related, consistent with published reports of hospitalized TBI patients[61]. In the total sample, the rate of depression (using BDI-II manual divisions), is consistent with published reports[2–6].

A comparison of persons with mild injury with those who sustained moderate-severe injury revealed significant between group differences for age, educational level, hospital length of stay, BDI-II, GOS-E, FSE total, and most domains of the FSE.

When the population was divided according to the BDI-II manual divisions (i.e., not depressed vs. depressed), there were no statistically significant group differences for age, GCS, duration of hospital stay and time to follow up. The depressed group had a significantly lower level of education. At the time of outcome measure (6 months), the depressed group had worse function measured by both the GOS-E and FSE and all the domains of the FSE. Dividing BDI-II scores into levels of depression revealed significant between group differences for educational level, FSE and GOS-E.

Using the Homaifar[55] divisions of the BDI-II for mild TBI, the only significant differences between the not depressed and depressed groups involved the GOS-E and FSE, where the depressed group had lower functional levels. The rate of significant depression symptoms drops substantially when the using the divisions proposed by Homaifar[55]. A similar analysis could not be performed for the moderate to severely injured group due to small sample size.

The correlations between BDI-II and the FSE and GOS-E were of mild to moderate strength for the total group, the mild injury group, the moderate-severe injury group, the BDI-II manual division not depressed group, the mild injury Homaifar[55] not depressed group and the moderate to severe injury Homaifar[55] not depressed group. For each significant correlation, the correlation was always stronger between the BDI-II and the FSE than with the GOS-E, except for the mildly depressed group (BDI-II manual divisions), where the correlation was stronger for between the GOS-E and BDI-II. Worse function (GOS-E) has previously been correlated with depression symptoms, but only one study reported using the more finely grained FSE[45]. Our results reveal a stronger correlation between the FSE and BDI-II than was found between the FSE and CES-D (rho 0.51 vs. 0.42, respectively).

The primary hypothesis for improving function correlating with worsening depression symptoms was supported in the mildly depressed group (BDI-II manual) for the GOS-E, but not with the FSE. This group shows an increase in symptoms correlating with better function. For the total population, mild TBI, moderate-severe TBI, BDI-II manual not depressed, mild TBI not depressed (Homaifar[55] scoring), and moderate-severe not depressed (Homaifar[55] scoring), significant but inverse relations for both the FSE and GOS-E are reported. In these instances, more severe depression symptoms correlated with worse function. For the entire population, the domains of the FSE when examined for individual correlation with BDI-II were all significant. The strongest relation with depression symptoms was found with the Social Integration domain of the FSE. This domain also plays a strong role in Model 2. The influence of depressive symptoms on social integration after TBI has been previously reported[11, 32, 60]. The domains of the FSE encompass more than physical function and may in fact have some overlap with qualities measured in the BDI-II. This would explain the predominant finding of stronger correlations between the FSE and the BDI-II. The ability of the FSE to measure individual domains makes it an instrument that permits a better understanding of the components of function, rather than providing a global less precise assessment.

Interestingly, the time of outcome measurement did not correlate with either the FSE or the GOS-E, indicating that is not strictly the passage of time since injury affecting these findings. A possible explanation is the degree of functional recovery, which tends to improve with time. This may have been influenced by the distribution of assessment time, with the majority clustered around the six month time. Our findings are not consistent with previous longitudinal reports of point prevalence of depression within the first year after TBI, showing a decrease as time from injury increases[2, 13, 22, 34, 39, 62]. Some authors have theorized that there are subpopulations of depression after TBI, whose mechanism and onset of depression differ[63]. Drawing from Prigatano's work[63], Moldover[64] theorized that reactive depression occurs when sufficient time had elapsed for the patient to have improved self awareness to be cognizant of their new functional state. They place this period as occurring about 7 to 12 months after injury. Pagulayan's[62] work indicates worse function precedes depression.

