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Prevalence and predictors of affective lability after pediatric traumatic brain injury

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Abstract

Objective—Paediatric severe traumatic brain injury (TBI) is associated with significant postinjury affective and behavioral problems. Few studies have examined the prevalence and characteristics of affective lability after paediatric TBI.

Methods—97 children with severe TBI were evaluated one year postinjury for the presence of affective lability using the Children's Affective Lability Scale (CALs). Demographic, clinical, and brain lesion characteristics were also assessed.

Results—Affective lability significantly increased after injury. Eighty-six children had a preinjury CALs score of 1SD or less from the group preinjury mean ($M = 8.11$, $SD = 9.31$) of which 35 and 15 children had a 1SD and 2SD increase in their CALs score from pre- to postinjury, respectively. A variety of affective shifts manifested postinjury including anxiety, silliness, dysphoria, and irritability. The most severe symptoms were irritability and unpredictable temper outbursts. Risk factors for affective lability included elevated preinjury affective lability and psychosocial adversity as well as greater damage to the orbitofrontal cortex. Postinjury affective lability was most frequently associated with a postinjury diagnosis of attention-deficit hyperactivity disorder.

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DECLARATION OF INTEREST

The authors report no declarations of interest.

Conclusions—Affective lability is common after paediatric TBI and frequently manifests as irritability and unpredictable outbursts. Early intervention is needed to improve psychiatric outcomes.

Keywords

children; traumatic brain injury; affective lability; irritability; outbursts

INTRODUCTION

Affective lability is a psychiatric condition that occurs after severe traumatic brain injury (TBI). The condition is characterized by sudden and rapid change of emotions in which a person responds to environmental and internal stimuli in an unpredictable, hyperresponsive fashion [1]. Labile affect can manifest immediately after the injury in the form of rapid mood shifts associated with delirium [2], or months after the injury as the labile type of persistent personality change due to head trauma as conceptualized in the neuropsychiatric literature and the Diagnostic and Statistical Manual of Mental Disorders [3–7].

There is ongoing debate in the field regarding the origins of personality change after TBI. Some have argued that personality change is a direct result of the brain injury itself whereas others attribute these changes to the devastating psychosocial consequences after the injury. This issue is particularly controversial in the case of mild TBI where brain scans can appear to be normal but the individual exhibits affective and behavioral disturbances. In severe TBI, widespread and varied neurological damage is evident, but brain regions that may be contributing to emotional dyscontrol are not well localized. With the advent of neuroimaging, however, research in adults with severe TBI has shown that lesions to select frontal regions of the brain are correlated with personality change. In children, one study of severe TBI found that lesions in the frontal white matter are positively associated with personality change [6]. Psychosocial stressors after TBI are overwhelming and include lack of friendships, social isolation, dependence on caregivers, and employment difficulties, all of which can contribute to personality change. Both the neurological damage and psychosocial consequences persist long after the injury. It is possible that for some children, psychosocial stressors primarily contribute to organic personality after TBI, whereas for others it is the brain injury itself or the combination of both.

Few studies have examined the characteristics of affective lability after paediatric TBI. Max et al. [7–8] conducted a set of prospective studies examining personality change after paediatric TBI. Personality change was assessed using the Neuropsychiatric Rating Schedule (NPRS) [9], a clinician administered parent report and child interview that assessed a wide range of symptoms including affective lability, aggression, disinhibition, apathy and paranoia. Findings showed that among the 37 children with severe TBI, 40% exhibited persistent personality change (present at 2 years). Labile affect was the most common type of personality change affecting 49% of children and included irritability (41%) as well as depression (8%), anxiety (5%), and euphoria (8%) [7]. In contrast, children with mild to moderate TBI did not demonstrate any long-term personality changes. Post-resuscitation Glasgow Coma Scale (GCS) score was found to be a consistent predictor of personality

change from six months to two years after TBI [5–6], whereas psychosocial adversity and preinjury adaptive functioning were associated only with long-term personality change [6]. Lesions in the superior frontal gyrus and frontal white matter were associated with personality change at 12 months and 24 months, respectively, suggesting that damage to the dorsal frontal regions predicts both short- and long-term outcomes [5, 6, 8]. Personality change was also commonly associated with attention-deficit hyperactivity disorder (ADHD) and other externalizing disorders [5, 6, 8].

