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Subtle Motor Signs in Children With Chronic Traumatic Brain Injury

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Abstract

Objective: The aim of the study was to characterize subtle motor signs in children with moderate-severe traumatic brain injury in the chronic phase of injury.

Design: Fourteen children with moderate ($n = 6$) or severe ($n = 8$) traumatic brain injury, ages 11–18 yrs, who had sustained their injury at least 1-yr before study participation (range 1–14 yrs since injury), and 14 matched typically developing controls were examined using the Physical and Neurological Examination of Subtle Signs (PANESS). To examine the neural correlates of subtle motor signs, measures of total cerebral volume and motor/premotor volume were derived from magnetic resonance imaging.

Results: Children with traumatic brain injury had significantly poorer PANESS performance than controls on the total timed subscore, proximal overflow, and the PANESS total score. Participants with severe traumatic brain injury had greater proximal overflow than those with moderate injury, after controlling for age at injury. Across all participants, greater proximal overflow correlated with reduced total cerebral volume, whereas within the traumatic brain injury group, reduced motor/premotor volume correlated with lower PANESS total score.

Conclusions: The study highlights the importance of examining subtle motor signs including overflow during clinical evaluation of chronic pediatric traumatic brain injury and establishes the clinical utility of the PANESS as a measure sensitive to chronic subtle motor signs in this population.

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Keywords

Traumatic Brain Injury; Pediatric; Subtle Motor Signs; PANESS

Traumatic brain injury (TBI) is a major cause of long-term disability worldwide. The public health burden is disproportionately high in children, especially adolescents, because of the higher incidence of TBI in young people compared with adults.¹ Pediatric TBI can lead to a wide range of impairments with varying severity in the cognitive, motor, behavioral, and developmental aspects of functioning, resulting in an inability or reduced ability to perform daily activities.² Moreover, due to injury to a maturing brain, consequences of brain injury during development may or may not become evident immediately after the injury. The chronic effects of pediatric brain injury may be dependent on the extent of ongoing brain maturation, such that some symptoms may appear as the developmental expectations change as the child ages.³ Chapman referred to this as a “neurocognitive stall” manifested by the presence of halting or slowing in later stages of cognition, social, and motor development beyond the initial recovery after brain injury.⁴ Thus, research examining the consequences of pediatric TBI must not only focus on the acute injury phase but should also examine the chronic sequelae.

Several studies have reported chronic motor deficits after moderate-severe pediatric TBI in speeded motor tasks, balance, fine motor control, and coordination.^{5,6} Caeyenberghs et al.⁵ reported chronic deficits in manual dexterity, balance, and overall gross and fine motor skills in children with moderate-severe TBI assessed 4 yrs after injury. Deficits in processing speed on tasks requiring a motor response were found in children with severe TBI 10 yrs after injury.⁷ In addition, recovery trajectories differ based on the severity of brain injury, with a higher risk of chronic motor deficits after severe TBI in early childhood.⁸ However, there is a lack of research examining developmentally relevant subtle motor signs (such as overflow and dysrhythmia) in moderate-severe chronic pediatric TBI.

Developmental changes in motor control include improvements in speed as well as reduced subtle signs, such as overflow. Subtle signs are defined as abnormal motor or sensory abnormalities that are not readily localizable to specific brain regions.⁹ Motor overflow is defined as co-movement of body parts not specifically needed to efficiently perform a task.¹⁰ Motor overflow is age-appropriate in children younger than 10 yrs and persistence of overflow into later childhood and adolescence is considered a marker of developmental lag.¹⁰ The Physical and Neurological Examination of Subtle Signs (PANESS),¹¹ a reliable measure of balance, fine motor control, processing speed, and subtle signs such as motor overflow has been shown to effectively capture the maturation of motor function¹⁰ and the atypical persistence of subtle motor deficits in children with milder forms of TBI¹² and developmental disabilities such as attention-deficit hyperactivity disorder (ADHD).¹³ The presence of subtle motor signs in ADHD is of particular relevance to TBI due to the presence of comorbid ADHD (both developmental and secondary) in TBI and similarities across cognitive symptoms.¹⁴