The impact of the method of injury, specifically violence, was examined for its influence on depressive symptoms. We hypothesized that violent injuries may be more associated with depressive symptoms. Contrary to what was reported by Glenn[35] and consistent with Bombardier's[7] findings, violence as a mechanism of injury did not influence the appearance of depressive symptoms in this population. This is especially interesting when the potential co-existence of post-traumatic stress disorder is considered. Work in the mass violence realm indicates strong associations between violently acquired TBI and depression but less so between brain injury and PTSD[36]. The variations in these findings may be due to pre-morbid personality and social support, neuro-anatomical differences of the injury, and differences in psychosocial support after injury.

Comparison of the educational level for the total population categorized by the BDI-II manual divisions revealed a significantly lower educational level for the depressed group. This is a confirmation of earlier work[23, 39] and may be a reflection of the additional socio-economic stressors in the under-educated population.

Two models were developed using exploratory modeling. Both models included GCS over hospital length of stay, indicating the level of brain injury severity plays a role in the development of depressive symptoms. The remaining components of models 1 and 2 diverge. Model 1 contains total FSE score and age in addition to GCS. Model 2 contains educational level, GOS-E, and the FSE domains of Social Integration, Standard of Living and Home Management. The association of educational level and the development of depression symptoms after TBI has previously been reported[23, 65] and Model 1 supports the link. The standard of living domain reflects the change in financial situations and may reflect economic stress triggering or magnifying symptoms of depression. Further, lower educational levels are often found in the lower socio-economic strata. Functional outcome measured by GOS-E support the link between depressive symptoms and functionality. The Social Integration factor may reflect the isolation experienced by many TBI survivors, or may reflect a continuation of pre-injury status, that is, as a factor of resistance or susceptibility to depression. The inability to manage one's home may indicate increased dependency causing reactionary depression.

This study supports the link between a higher level of depression symptoms and worse functional status. Clinicians should be attentive to depression in all levels of brain injury severity and consider treatment to help mitigate the negative functional outcome.

Limitations

These data were gathered as part of another research project. Longitudinal data collection, including time points greater than one year post-injury, would be revealing. The utilization of a symptom severity tool for depression is not equivalent to performing a diagnostic interview and does not firmly establish a diagnosis of major depression or other DSM-IV-TR66 recognized diagnoses. A reflection of the use of data collected for other purposes, administration of the BDI-II via the telephone, while having no basis in the literature, was the consistent method of administration. This is a recognized weakness and supports the need for verification of these findings. Impaired self-awareness potentially affects the ability to assess depression in the TBI population. There was no measure of impaired self-awareness in the available data. In theory, this sub-population would have been reduced as those subjects who were determined to be unable to accurately answer questions had outcome measures obtained from surrogates; therefore the BDI-II was not collected. As our data were collected for another study, information centering on the treatment of depression, pre-existing psychiatric disorders and family history of psychiatric disorders were not collected. This information would have been helpful to better understand out results.

Conclusion

This study finds the association between the FSE and the BDI-II is stronger than between the FSE and other reported depression measures. For the total population, a moderate correlation was found between FSE and BDI-II, with the worsening depressive symptoms correlating with worsening functional outcomes. The strength of the correlation between the FSE and BDI-II is stronger than the previously reported correlation between the CES-D and the FSE. The time of outcome measurement did not correlate with BDI-II scores. Predictive modeling resulted in two models. Both models contained the GCS but were divergent for the other remaining variables. Further work exploring the nuances of symptom and outcome measures needs to be performed to further understand the complex relation between depression after TBI and the functional state of the individual.