Only one other study examined neuroimaging correlates of affective lability after paediatric TBI [10]. This study evaluated 20 children with mild-to-moderate TBI and 21 children with orthopaedic injury. Affective lability was assessed using the behavioural dysregulation and emotional control domains of the Behavioral Rating Inventory of Executive Function (BRIEF). Results demonstrated that greater cortical thickness in the medial frontal lobes and right anterior cingulate gyrus was associated with increased emotion dysregulation at 18 months postinjury.

Accelerating research on affective lability in children with severe TBI is important for two reasons. First, identifying children at risk for affective lability provides opportunities for early detection and intervention, which could improve short- and long-term outcomes. Second, the construct of affective lability, specifically irritability, is a defining feature of many childhood psychiatric disorders including ADHD [11], oppositional defiant disorder (ODD) [12], bipolar disorder [13], anxiety disorders [14], and autism spectrum disorders [15]. Studying affective lability after TBI may inform mechanisms underlying this construct in typically developing children.

This study has four aims. First, we hypothesized that the prevalence of affective lability would increase one year after paediatric TBI based on prior data demonstrating an increase in psychiatric disorders after paediatric TBI [5–6, 16–18]. Second, we examined the specific types of affective states that manifested one year after severe TBI in children. Based on preliminary data in children with TBI [7], we expected that irritability would be the most common affect after injury. Third, we examined risk factors for affective lability after TBI. These included preinjury psychopathology and psychosocial adversity, two established risk factors for postinjury psychiatric disorders [5–6, 16, 18], as well as the degree of damage to the prefrontal cortex, a region implicated in emotion regulation [19–20]. Last, we cross-sectionally examined the types of psychiatric disorders associated with affective lability after injury. Based on data in typically developing and TBI children, we expected strong associations with postinjury attention-deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), mood and anxiety disorders [5–6, 8, 14, 21–22].

METHODS

Participants and Study Design

Ninety-seven children, aged 4 to 19 years, with severe TBI participated in this study. Participants were referred from tertiary care trauma centres and recruited from consecutive admissions between 1992 and 1996 to a university-affiliated neurorehabilitation unit located in a major metropolitan area. Inclusion criteria consisted of a non-penetrating head injury

with the earliest available GCS score of 8 or less. Exclusion criteria consisted of previous hospitalizations or emergency room visits for TBI, documented child abuse, premorbid intellectual disability, and central nervous system pathology. Over half of the participants were enrolled into the study less than a month postinjury (range = 4–246 days, $SD = 39.2$, Mean = 39.9, Median = 28 days). No participants were lost to follow-up.

There were two study visits. Immediately after enrollment, parents recalled data regarding their child's psychiatric status during the two weeks prior to injury. Immediately after enrollment and at one year, a board-certified senior psychiatrist (J.P.G.) performed psychiatric and psychosocial assessments. With data gathered at two time points, this study design is similar to a case-crossover design. The latter is useful for brief, rare exposures such as TBI-related injuries, which result in a precipitous change in an outcome such as affective lability. Since children served as their own controls (i.e., pre-TBI), the design minimizes confounding by controlling for characteristics of the child that are associated with increased affective lability but do not change over the short period of time (e.g., socioeconomic status). The Johns Hopkins Medicine Institution Review Board approved this study. Informed consent was obtained from the child's parent/caregiver at enrollment.

Table 1 presents the sample characteristics. Males and females were equally represented. The mean age was 10.6 years ($SD = 3.8$). The majority of injuries involved motor vehicles. The sample had low levels of preinjury psychopathology as measured by the Child Behavior Checklist (CBCL) internalizing and externalizing scales.

Psychiatric measures

Children's Affective Lability Scale (CALs)—The CALs is a 20-item scale that measures how frequently a child's affect switches from a 'normal' to a hyperresponsive affect [1]. Changes in positively dysregulated (e.g. bursts of silliness, incongruent humor) and negatively dysregulated (e.g. shift to dysphoria, unpredictable mood) affects were measured [1]. Parents rated the frequency of each affect as follows: 0 = never or rarely, 1 = occurs one to three times per month, 2 = occurs one to three times per week, 3 = occurs four to six times per week, 4 = occurs one or more times per day. The scale ranges from 0 to 80 with higher scores representing more affective lability.