In a preliminary report examining longitudinal changes in subtle motor performance 2 and 12 mos after injury, children with mild-moderate TBI (but without overt motor impairment)

showed improvements in subtle motor function for the first year after injury although deficits in balance remained at 12 mos after injury.¹² Persistent subtle motor deficits on the PANESS have also been observed in children with sports-related concussion.¹⁵ Despite deficits in global subtle motor function, motor overflow was not found to be higher in children with milder TBI as compared with neurotypical controls.¹² Anecdotally, overflow is more commonly observed in clinical evaluation of children with moderate-severe TBI. In addition, atypical motor overflow is reported in children with ADHD.^{13,14} Cole et al.¹³ found that boys with ADHD did not show typical age-related improvements in overflow and found a significant association between overflow and difficulties with motor inhibition. Thus, a detailed examination of subtle motor deficits in chronic pediatric TBI might add insight into the developmental maturation of the motor system after TBI, while also potentially providing a more sensitive bedside measure to examine broader effects of injury.

Understanding the relationship between subtle motor deficits and imaging findings in pediatric TBI would be useful for understanding the neural basis of deficits in the chronic stage of injury and may assist with improving prognostication earlier after injury. Motor deficits after TBI have been associated with both focal and global imaging findings.^{5,6} In chronic pediatric TBI, lower manual dexterity and balance scores were associated with lower fractional anisotropy of the cerebellum and the corticospinal tracts as measured using diffusion tensor imaging.⁵ Compared with controls, greater motor overflow during finger tapping in children with ADHD has been attributed to reduced activation of the contralateral primary motor cortex, potentially representing insufficient recruitment of resources necessary to mobilize transcallosal interhemispheric inhibition.¹⁶ In contrast, by definition, subtle signs are abnormal motor or sensory abnormalities that are not readily attributable to focal lesions.⁹ Thus, it is unclear whether subtle motor signs after TBI are associated with focal injury to motor regions versus more global injury.

This study aimed to characterize subtle motor signs, with an emphasis on motor overflow, in children with moderate-severe TBI in the chronic phase of injury, at least 1 yr after TBI. In addition, to better understand the neuroanatomical correlates of persistent motor deficits, this study examined the relationship between subtle signs and measures of focal (motor/premotor cortex volume) and global (total cerebral volume) injury. The research questions include the following:

1. Do children with moderate-severe TBI show long-term subtle motor signs as compared with neurotypical controls?
2. Are there particular subtle motor deficits that distinguish children with moderate TBI from those with severe TBI?
3. Are subtle motor signs associated with focal or global imaging findings?

Compared with controls, children with TBI were expected to have more subtle motor impairments on gross and fine motor tasks, including more overflow during tasks. Children with severe TBI were expected to have more motor impairments than those with moderate TBI. The authors hypothesized that reduced total cerebral and motor/premotor cortex volumes in children with TBI would correlate with greater subtle motor dysfunction.

METHODS

Participants included 14 children and adolescents with moderate or severe TBI, ages 11–18 yrs (M [SD] = 15.7 [2.15] yrs; 8 males) who had sustained their injury at least 1 yr before study participation. The Johns Hopkins Medicine Institutional Research Board approved this study. Written informed consent and assent were obtained from a parent/legal guardian and child participants, respectively. Participants with TBI were recruited from outpatient pediatric brain injury rehabilitation clinics. Study inclusion criteria were TBI characterized by at least one of the following: Emergency Department Glasgow Coma Scale Score (GCS) of 12 or lower, posttraumatic amnesia lasting at least 24 hrs, loss of consciousness lasting more than 30 mins, or presence of injury-related intracranial findings on clinical imaging. Exclusion criteria were penetrating TBI; presence of hardware interfering with imaging; inability to complete paper-pencil tasks, push buttons, or lie in the MRI scanner for up to an hour due to cognitive or motor impairment; and pregnancy. Severe TBI was defined as hospital Glasgow Coma Scale Score of 8 or lower, loss of consciousness of more than 6 hrs, or posttraumatic amnesia of more than 1 wk; all other injuries were categorized as moderate.^{17,18} Of the 14 children with TBI, six had moderate TBI while eight had severe TBI. Data from four children with moderate TBI have been reported previously in a longitudinal study of subtle motor function for the first year after TBI.¹²