Acknowledgments

This work was supported in part by:

NIH 5 KL2 RR024983-02

NIH R01 HD48179

NIH U01 HD42652

NIDRR H133AO252604

Bibliography

1. Langlois JA, Kegler SR, Butler JA, Gotsch KE, Johnson RL, Reichard AA, Webb KW, Coronado VG, Selassie AW, Thurman DJ. Traumatic brain injury-related hospital discharges. Results from a 14-state surveillance system, 1997. MMWR Surveill Summ. 2003; 52(4):1–20. [PubMed: 12836629]
2. Jorge RE, Robinson RG, Arndt SV, Starkstein SE, Forrester AW, Geisler F. Depression following traumatic brain injury: a 1 year longitudinal study. J Affect Disord. 1993; 27(4):233–43. [PubMed: 8509524]
3. van Reekum R, Bolago I, Finlayson MAJ, Garner S, Links PS. Psychiatric disorders after traumatic brain injury. Brain Inj. 1996; 10(5):319–27. [PubMed: 8735663]
4. Gomez-Hernandez R, Max JE, Kosier T, Paradiso S, Robinson RG. Social impairment and depression after traumatic brain injury. Arch Phys Med Rehabil. 1997; 78(12):1321–6. [PubMed: 9421985]
5. Rapoport MJ, Kiss A, Feinstein A. The impact of major depression on outcome following mild to moderate traumatic brain injury in older adults. J Affect Disord. 2006; 92(2-3):273–6. [PubMed: 16504305]
6. Kim E, Lauterbach EC, Reeve A, Arciniegas DB, Coburn KL, Mendez MF, Rummans TA, Coffey EC, ANPA Committee on Research. Neuropsychiatric complications of traumatic brain injury: a critical review of the literature (a report by the ANPA Committee on Research). J Neuropsychiatry Clin Neurosci. 2007; 19(2):106–27. [PubMed: 17431056]
7. Bombardier CH, Fann JR, Temkin NR, Esselman PC, Barber J, Dikmen SS. Rates of major depressive disorder and clinical outcomes following traumatic brain injury. JAMA. 2010; 303(19): 1938–45. [PubMed: 20483970]
8. Kessler RC, Berglund P, Demler O, et al. The epidemiology of major depressive disorder. Results from the National Comorbidity Survey Replication (NCS-R). JAMA. 2003; 289(23):3095–3105. [PubMed: 12813115]
9. Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry. 2005; 62(6):593–602. [PubMed: 15939837]
10. Levin HS, Brown SA, Song JX, McCauley SR, Boake C, et al. Depression and posttraumatic stress disorder at three months after mild to moderate traumatic brain injury. J Clin Exp Neuropsychol. 2001; 23(6):754–769. [PubMed: 11910542]
11. Hibbard MR, Ashman TA, Spielman LA, Chun D, Charatz HJ, Melvin S. Relationship between depression and psychosocial functioning after traumatic brain injury. Arch Phys Med Rehabil. 2004; 85(4 Suppl 2):S43–53. [PubMed: 15083421]
12. Chamelian L, Feinstein A. The effect of major depression on subjective and objective cognitive deficits in mild to moderate traumatic brain injury. J Neuropsychiatry Clin Neurosci. 2006; 18(1): 33–8. [PubMed: 16525068]
13. Jorge RE, Robinson R, Moser D, Tateno A, Crespo-Facorro B, Arndt S. Major depression following traumatic brain injury. Arch Gen Psych. 2004; 61:42–50.
14. Rapoport MJ, McCullagh S, Shammi P, Feinstein A. Cognitive impairment associated with major depression following mild and moderate traumatic brain injury. J Neuropsych and Clin Neurosci. 2005; 17:61–65.
15. Franulic A, Carbonell CG, Pinto P, Sepulveda I. Psychosocial adjustment and employment outcome 2, 5, and 10 years after TBI. Brain Inj. 2004; 18(2):118–129.
16. Whelan-Goodinson R, Ponsford J, Schönberger M. Association between psychiatric state and outcome following traumatic brain injury. J Rehabil Med. 2008; 40(10):850–7. [PubMed: 19242623]
17. Fann JR, Katon WJ, Uomoto JM, Esselman PC. Psychiatric disorders and functional disability in outpatients with traumatic brain injuries. Am J Psychiatry. 1995; 152:1493–1499. [PubMed: 7573589]
18. Brown M, Gordon WA, Spielman L. Participation in Social and Recreational Activity in the Community by Individuals With Traumatic Brain Injury. Rehabil Psychol. 2003; 48(4):266–274.