The CALs is internally consistent among typically developing individuals and psychiatric samples [1, 23]. Test-retest and inter-rater reliability [1] as well as construct [24], convergent [9], and discriminative validity [1, 16, 23] have also been demonstrated. The CALs is also sensitive to changes in treatment [24].

Normative data on the CALs have been collected from 290 typically developing children ($M = 8.39$, $SD = 9.46$), 89 children receiving inpatient psychiatric care ($M = 37.58$, $SD = 19.48$), and 38 children receiving outpatient psychiatric care ($M = 29.08$, $SD = 18.39$). The CALs scores were significantly greater for the inpatient versus typically developing sample as well as the inpatient versus outpatient sample [1].

In this study, an increase in affective lability from pre- to postinjury was examined both continuously and categorically. The former examined the mean increase in CALs scores

from pre- to postinjury. The latter examined the percentage of children who had a preinjury CALS score of 1SD or below the preinjury group mean and who had a 1SD or 2SD increase in their CALS score from pre- to postinjury. The preinjury mean and SD were chosen because they approximated the data for the normative sample [1].

Changes in individual symptoms before and after injury were also analyzed by comparing the mean CALS rating for each symptom as well as the number of children with symptoms occurring 4–6 times per week or daily (i.e. CALS rating of 3 or 4). The CALS symptom data were available on 87 (90%) of the sample. There were no significant overall mean differences in pre- and postinjury CALS scores between children with and without missing item-level data (both $p > 0.2$).

The relationship between postinjury clinical affective lability and psychopathology was also analyzed. In this analysis, clinical affective lability was defined as a CALS score of 29 or above, which is consistent with the mean CALS score in an outpatient psychiatric sample [1].

Diagnostic Interview for Children and Adolescents, parent version (DICA-P)—

The DICA-P is a structured diagnostic interview with established reliability and validity [25]. This instrument assessed the presence of the common childhood DSM III-R disorders before and after injury.

Child Behavior Checklist (CBCL)—The CBCL is a widely used parent-report measure of child psychopathology with strong psychometric properties in typically developing children [26–29]. The CBCL internalizing and externalizing scales were used in this study; the clinical cutoff for these scales was a T-score > 63 .

Psychosocial measures

Hollingshead Four Factor Index of Social Status [30]—This scale was used to assess preinjury socioeconomic status (SES). Scores range from 8 to 66 with higher scores reflecting higher SES.

Modified Psychosocial Adversity Scale [31]—This measure was used to assess the degree of preinjury psychosocial adversity on a scale from 0 to 8, with 8 representing the highest adversity. The scale is based on eight risk factors related to childhood psychopathology: (1) single-parent household, (2) verbally or physically aggressive interactions between parents on a regular basis, mental health treatment for (3) mother or (4) father, substance abuse by (5) mother or (6) father, and criminal conviction of (7) mother or (8) father.

Injury measures

Glasgow Coma Scale Score (GCS)—The GCS is a widely used measure of TBI severity based on evaluation of eye opening and verbal and motor responses [32]. The earliest GCS score recorded during clinical care was used as a measure of injury severity.

Brain lesions—Magnetic resonance imaging (MRI) methods are outlined in previous studies of this cohort [33–34]. The MRI was performed three months after injury to delineate brain lesions. This time interval has been shown to correlate with postinjury neuropsychological outcomes [35]. Three sets of images were obtained on a 1.5Tesla General Electric scanner: a T1-weighted sagittal sequence, an axial spin-density/T2-weighted (double echo) sequence, with 5mm thick contiguous images; and an axial T1-weighted three dimensional volumetric SPGR (spoiled gradient recalled echo in steady state) with 1.5mm thick contiguous images. Focal injuries, such as contusions, and diffuse axonal injury lesions (DAI) were identified.

Brain lesions in five regions were delineated: orbitofrontal cortex (OFC), dorsolateral cortex, mesial cortex, temporal lobe, and ‘other brain regions’. The area called ‘other brain regions’ consisted of the non-prefrontal cortex (PFC) and non-temporal regions of the brain. The number and volume of brain lesions in each region were calculated. Lesion volumes had a skewed distribution that could not be corrected after logarithmic transformation and therefore these data were not examined in the analyses. It should be noted that the associations discovered with the number of brain lesions were identical to those found with the log volume variables (data not shown).