Fourteen age- and sex-matched typically developing controls, ages 11–18 yrs (M [SD] = 16.01 [1.58] yrs) were included. The neurotypical participants were recruited using flyers, word of mouth, and radio advertisements. None of the participants in the control group met criteria for educational or behavioral diagnosis on a structured interview, Diagnostic Interview for Children and Adolescents¹⁹; the same was true for the children with TBI with regard to their preinjury history. All participants with TBI had independent ambulation with no to minimal impairment in ambulation for community distances, as measured by the physical health summary score of the Pediatric Quality of Life Parent-Report Assessment.¹⁷ One participant with TBI reported the use of a Baclofen pump. No participants were taking enteral medications for tone. Analyses were conducted with and without this participant to ensure that this participant did not drive the results. All participants had an IQ score of 80 or higher as measured by the two-scale Weschler's abbreviated scale of intelligence.¹² This study conforms to all STROBE guidelines and reports the required information accordingly (see Supplemental Checklist, Supplemental Digital Content 1, <http://links.lww.com/PHM/A712>).

Measures

The revised PANESS is an assessment tool that examines subtle signs of motor impairment during gait, balance, and timed basic motor functions in children.¹¹ The PANESS has two primary subscores (gaits and stations and total timed) that are summed to determine the PANESS total score. See Table 1. Stressed gait tasks include heel walking, toe walking, walking on the sides of the feet, and tandem gait. The examiner scores the number of errors in a sample of 10 steps. In balance tasks, children stand and hop on one foot. The number of seconds standing (maximum of 30 for each foot) and hopping (maximum of 50) was recorded.

The total timed score includes the time to complete 20 movements of six timed repetitive tasks, namely foot tapping, heel-toe tapping, hand patting, hand pronation/supination, finger tapping, and finger apposition (tapping the thumb to each of the four other fingers on each hand in a fixed sequence). Motor overflow (proximal, mirror, and orofacial) was assessed during these six timed motor tasks. Proximal overflow indicates extraneous movement on the same side involving larger muscle groups, even if in a different limb (e.g., lifting at elbow rather than wrist during hand patting). Mirror overflow refers to extraneous movement on the same limb on the opposite side, whereas orofacial overflow denotes any extraneous orofacial movement during the tasks. For the current study, the variables of interest were proximal and mirror overflow.

The PANESS has been shown to have good test-retest reliability in both younger and older children.¹⁵ All study participants were able to perform all the tasks of the PANESS. The variables examined for this study included the total PANESS score, gaits and stations and total timed subscores, and motor overflow (proximal and mirror) scores.

Imaging Protocol

All participants participated in a mock scan session to decrease anxiety and were trained to lay still. No sedation was used. Magnetic resonance images were acquired on a 3.0 T Philips “Achieva” MRI scanner (Best, the Netherlands). Magnetization prepared rapid gradient recalled echo images (slice thickness = 1.0 mm; field of view = 26 cm; matrix size = 256 × 256) were used for volumetric assessment and analysis of macroscopic features of the cortex. Only images with minimal motion (<3 mm of head displacement between frames) were used for FreeSurfer processing. FreeSurfer was used to obtain total cerebral volume (TCV) measurements and parcellation using the Ranta atlas¹⁸ was performed to obtain grey matter volume of the primary motor cortex, lateral premotor cortex, and supplementary motor complex. The volumes from these three cortical regions from each hemisphere were combined to yield one measurement called “motor/premotor cortex volume.” FreeSurfer parcellation quality was visually inspected for each subject. None of the TBI participants had focal findings in the study regions of interest areas on their acute scans. Scans were not obtained for one TBI participant because of scheduling conflicts.