19. Rapoport MJ, McCullagh S, Streiner D, Feinstein A. The clinical significance of major depression following mild traumatic brain injury. Psychosomatics. 2003; 44(1):31–37. [PubMed: 12515835]
20. Underhill AT, Lobello SG, Stroud TP, Terry KS, Devivo MJ, Fine PR. Depression and life satisfaction in patients with traumatic brain injury: a longitudinal study. Brain Inj. 2003; 17(11): 973–982. [PubMed: 14514448]
21. Satz P, Forney DL, Zaucha K, Asarnow R, Light R, McCleary C, et al. Depression, cognition and functional correlates of recovery outcome after traumatic brain injury. Brain Inj. 1998; 12(7):537–553. [PubMed: 9653518]
22. McCleary C, Satz P, Forney D, Light R, Zaucha K, Asarnow R, Namerow N. Depression after traumatic brain injury as a function of Glasgow Outcome Score. J Clin Exp Neuropsychol. 1998; 20(2):270–279. [PubMed: 9777481]
23. Deb S, Lyons I, Koutzoukis C, Ali I, McCarthy G. Rate of Psychiatric Illness 1 year after traumatic brain injury. Am J Psychiatry. 1999; 156(3):374–378. [PubMed: 10080551]
24. Henry JD, Crawford JR. A meta-analytic review of verbal fluency deficits in depression. J Clin Exp Neuropsychol. 2005; 27:78–101. [PubMed: 15814444]
25. Kreutzer JS, Seel R, Gourley E. The prevalence and symptom rates of depression after traumatic brain injury: a comprehensive examination. Brain Inj. 2001; 15(7):563–576. [PubMed: 11429086]
26. Busch CR, Alpern HP. Depression after mild traumatic brain injury: a review of current research. Neuropsychol Rev. 1998; 8(2):95–108. [PubMed: 9658412]
27. Godfrey HPD, Partridge FM, Knight RG, Bishara S. Course of insight disorder and emotional dysfunction following closed head injury. J Clin Exp Neuropsychol. 1993; 15:503–515. [PubMed: 8354704]
28. Fleming JM, Strong J. Self-awareness of deficits following acquired brain injury: Considerations for rehabilitation. Br J Occup Ther. 1995; 58:55–58.
29. Malec JF, Machulda MM, Moessner AM. Differing problem perceptions of staff, survivors, and significant others after brain injury. J Head Trauma Rehab. 1997; 12(3):1–13.