Data Analyses

McNemar’s test was used to examine the number of children with a preinjury affective lability score at or below 1SD who had an overall increase of 1SD or 2SD increase in affective lability postinjury. The Wilcoxon sign rank sum test was used to examine overall or average (dimensional) as well as item-level increases in CALS scores after TBI. McNemar’s test was also used to examine the number of children who had an increase in CALS ratings from 0 to 2 (coded as ‘0’ or low frequency for values ranging from 0–2) before injury to 3 or 4 (coded as ‘1’ or high frequency for values of 3 or 4) after injury for each symptom. These paired, non-parametric tests were chosen since the assumptions of independence and normality could not be held. Similarly, Glass’s η^2 was used to calculate effect sizes since this metric does not assume equal variances between groups. For all item-level analyses, a Bonferroni adjusted p -value of less than 0.0025 (.05/20 or the number of total items) was considered significant.

Linear regression analyses were performed to examine predictors of postinjury CALS scores. Only the dimensional outcome was examined in order to maximize power. Prior research informed the predictor variables, which included demographic, preinjury psychiatric, psychosocial, and brain injury variables (i.e., GCS, number of brain lesions). Predictor variables that were significant ($p < 0.05$) in the univariate models were entered into a 3-stage sequence of multivariate regression models to examine the relative contribution of each variable(s) to postinjury CALS scores. Stage 1 included demographic variables. Stage 2 added preinjury psychiatric variables, and Stage 3 included brain injury variables. The CBCL variables were each correlated with the preinjury CALS score (internalizing: $r = .55$; externalizing $r = .57$; both $p < 0.001$) and were therefore excluded from the univariate analyses due to concerns about multicollinearity. Each regression model

showed evidence of heteroscedasticity. Therefore, Huber-White standard errors were used for all models [36].

Finally, chi-square analyses were performed to assess the presence or absence of psychiatric disorders in children with and without clinical affective lability after injury. Analyses were performed using STATA (Version 12; College Station, TX).

RESULTS

Aim 1: Does affective lability increase after paediatric TBI?

There was a significant increase in CALS scores from preinjury (mean preinjury score for the entire group = 8.11, $SD = 9.31$) to postinjury ($M = 15.59$, $SD = 14.98$) ($z = 5.14$, $p < 0.001$, $d = 0.50$). There was also a significant increase in the number of children with postinjury affective lability. Eighty-six children had a preinjury score at or below 1SD from the group preinjury mean (i.e., CALS score of 17; $n = 86$; $M = 5.61$; $SD = 4.45$) of which 35 (36%) and 15 (15%) children had an increase of 1SD or 2SD CALS score between pre and post-injury, respectively.

Aim 2: What are the specific types of affective states that increased the most after TBI?

Table 2 compares item level increases in the mean CALS scores as well as the number of children who received a score of 3 or 4 (vs. 0, 1, or 2) on each symptom. There was a significant increase in the mean CALS score for 12 symptoms ($p < 0.0025$) including irritability, temper outbursts, silliness, anxiety, and dysphoria.

Aim 3: What are the predictors of affective lability as a continuous variable one year after injury?

Results of the univariate regression analyses are presented in Table 3. The following variables predicted increased postinjury affective lability: greater psychosocial adversity, higher preinjury CALS score, and fewer number of OFC lesions (all $p < 0.01$). Increased postinjury affective lability was negatively correlated with number of OFC lesions, suggesting that increased OFC lesions may negate the emergence of affective lability.

Table 4 displays the results from each multivariate model. Preinjury CALS scores contributed the most variance ($R^2 = 0.17$) followed by psychosocial adversity ($R^2 = 0.14$). The number of OFC lesions contributed the least, although it still remained highly significant ($R^2 = 0.04$; $p = 0.003$).

Aim 4: What are the most common psychiatric disorders in children with clinical affective lability after TBI?