Statistical Analyses

For PANESS (total score, timed total, gaits and stations, proximal and mirror overflow) and brain measures, independent samples *t* tests were used for group comparisons and between children with moderate versus severe TBI. Effect sizes are reported using Cohen’s *d*. Pearson’s correlation was used to examine whether age or age at injury correlated with any of the dependent variables, and they were used as a covariate in brain-behavior correlations or in an analysis of covariance examining group differences if a significant relationship was found. For *t* tests for the five PANESS scores, the α value was set at .01 (.05/5) based on Bonferroni correction to account for multiple comparisons. α was set at .05 for all other tests. All statistical analyses were performed using the Statistical Package for Social Sciences (SPSS), Version 24.0.

RESULTS

Participant Characteristics

The demographic and clinical characteristics of the TBI group are shown in Table 2. The mean (SD) time since injury for participants with TBI was 3.6 (4.2) yrs (range, 1–14.5 yrs). Of the three children with moderate TBI and normal study MRI, one had acute imaging findings (small subdural hemorrhage). There was no group difference on age ($t_{(26)} = -.44$, $P = 0.66$). Within the TBI group, there was no age difference between those with moderate versus severe injury ($t_{(12)} = .48$, $P = 0.64$). Analyses with and without the participant with the Baclofen pump were comparable, and hence, the analyses presented hereinafter include this participant.

Across all participants, Pearson's correlation revealed no significant association of age and total PANESS scores ($r_{(28)} = .002$, $P = 0.99$), total timed ($r_{(28)} = .05$, $P = 0.82$), gaits and stations ($r_{(28)} = -.07$, $P = 0.73$), proximal overflow ($r_{(28)} = -.23$, $P = 0.23$), or mirror overflow ($r_{(28)} = -.37$, $P = 0.051$) or brain volume measures of TCV ($r_{(27)} = .29$, $P = 0.14$) or motor/premotor cortex volume ($r_{(27)} = .07$, $P = 0.72$). Within the TBI group, age at injury negatively correlated with proximal overflow, ($r_{(14)} = -.54$, $P = 0.047$), such that injury at a younger age was associated with greater overflow. Age at injury did not correlate with PANESS total score ($r_{(14)} = -.26$, $P = 0.37$), total timed ($r_{(14)} = -.23$, $P = 0.43$), gaits and stations subscores ($r_{(14)} = -.17$, $P = 0.56$), or mirror overflow ($r_{(14)} = -.21$, $P = 0.48$), or with brain volume measures of TCV ($r_{(13)} = .55$, $P = 0.05$) or motor/premotor cortex volume ($r_{(13)} = .03$, $P = 0.93$).

Subtle Motor Performance

Participants with TBI showed significantly more subtle motor deficits than controls on PANESS total score ($t_{(26)} = 3.9$, $P = 0.001$, $d = 1.48$) and total timed subscore ($t_{(26)} = 3.85$, $P = 0.001$, $d = 1.45$). The group difference on the gaits and stations score did not meet the alpha criterion based on correction for multiple comparisons ($t_{(26)} = 2.26$, $P = 0.032$, $d = .86$). On overflow measures, the TBI group showed significantly more proximal overflow compared with the controls ($t_{(26)} = 3.12$, $P = 0.005$, $d = 1.2$). No significant group differences were observed on mirror overflow ($t_{(26)} = .16$, $P = 0.88$, $d = .06$).