30. Wallace C, Bogner J. Awareness of deficits: emotional implications for persons with brain injury and their significant others. Brain Inj. 2000; 14(6):549–562. [PubMed: 10887888]
31. Malec JF, Testa JA, Rush BK, Brown AW, Moessner AM. Self-assessment of impairment, impaired self-awareness, and depression after traumatic brain injury. J Head Trauma Rehabil. 2007; 22(3):156–66. [PubMed: 17510591]
32. Ownsworth T, Fleming J, Strong J, Radel M, Chan W, Clare L. Awareness typologies, long-term emotional adjustment and psychosocial outcomes following acquired brain injury. Neuropsychol Rehabil. 2007; 17(2):129–50. [PubMed: 17454690]
33. Fordyce DJ, Roueche JR, Prigatano GP. Enhanced emotional reactions in chronic head trauma patients. J Neurol Neurosurg Psychiatry. 1983; 46:620–624. [PubMed: 6886698]
34. Lezak M, O'Brien KP. Longitudinal study of emotional, social, and physical changes after traumatic brain injury. J Learn Disabil. 1988; 21(8):456–463. [PubMed: 2972786]
35. Glenn MB, O'Neil-Pirozzi T, Goldstein R, Burke D, Jacob L. Depression amongst outpatients with traumatic brain injury. Brain Inj. 2001; 15(9):811–818. [PubMed: 11516349]
36. Weinstein CS, Fucetola R, Mollica R. Neuropsychological issues in the assessment of refugees and victims of mass violence. Neuropsychol Rev. 2001 Sep; 11(3):131–41. [PubMed: 11795840]
37. Mollica RF, Henderson DC, Tor S. Psychiatric effects of traumatic brain injury events in Cambodian survivors of mass violence. Br J Psychiatry. 2002; 181:339–47. [PubMed: 12356662]
38. Holsinger T, Steffens DC, Phillips C, Helms MJ, Havlik RJ, Breitner JC, Guralnik JM, Plassman BL. Head injury in early adulthood and the lifetime risk of depression. Arch Gen Psychiatry. 2002; 59(1):17–22. [PubMed: 11779276]
39. Dikmen SS, Bombardier CH, Machamer JE, Fann JR, Temkin NR. Natural history of depression in traumatic brain injury. Arch Phys Med Rehabil. 2004; 85:1457–1464. [PubMed: 15375816]
40. Dikmen SS, Machamer JE, Miller B, Doctor J, Temkin N. Functional Status Examination: a new instrument for assessing outcome in traumatic brain injury. J Neurotrauma. 2001; 18:127–140. [PubMed: 11229707]