Children with clinical affective lability ($n = 15$) had 3.7 psychiatric disorders versus 0.9 disorders in the nonclinical group ($\chi^2 = 32.59$, $p < 0.001$). Of note, the mean CALS score in the non-clinical group was 6.6 ($SD = 7.50$), which somewhat approximates the normative data in typically developing children [1]. The most common disorder in the clinical group was ADHD (94% versus 14%), which included eight new cases as well as six cases that were present before the injury and persisted. Other disorders that were more common in the

clinical group included ODD (40% vs. 8%), conduct disorder (33% vs. 6%), mania (33% vs. 2%), dysphoria (14% vs. 2%), and depression (13% vs. 1%) (all $p < 0.01$).

DISCUSSION

This is one of the first studies to examine the prevalence and characteristics of affective lability in a cohort of children with severe TBI followed for one year. The data show that children with severe TBI exhibited a significant increase in affective lability one year after injury with 15% of the sample exhibiting over a 2SD increase in symptoms. The only other prospective study of affective lability after severe TBI found a 49% prevalence of affective lability in children with severe TBI using the NPRS, a lengthy semi-structured interview of both the parent and child [7]. The interview included eight questions about 'affective instability' and queries about distress and impairment. The CALS, in contrast, is a 20-item parent report questionnaire that measures the frequency of specific types of affective shifts. The CALS and NPRS therefore vary considerably in their measurement, which may account for the discrepant findings.

The most frequent affective symptoms manifesting after injury included irritability and unpredictable temper. Lishman [37] was one of the first to report that organic brain disorders in adults are associated with irritability that can lead to 'querulous, morose behaviour and sometimes outburst of anger or hostility'. Irritability in our study appears to be similarly characterized, i.e. a continuum from mild annoyance to a feeling of edginess to rage attacks. Irritability can also be episodic or chronic, and each is associated with different longitudinal outcomes [38]. The CALS measured episodic irritability based on the nature of the questions, e.g. 'Has bursts of crabbiness or irritability', 'Suddenly loses temper', and 'Temper blow ups are unpredictable'. Other affective symptoms also increased after injury including anxiety, sadness, silliness, and incongruent humor. As such, a wide spectrum of rapidly shifting negative and positive affects emerged after severe TBI.

As expected, preinjury affective lability and psychosocial adversity were significant risk factors for postinjury affective lability and collectively contributed to about 31% of the variance. Preinjury affective lability was highly correlated with both preinjury internalizing and externalizing problems, indicating that various types of psychopathology can increase risk for postinjury affective lability. With regard to psychosocial adversity, the instrument used to measure this construct focused on mental health in the parents (e.g. aggression, substance abuse), indicating that both genetic and environmental factors contribute to outcome. Stabilizing the home environment after the child's injury is therefore a critical intervention that could potentially reduce affective lability.

The inverse correlation between OFC lesions and postinjury affective lability is consistent with prior data implicating this region in psychiatric outcomes after TBI [34]. These data, however, are contrary to adult findings showing that damage to the OFC increases affective lability and aggression [39–40], and also data implicating the OFC as a key region in top down control of emotional responses [41–42]. One explanation for our data pertains to the OFC's role in detecting the affective valence of a stimulus. Research suggests that both the OFC and the amygdala both play a role in the representation of an emotional stimulus

[43A]. Damage to the OFC may therefore compromise stimulus representation, thereby impairing encoding of its emotional characteristics (e.g. valuation, valence, intensity) and the generation of an emotional response. Another explanation is that increased OFC damage may compromise a child's ability to attend to emotional cues in the environment. Children may thereby appear indifferent in emotionally laden contexts [44]. The contribution of the OFC to affective lability was fairly (4%) small but considered unique because it cannot be explained by any of the other variables in the regression model. The findings from this study provide preliminary evidence that damage to the OFC contributes to reduced emotional processing. Further investigation is needed to establish the clinical significance of this association.

As expected, affective lability was associated with multiple psychiatric disorders, most commonly ADHD but also ODD, conduct disorders, and mood disorders. The occurrence of ADHD after TBI, which is termed secondary ADHD, is a well-established phenomenon that has distinct deficits from those of primary ADHD [16, 22, 45]. Similar to findings in primary ADHD, the co-occurrence between secondary ADHD and affective lability, may stem from deficits in self-regulation and inhibitory control, which are common after severe TBI. Further research is needed to better understand the degree to which underlying neuropsychological deficits in children with TBI are reflected in the diagnosis of ADHD and the dimensional entity of affective lability.