Within the TBI group, participants with severe TBI did not differ from those with moderate TBI on the PANESS total score ($t_{(12)} = -1.02$, $P = 0.33$, $d = .59$) or subtotals of total timed ($t_{(12)} = -.55$, $P = 0.60$, $d = .31$) and gaits and stations ($t_{(12)} = -1.06$, $P = 0.31$, $d = .61$). On measures of overflow, participants with severe TBI showed significantly more proximal overflow than those with moderate TBI after controlling for age at injury ($F_{(11)} = 12.72$, $P = 0.004$). No difference was observed between severity levels on mirror overflow ($t_{(12)} = .30$, $P = 0.77$, $d = .17$). See Table 3.

Brain Volume Differences in Participants With TBI

As expected, participants with TBI showed significantly reduced TCV ($t_{(25)} = -2.88$, $P = 0.008$, $d = 1.12$). There was a trend toward significant group difference on motor/premotor cortex volume ($t_{(25)} = -1.8$, $P = 0.084$, $d = .69$). Within the TBI group, there were no

significant differences between the participants with moderate TBI and severe TBI on TCV ($t_{(11)} = .48$, $P = 0.64$, $d = .27$) and motor/premotor cortex volume ($t_{(11)} = -.45$, $P = 0.66$, $d = .25$). See Table 3.

Neural Correlates of Subtle Motor Performance

Across all participants, TCV significantly correlated with PANESS total timed ($r_{(27)} = -.39$, $P = 0.047$) and proximal overflow scores ($r_{(27)} = -.39$, $P = 0.046$) such that lower TCV was associated with greater subtle motor signs. Within the TBI group, although not significant, correlations between TCV and PANESS total ($\rho_{(13)} = -.35$, $P = 0.24$), total timed ($\rho_{(13)} = -.35$, $P = 0.24$), and proximal ($\rho_{(13)} = .006$, $P = 0.99$, after accounting for age at injury) indicated small/moderate effect sizes. On the other hand, for the control group, correlations between TCV and PANESS total ($\rho_{(14)} = -.11$, $P = 0.71$), total timed ($\rho_{(14)} = -.08$, $P = 0.80$), and proximal ($\rho_{(14)} = .03$, $P = 0.9$) indicated small-effect sizes.

Within the TBI group, motor/premotor cortex volume significantly correlated with PANESS total ($\rho_{(13)} = -.64$, $P = 0.018$, see Fig. 1) and the correlation with total timed trended toward significant ($\rho_{(13)} = -.49$, $P = 0.09$). Motor/premotor cortex volume did not correlate with proximal overflow ($\rho_{(13)} = .17$, $P = 0.60$, after accounting for age at injury). On the other hand, for the control group, there were no significant correlations between motor/premotor cortex volume and PANESS total ($\rho_{(14)} = -.20$, $P = 0.49$), total timed ($\rho_{(14)} = -.35$, $P = 0.22$), and proximal ($\rho_{(14)} = .17$, $P = 0.56$).

DISCUSSION

This study examined subtle motor signs, including motor overflow, as measured using the PANESS in children with moderate-severe TBI compared with age- and sex-matched typically developing controls. Consistent with existing literature,⁵ compared with controls, children with TBI showed more subtle motor deficits, namely, reduced balance as measured by the gaits and stations subscore, reduced motor speed as measured by the timed total score, along with greater proximal overflow. Chronic deficits in balance and processing speed after TBI have been previously reported, with processing delays noted in children with severe TBI up to 10 yrs after injury.⁷ As would be expected, compared with previous work in children with mild-moderate TBI 1 yr after injury,¹² the current cohort showed more extensive differences on the PANESS compared with controls, demonstrating that chronic subtle motor impairments are more extensive after more severe TBI in children. However, in this cohort, the broadest PANESS scores did not differentiate between children with moderate and severe TBI. Similar results were found in a study examining longitudinal recovery after TBI, where although children with severe TBI performed significantly worse than those with moderate TBI immediately after TBI, there was no difference on speeded motor response and strength in children with moderate compared with severe TBI at 1 and 3 yrs after TBI.⁸ Although motor recovery trajectories for moderate and severe TBIs are different for the first-year postinjury,⁸ this suggests that subtle motor deficits in the chronic phase of recovery may be similar for those with moderate and severe injuries, at least when considering those children with good enough recovery to participate in a study of higher level motor function.