41. Wilson JT, Pettigrew EL, Teasdale GM. Structured interview for the Glasgow Outcome Scale and the Extended Glasgow Outcome Scale. J Neurotrauma. 1998; 15:573–585. [PubMed: 9726257]
42. Rappaport M, Hall KM, Hopkins K, Belleza T, Cope DN. Disability rating scale for severe head trauma: coma to community. Arch Phys Med Rehabil. 1982; 63(3):118–23. [PubMed: 7073452]
43. Hudak A, Caesar R, Frol AB, Krueger K, Harper CR, Temkin NR, Dikmen SS, Carlile M, Madden C, Diaz-Arrastia R. Functional outcome scales in traumatic brain injury: a comparison of the Glasgow Outcome Scale (Extended) and the Functional Status Examination. J Neurotrauma. 2005; 22(11):1319–1326. [PubMed: 16305320]
44. World Health Organization. A Manual of Classification, Relating to the Consequences of Disease. WHO: Geneva, Switzerland; 1993. International Classification of Impairments, Disabilities, and Handicaps.
45. Chaytor N, Temkin N, Machamer J, Dikmen S. The ecological validity of neuropsychological assessment and the role of depressive symptoms in moderate to severe traumatic brain injury. J Int Neuropsychol Soc. 2007; 13(3):377–85. [PubMed: 17445285]
46. Beck, AT.; Steer, RA.; Brown, GK. Manual for the Beck Depression Inventory-II. San Antonio, TX: Psychological Corporation; 1996.
47. Teasdale G, Jennet B. Assessment of coma and impaired consciousness - a practical scale. The Lancet. 1974; 2:81–84.
48. Prasad K. The Glasgow Coma Scale: a critical appraisal of its clinimetric properties. J Clin Epid. 1996; 49(7):755–763.
49. Kirkness C, Thompson J, Ricker B, et al. The impact of aneurysmal subarachnoid hemorrhage on functional outcome. J Neurosci Nurs. 2002; 34:134–141. [PubMed: 12080869]
50. Pettigrew EL, Wilson JT, Teasdale G. Assessing disability after head injury: improved use of the Glasgow Outcome Scale. J Neurosurg. 1998; 89:939–943. [PubMed: 9833819]
51. Pettigrew EL, Wilson JT, Teasdale G. Reliability of ratings on the Glasgow Outcome Scales from in-person and telephone structure interviews. J Head Trauma Rehab. 2003; 18:252–258.
52. Whisman M, Perez JE, Ramel W. Factor Structure of the Beck Depression Inventory-Second Edition in a student sample. J Clin Psychology. 2000; 56(4):545–551.
53. Storch EA, Roberti JW, Roth DA. Factor structure, concurrent validity, and internal consistency of the Beck Depression Inventory-Second Edition in a sample of college students. Dep & Anx. 2004; 19:187–189.
54. Dozois DJA, Dobson KS, Ahnberg JL. A psychometric evaluation of the Beck Depression Inventory -II. Psychol Assess. 1998; 10(2):83–89.
55. Homaifar BY, Brenner LA, Gutierrez PM, Harwood JF, Thompson C, Filley CM, Kelly JP, Adler LE. Sensitivity and specificity of the Beck Depression Inventory-II in persons with traumatic brain injury. Arch Phys Med Rehabil. 2009; 90(4):652–6. [PubMed: 19345782]
56. Rowland SM, Lam CS, Leahy B. Use of the Beck Depression Inventory-II (BDI-II) with persons with traumatic brain injury: analysis of factorial structure. Brain Inj. 2005; 19(2):77–83. [PubMed: 15841751]
57. Hosmer, DW.; Lemeshow, S. Applied Logistic Regression. New York: John Wiley and Sons; 1989. p. 86
58. SPSS for Windows, version 17. Chicago: SPSS Inc.; 2008.
59. SAS for Windows, version 9.2 Statistical Analysis System. SAS Institute, Inc.; Cary, NC: 2008.
60. Draper K, Ponsford J, Schönberger M. Psychosocial and emotional outcomes 10 years following traumatic brain injury. J Head Trauma Rehabil. 2007 Sep-Oct; 22(5):278–87. [PubMed: 17878769]
61. Pickelsimer EE, Selassie AW, Gu JK, Langlois JA. A population-based outcomes study of persons hospitalized with traumatic brain injury: operations of the South Carolina Traumatic Brain Injury Follow-up Registry. J Head Trauma Rehabil. 2006; 21(6):491–504. [PubMed: 17122680]
62. Pagulayan KF, Hoffman JM, Temkin NR, Machamer JE, Dikmen SS. Functional limitations and depression after traumatic brain injury: examination of the temporal relationship. Arch Phys Med Rehabil. 2008; 89(10):1887–92. [PubMed: 18929017]