In the child psychiatry literature, the term affective lability is often used interchangeably with terms such as affective instability, mood lability, and emotional dysregulation [11]. These terms tend to refer to the presence of negative (e.g. irritability and tantrums) rather than positive affects [11]. In a recent review, Renaud and Zacchia [46] indicate that affective lability is a general term that refers to the rapid shifting of emotions, and that the 'dimensions concerning valence, intensity, duration, and speed of shifting should be considered relevant to the characterization and definition'. A granular analysis of the types of affective shifts that occur in different childhood psychiatric disorders is therefore needed. Such findings may inform phenotypes for pathophysiological research as well as customized treatment approaches.

This study had several limitations. First, the study lacked a non-TBI control group, which precludes firm conclusions about the impact of TBI on outcome. The study does, however, use a case-crossover design, which is superior to case-control designs since it minimizes between-group confounding. Second, the CALS was administered only to the parents. In future studies, it will be important to collect multi-informant data and examine cross-informant correlations of affective lability. Third, the data reflect outcomes in children who were referred to a rehabilitation centre and not all children with severe TBI. Fourth, some children were enrolled several months after injury, which may have resulted in recall bias regarding the child's preinjury psychiatric status. Post-hoc analyses, however, revealed no significant association between time lag and preinjury affective lability or any of the other variables, e.g. postinjury affective lability, psychosocial adversity, or orbital lesions. Another limitation is that the emergency room GCS scores were not always included in the referral materials from the acute care admission. As such, for some subjects, the GCS from the field was recorded.

Finally, this study was based on an older dataset. The researchers thought that the data were important to analyze because of the significance of the topic, large and well-characterized sample, rigorous study design, and shortage of data in this area. Two factors, however, must be acknowledged in regards to the dataset. First, the study assessed psychopathology using DSM IIR criteria. The changes from the DSM IIR to DSM 5 for the disorders of interest including ADHD, ODD, and mood disorders, however, were minimal although the criteria for some DSM 5 disorders appear less stringent. For example, the diagnostic criteria for ADHD were expanded in the DSM 5 to include inattentive and hyperactive-impulsive subtypes; however the individual diagnostic criteria were quite similar. The DSM IIR ADHD criteria may actually be more restrictive as the disorder must manifest before age 7 whereas the DSM 5 criteria require onset before age 12. The diagnostic criteria of ODD in DSM IIR requires the presence of 5/9 symptoms whereas DSM 5 requires 4/8 symptoms; these symptoms overlap exactly with the exception of “often swears or uses obscene language,” which was removed from the DSM IIR criteria [4, 47]. If DSM 5 criteria were used, the psychopathology prevalence data in this study may actually be higher than reported, which may affect the relationship between affective lability and specific disorders.

Another point to consider is the possibility of cohort effects. Recent data, however, indicate considerable variability in TBI outcomes across the country [48]. As such, the prevalence of affective lability may decrease with improved interventions but could also increase due to enhanced survival rates. The relationships, however, between predictor variables and affective lability would not be expected to change over time.

In summary, severe TBI in children can result in significant affective lability, particularly irritability and outbursts. Risk factors for postinjury affective lability include the presence of preinjury affective lability, the degree of psychosocial adversity, and the extent of OFC damage. Future research on the prevention and treatment of this condition will be critical to improving short- and long-term outcomes.

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Table 1

Sample characteristics of the TBI cohort (n=94).

	Mean (SD) or n (%)	Range in cohort
Demographic variables		
Age at injury (years)	10.5 (3.8)	4–19
Males	56 (58%)	
African American	53 (55%)	
Caucasian	40 (41%)	
Other race	4 (4%)	
Mechanism of injury		
Pedestrian	50 (58%)	
Passenger/driver	27 (31%)	
Bicyclist	9 (10%)	
Other	10 (11%)	
Injury severity variables		
Initial GCS score	5.4 (1.8)	3–8
Preinjury psychosocial variables		
Socioeconomic status	34.5 (13)	8–66
Psychosocial adversity	1.4 (1.4)	0–5
Preinjury psychopathology		
Preinjury affective lability	8.11 (9.31)	0–49
Preinjury CBCL Internalizing Scale	49.59 (9.52)	31–73
Preinjury CBCL Externalizing Scale	52.06 (11.64)	30–76
Number of brain lesions		
Orbital	0 (59%), 1–2 (31%), 3+ (10%)	0–6
Dorsal	0 (46%), 1–2 (40%), 3+ (14%)	0–7
Mesial	0 (41%), 1–2 (38%), 3+ (21%)	0–10
Temporal	0 (31%), 1–2 (42%), 3+ (26%)	0–15
Other brain regions ^a	0 (17%), 1–2 (16%), 3+ (67%)	0–15