Although motor overflow has not previously been closely examined in pediatric TBI, in children with ADHD, the relationship between motor overflow and the maturation of cortical motor systems is well established. For instance, greater motor overflow as measured by the PANESS was associated with reduced activation in contralateral primary motor cortex, bilateral premotor cortex, and supplementary motor cortex. This reduced activation reflects decreased recruitment of neural circuitry involved in active inhibition of homologous motor circuitry unnecessary for task execution.²⁰ According to the transcallosal facilitation hypothesis, activation of a cortical region during voluntary movement will facilitate activation of the same area in the opposite hemisphere, via interhemispheric connections.^{21,22} Such facilitation will lead to motor overflow unless it is inhibited by the contralateral hemisphere via the interhemispheric transfer of inhibition. This reduced inhibition by the contralateral motor areas seems to be the cause of motor overflow in children with ADHD.^{21,22} Across populations of typically developing children and those with ADHD, study of overflow has traditionally emphasized mirror movements, which may result from bilateral cortical activity secondary to a lack of interhemispheric inhibition mediated by transcallosal fiber tracts.²¹ In typically developing children, the developmental trend of reduced overflow with increasing age, with negligible overflow beyond age 10 yrs, is attributed to increased myelination of white matter tracts.²¹ In this study sample, seven children (46.6%) in the control group presented with some mirror overflow, comparable with eight children in the TBI group presenting with mirror overflow. In addition, there was a significant relationship between age and mirror overflow, such that mirror overflow reduced with increasing age. Future research in an older age group is required to examine the sensitivity of this marker to sequelae of TBI.

An important finding of this study is the presence of greater proximal (but not mirror) motor overflow in the TBI group compared with controls. Developmentally, proximal overflow is considered to resolve earlier than mirror overflow.²³ Proximal overflow is thought to occur because of immaturity of the cortical systems necessary for automatically (unconsciously, without explicit effort) inhibiting the spread of excitatory activity to the adjacent motor cortex.²⁴ In this cohort, the association between proximal overflow and injury severity suggests either disruption of maturation of these cortical systems (in younger children) or the disruption of previously matured systems (in older children). In addition, younger age at injury was associated with greater proximal overflow, suggesting a disruption of maturation resulting in more chronic deficits compared with the disruption of acquired skills. However, the relationship between age at injury and persistence of overflow beyond childhood needs to be further examined in future studies.

In a previous study, children with mild-moderate TBI showed more motor overflow (combined across proximal, mirror, and orofacial) than controls at 2 mos after injury; however, this difference did not persist at 12 mos after injury. In the current study, proximal overflow was identified in children with severe but not moderate TBI. Given that other PANESS scores did not differentiate between the children with moderate versus severe TBI, this pattern suggests that persistence of proximal overflow may be particularly sensitive to the severity of TBI. More quantitative measures of mirror overflow, using finger electrogoniometers, may also prove useful, as they have in children with ADHD.¹⁸

Children with TBI had reduced TCV, which has been implicated as a measure of cortical atrophy after TBI. Generalized cerebral atrophy after moderate-severe TBI has been associated with long-term functional status and injury severity.²⁵ In the current study, across all participants, presence of subtle motor signs including overflow correlated with reduced TCV. Specifically, this relationship was driven by the TBI group, indicating that persistent subtle motor signs are associated with brain pathology. Because age at injury correlated with proximal overflow and nearly correlated with TCV, brain development at the time of TBI may contribute to this relationship. Consistent with literature,²⁶ the PANESS total score significantly correlated with motor/premotor cortex volume. In contrast, proximal overflow correlated with TCV but not motor/premotor volume. This is consistent with the definition of subtle signs, which are considered to not be attributable to focal brain lesions. Uttner et al.²⁷ studied patients with pathologic motor overflow with a distributed range of lesion sites and found that overflow could not be localized to one specific region. Rather, Uttner et al.²⁷ suggested that overflow may at least partially be attributed to an impaired ability to inhibit additional cortical activation through attentional control mechanisms. However, in children with ADHD, subtle motor deficits including overflow have been associated with white matter abnormalities in circuits originating in the motor (supplementary motor area) and premotor areas.²⁰ This observation suggests that the neural correlates of subtle motor deficits may differ in pediatric clinical populations and that the impact of deficits beyond pure motor systems need to be considered. In addition, because of the heterogeneity within the TBI group, the cortical origins of overflow may differ for each child based on injury to the motor versus attention networks. None of the participants with TBI in the current study had a history of ADHD; however, because ADHD is a common comorbidity in children with TBI, further research is needed to better understand the significance of these subtle movements in children with a history of ADHD.