63. Prigatano, GP.; Summers, JD. Depression in traumatic brain injury patients. In: Roberts, MDD.; Katona, CLE., editors. Depression and Physical Illness. New York: Wiley; 1997. p. 341-358.
64. Moldover JE, Goldberg KB, Prout MF. Depression after traumatic brain injury: A review of evidence of clinical heterogeneity. Neuropsychol Review. 2004; 14(3):143–154.
65. Langlios 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. [PubMed: 16983222]
66. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR). Arlington, VA: American Psychiatric Publishing, Inc.; 2000.

Table 1

Correlation coefficients for the domains of FSE with BDI-II

Domains of FSE	N	rho	p
Social Integration	442	0.472	<0.0001
Leisure And Recreation	441	0.408	<0.0001
Home Management	442	0.393	<0.0001
Travel	443	0.373	<0.0001
Executive Function	442	0.364	<0.0001
Ambulation	443	0.353	<0.0001
Major Activity Involving Work/School	442	0.331	<0.0001
Financial Independence	441	0.319	<0.0001
Personal Care	443	0.290	<0.0001
Standard of Living	442	0.273	<0.0001

FSE = Functional Status Examination

BDI-II = Beck Depression Inventory–II

N = number of subjects

Table 2

Groups by Injury Severity

	Mild Injury GCS 13-15; N=214		Moderate-Severe Injury GCS 3-12; N=237	
Variable	**Median (IQR)**	**N (%)**	**Median (IQR)**	**N (%)**
Age (yrs) *	39 (24-55)	214	27 (20-41)	237
Male Gender		156 (73)		165 (70)
Race/ethnicity				
Caucasian		134 (63)		187 (79)
Hispanic		49 (23)		32 (14)
African American		19 (9)		16 (7)
Asian		9 (4)		2 (1)
Native American		2 (1)		0
Educational Level (yrs) *	12 (11-15)	173	12 (11-13)	158
Glasgow Coma Scale	15 (14-15)	214	3 (3-7)	237
Hospital stay duration* (d)	5 (3-10)	194	19 (11-26)	218
Time to follow up (mon)	6 (6-8)	211	6 (6-8)	233
BDI-II*	3 (0-9)	214	5 (1-12)	237
GOS-E*	7 (6-8)	213	6 (4-8)	234
FSE*	14 (10-20)	204	17 (12-28)	220

d = days

m = months

BDI-II = Beck Depression Inventory–II

GOS-E = Glasgow Outcome Scale Extended

FSE = Functional Status Examination

* = significant between group differences

Table 3

Group Characteristics by Depression Symptom Severity per the BDI-II manual

	Minimal (BDI-II ≤ 13), N=384		Mild (BDI-II 14-18), N=38		Moderate (BDI-II 19-28), N=33		Severe (BDI-II >28), N=16	
	Median (IQR)	**N (%)**	**Median (IQR)**	**N (%)**	**Median (IQR)**	**N (%)**	**Median (IQR)**	**N (%)**
Age	32 (21-49)	383	35.5 (26-46)	38	32(20-42)	33	37 (25.5-60)	16
Male gender		276 (72)		27 (71)		23 (70)		9 (56)
Race/Ethnicity		382		38		33		16
Caucasian		278 (72)		31 (82)		19 (58)		10 (63)
Hispanic		61 (16)		6 (16)		10 (30)		4 (25)
African American		30 (8)		0		2 (6)		2 (13)
Asian		10 (3)		1 (3)		2 (6)		0
Native American		2 (0.5)		0		0		0
Education	12(11-15)	278	11.5(11-14)	22	12(12-12)	18	12(12-15)	9
Injury Mechanism		377		37		31		16
Motor vehicle		218 (57)		24(63)		17 (52)		8 (50)
Violence		30 (8)		1(3)		3 (9)		0
Sports		13 (3)		0		0		0
Other		116 (30)		12 (32)		11 (33)		8 (50)
Glasgow Coma Scale	12 (3-15)	365	5.5 (3-14)	38	11.5 (3.5-15)	32	14 (6.5-15)	16
Hospital Stay Duration (d)	12 (5-22)	348	17 (8-26)	34	11 (4-22)	30	10 (7-23)	16
Time to Follow up (m)	6 (6-8)	379	6 (6-8)	38	7 (6-9)	32	6 (6-8)	15
BDI-II	2 (0-6)	384	16 (14-17)	38	22 (20-24)	33	37.5 (31-40.5)	16
GOS-E*	7 (5-8)	381	5 (3-6)	37	5 (4-6)	32	5 (3-6)	16
FSE*	14 (10-20)	363	26 (21-31)	34	25 (17-30)	31	29 (25-34)	14