^a Other brain regions refers to non-prefrontal cortex and non-temporal lesions

Table 2

Comparison of affective lability symptoms^a before and one year after severe TBI (n = 85).

CALS Symptoms	Mean Symptom Scores ^b			Number of Children with Symptoms occurring 4–6 times per week or daily		
	Preinjury	Postinjury	Change	Preinjury	Postinjury	Change
Suddenly cries	0.16	0.57	0.41	1	9	8
Antecedents of temper blow ups unpredictable	0.37	0.96	0.60*	2	12	10
Suddenly anxious	0.36	0.85	0.49*	3	12	9
Overly affectionate	0.66	0.62	−0.04	7	6	−1
Loss of interest	0.62	1.11	0.49*	4	15	11*
Unpredictable mood	0.44	1.01	0.57*	3	15	12*
Suddenly loses temper	0.39	0.91	0.52*	1	15	14*
Increased talking	0.70	1.18	0.48*	7	15	8
Shaky, heart pounds	0.04	0.14	0.09	0	2	2
Unpredictable crying	0.15	0.46	0.31	0	4	4
Bursts of silliness	0.68	1.04	0.37*	5	11	6
Task impersistence	0.55	1.14	0.58*	4	14	10
Temper blow ups unpredictable	0.29	0.90	0.61*	1	14	13*
Perseverative speech	0.48	1.03	0.55*	3	12	9
Incongruent humor	0.44	0.83	0.39	5	10	5
Shift to dysphoria	0.21	0.57	0.37*	0	5	5
Bursts of nervousness	0.25	0.61	0.36*	4	6	2
Bursts of irritability	0.44	0.83	0.40	2	13	9*
Overly familiar	0.16	0.38	0.22	1	3	2
Angry reaction	0.33	0.65	0.32	2	9	7

* $p < 0.0025$

^a Affective lability was measured using the Children's Affective Lability Scale (CALS, Gerson et al., 1996)

^b Mean symptom scores refers to the mean CALS score before and after injury

Table 3

Univariate linear regression analyses predicting affective lability after brain injury (n=97).

Predictor Variables	β^a	95% CI
Age at injury	-0.53	(-1.33, 0.26)
Male gender	3.25	(-2.60 – 9.11)
Socioeconomic status	-0.13	(-0.36, 0.09)
Preinjury CALS score ^b	0.81 **	(0.51, 1.10)
Psychosocial adversity	3.81 **	(1.44, 6.18)
Initial GCS score	1.01	(-0.76, 2.79)
Number of lesions		
Orbitofrontal	-2.50 **	(-3.92, -1.09)
Dorsolateral	0.90	(-1.15, 2.94)
Mesial	-0.36	(-1.73, 1.02)
Temporal	0.56	(-0.82, 1.94)
Other brain regions ^c	0.00	(-0.70, -0.70)
Log of whole brain volume	-0.74	(-21.04, 19.56)

**
 $p < 0.01$

^a Unstandardized Beta Coefficients are presented

^b CALS = Children's Affective Lability Scale

^c Other brain regions refers to non-prefrontal cortex and non-temporal lesions

Table 4

Multivariate regression models predicting affective lability after severe TBI (n=97).

Predictor Variables	Model 1 β (95% CI)	Model 2 β (95% CI)	Model 3 β (95% CI)
Psychosocial adversity	3.81* (1.43 – 6.18)	2.50* (0.39 – 4.62)	2.40* (0.14 – 4.65)
Preinjury CALS score	–	0.68** (0.39 – 0.97)	0.67** (0.26 – 1.08)
Number of OFC lesions	–	–	–1.87** (–3.11 – –0.63)
Adjusted R ²	0.14	0.31	0.36

*
 $p < 0.05$,**
 $p < 0.01$