Developmentally, the persistence of subtle motor signs such as overflow in chronic pediatric TBI could imply a potentially altered developmental trajectory of global white matter myelination due to diffuse cerebral injury. The systems supporting motor and cognitive control have a protracted period of development and are vulnerable to disruption,²⁸ causing long-lasting motor and cognitive impairments after pediatric TBI. Longitudinal research is required to examine whether the phenomenon of a “neurocognitive stall” in the chronic phase of injury may be applicable to motor functioning as well.

The small sample size of the current study limits the generalizability of the study findings. In addition, the effect of sex should be examined in future studies with larger cohorts. Another limitation of this study is the heterogeneity in age at injury, with two participants being injured at a very young age. However, although the PANESS data from these two participants were not outliers, they may present differently than those injured at a later age. Future research is required to examine the relationship between age at injury and subtle motor function. In the current sample, age at injury was highly correlated with time since injury, and hence, the effect of time since injury could not be separately investigated. Although volumetric measures used in the current study provide preliminary support for the neural correlates of subtle motor deficits in chronic pediatric TBI, research using connectivity measures such as diffusion tensor imaging is required to identify specific white matter tracts affected in TBI and their relationship to chronic motor function and subtle signs

such as overflow. In addition, research examining the relationship between cerebellar volume and subtle motor function is required.

The current study highlights the importance of neurological examination of subtle motor signs during clinical evaluation of children with chronic moderate-severe TBI. The study results establish the clinical utility of the PANESS as a measure sensitive to chronic subtle motor signs in children with moderate-severe TBI. Motor examinations, such as the PANESS, which highlight both speed and subtle signs, may be sensitive to impaired neural maturation, even in children with TBI without overt motor deficits. The presence of subtle signs such as proximal overflow may be an indicator of injury severity and long-term motor outcomes. As the persistence of overflow (combined proximal, mirror, and orofacial) in older children with ADHD has been linked with difficulties in motor inhibition,¹³ the persistence of overflow after TBI may be an important marker for broader functioning. Further research is required to examine the relationship between subtle motor function and real-world adaptive behavior in children with TBI. During clinical examinations of children with chronic TBI who may not have adequate information about their TBI or early course of recovery, the simple bedside examination of subtle motor signs such as overflow may provide insight into injury severity. However, further longitudinal research is required to track the developmental trajectory of subtle motor signs following severe pediatric brain injury. Rehabilitation efforts in children with TBI must not only focus on a return to preinjury or near-normal level of performance with regard to skills directly disrupted by the brain injury but also involve long-term follow-up to ensure age-appropriate acquisition of skills as the child ages. The study findings further indicate the need for extended follow-up assessments with measures sensitive to motor deficits after pediatric TBI, with therapy targeting motor recovery to prevent deficits from appearing at later developmental stages.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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FIGURE 1.

Robust negative correlation between PANESS total score and motor/premotor cortex volume in the TBI group such that greater volume correlated with better motor skill.

TABLE 1.