d = days

m = months

BDI-II = Beck Depression Inventory-II

GOS-E = Glasgow Outcome Scale Extended

FSE = Functional Status Examination

* = significant between group differences

NIH-PA Author Manuscript
NIH-PA Author Manuscript

Table 4

Groups by Homaifar divisions of BDI-II

	Mild TBI				Moderate to Severe TBI	
Variable	**Not depressed BDI-II < 19; N=189**		**Depressed BDI-II >18; N=25**		**Not Depressed BDI-II <35; N=234**	
	Median (IQR)	**N (%)**	**Median (IQR)**	**N (%)**	**Median (IQR)**	**N (%)**
Age	39 (24-56)	189	38 (25-50)	25	27 (20-42)	234
Male Gender		138 (73)		18 (72)		164 (70)
Race/Ethnicity		188		25		234
Caucasian		122 (65)		12 (48)		186 (79)
Hispanic		41 (22)		8 (32)		30 (13)
African American		16 (8)		3 (12)		16 (7)
Asian		7 (4)		2 (8)		2 (1)
Native American		2 (1)		0		0
Education	13 (11-15)	154	12 (11-15)	19	12 (11-13)	157
Mechanism of Injury		187		24		231
Motor Vehicle		78 (41)		9 (36)		170 (73)
Violence		24 (13)		3 (12)		7 (3)
Sports		7 (4)		0		6 (3)
Other		78 (41)		12 (48)		48 (21)
GCS	15 (14-15)	189	15 (15-15)	25	3 (3-7)	234
Hosp Length of Stay (d)	5 (3-10)	170	7 (3.5-11)	24	19 (11-26)	215
Time to follow up (m)	6 (6-8)	187	7 (6-9)	25	6 (6-8)	231
BDI-II	2 (0-6)	189	25 (20-35)	25	5 (1-12)	234
GOS-E	7.5 (6-8)*	188	5 (4-6)*	25	6 (4-8)	231
FSE	13 (10-17.78)*	180	24.5 (17.89-28)*	24	16 (12-27)	217

d = days

m = months

BDI-II = Beck Depression Inventory-II

GOS-E = Glasgow Outcome Scale Extended

FSE = Functional Status Examination

* = significant between group differences

NIH-PA Author Manuscript
NIH-PA Author Manuscript

Table 5

Association of Injury Mechanisms

Variables	Cause of Injury								p-value
	Violence		Motorized Vehicle		Sports		Other		
	N	Median (IQR)	N	Median (IQR)	N	Median (IQR)	N	Median (IQR)	
Age*	34	26 (20-37.8)	267	29 (20-44)	13	23 (19-40.5)	147	45 (28-57)	<0.0001
GCS*	34	15 (13.8-15)	257	7 (3-14)	13	14 (7-15)	141	14 (6-15)	<0.0001
BDI-II*	34	3.5 (0-6.3)	267	4 (1-10)	13	0 (0-2)	147	4 (0-12)	0.0108
GOS-E	34	7.5 (5-8)	265	6 (5-8)	13	8 (6.5-8)	144	6 (5-8)	0.0573
FSE									
Total	31	13 (10-23)	251	16 (12-24)	13	13 (10-16.5)	137	16 (11-25)	0.0792
Ambulation*	31	1 (1-1)	251	1 (1-2)	13	1 (1-1)	138	1 (1-2)	0.0054
Travel*	31	1 (1-1)	251	1 (1-4)	13	1 (1-1)	138	1 (1-3)	0.0136
Social Integration	31	1 (1-1)	251	1 (1-2)	13	1 (1-1)	137	1 (1-3)	0.0882

* = statistically significant

GCS= Glasgow Coma Scale

BDI-II=Beck Depression Inventory II

GOS-E= Glasgow Outcome Scale-Extended

FSE= Functional Status ExaminationIQR = interquartile range

Table 6

Final Logistic Regression Models for prediction of depressive symptoms, using stepwise procedure

Model	Variable	Odds Ratio	95% Confidence Interval	Wald's p-value
Model 1	**Age**	.969	0.946-0.992	0.010
	FSE	1.200	1.140-1.262	0.000
	GCS	1.038	1.038-1.212	0.004
Model 2	**Education**	0.883	0.784-0.993	0.039
	GCS	1.097	1.010-1.192	0.028
	GOS-E	0.705	0.535-0.929	0.013
	FSE Home management	1.435	0.982-2.098	0.062
	FSE Social Integration	1.922	1.336-2.764	<0.0001
	FSE Standard of Living	1.543	1.049-2.269	0.028

FSE= Functional Status Examination

GCS= Glasgow Coma Scale

GOS-E= Glasgow Outcome Scale Extended