The PANESS tasks

Components	Tasks
Gaits and stations	Stressed gaits: heel walking, toe walking, walking on the sides of the feet, and tandem gait. Stations: Stand for 20 secs with (a) feet together, eyes closed, arms outstretched; (b) feet comfortable, eyes closed, tongue protruding; (c) tandem stance. Stand (30 secs) and hop on each foot (50 hops).
Total timed	Repetitive limb tasks: foot tap, heel-toe tap, hand pat, hand pronation/supination, finger tap, and sequenced finger apposition. One repetitive tongue task: wag (side to side).
Overflow	Assessed during the total timed limb tasks.
Proximal	Extraneous movement on the same side of the body involving larger muscle groups, even if in a different limb (e.g., lifting at elbow rather than wrist during hand patting).
Mirror	Extraneous movement on the same limb on the opposite side of the body.
Total	All gaits and stations and total timed tasks

TABLE 2.

The TBI participant demographics and clinical characteristics

No	Age/Sex	Age at Injury	Severity	MRI Findings at Time of Research Participation
1	16.85/F	13	Severe	Hemosiderin deposition at right anterior frontal and temporal lobes
2	17.8/M	14.2	Severe	Mild bifrontal cortical atrophy, microhemorrhages in subcortical frontal and temporal regions bilaterally
3	15.8/M	13.8	Severe	Left frontoparietal junction encephalomalacia, gliosis in subcortical white matter of the rostral part of the superior left frontal gyrus
4	14.4/M	13.2	Moderate	Normal
5	14.5/M	9.5	Severe	Bifrontal encephalomalacia
6	15.2/F	.66	Severe	Multifocal encephalomalacia in left parietal and occipital lobes, left temporoparietal regions, and center inferior frontal lobe.
7	18.99/M	17.2	Severe	Multicystic encephalomalacia in the inferior bilateral frontal lobes, Wallerian degeneration in the cerebral peduncles, scattered white matter lesions, ex vacuo dilatation of bilateral lateral ventricles
8	16.8/M	15.7	Moderate	Interval involution of previous left frontal hematoma with central cystic region, gliosis in center frontal lobe subcortical white matter
9	17.8/F	16.8	Moderate	Left inferolateral temporal encephalomalacia
10	17.4/M	16.4	Moderate	Normal
11	13.4/F	12.3	Moderate	Normal
12	13.2/F	1.9	Severe	Cystic encephalomalacia of bilateral frontal lobes (R > L), focal protrusion of CSF along the anterior margin of left frontal lobe into the frontal bone with marked thinning of the overlying cortex.
13	11.3/M	8.8	Severe	Left temporal lobe encephalomalacia, postsurgical changes of centerfrontal craniotomy, left frontal ventriculostomy tract
14	16.4/F	15.4	Moderate	Scan not completed

TABLE 3.

The Means and standard deviations for PANESS scores for control and TBI groups

PANESS	Controls		TBI		Moderate TBI		Severe TBI		P
	M (SD)		M (SD)		M (SD)		M (SD)		
Gaits and stations	3.21 (3.02)		7.29 (6.02)		5.33 (2.88)		8.75 (7.46)		NS
Total timed	11.29 (6.66)		21.64 (7.55)		20.33 (4.5)		22.63 (9.43)		NS
Overflow									
Proximal	.07 (.27)		1.29 (1.44)		0 (0)		2.25 (1.17)		<i>b</i>
Mirror	.86 (1.35)		.93 (1.0)		.83 (.98)		1.00 (1.07)		NS
Total	14.50 (8.58)		29.14 (11.05)		25.67 (2.81)		31.75 (14.3)		NS
Brain measures									
Total cerebral volume	1,085,114.2 (122,608.8)		950,835.4 (98,966.7)		967,934.62 (111,934.2)		940,148.3 (96,392.1)		NS
Motor/premotor cortex	54,289.4 (5641.2)		50212.8 (6136.6)		49,206.8 (7074.9)		50,841.6 (5897.3)		NS

NS indicates non-significant $P > 0.05$.^a $P < 0.05$.^b $P < 0.008$.