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The Neurological Outcome Scale for Traumatic Brain Injury (NOS-TBI): I. Construct Validity

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Abstract

The Neurological Outcome Scale for Traumatic Brain Injury (NOS-TBI) is a measure adapted from the National Institutes of Health Stroke Scale (NIHSS), and is intended to capture essential neurological deficits impacting individuals with traumatic brain injury (TBI) (see Wilde et al., 2010). In the present study we evaluate the measure's construct validity via comparison with a quantified neurological examination performed by a neurologist. Spearman rank-order correlation between the NOS-TBI and the neurological examination was $\rho = 0.76$, $p < 0.0001$, suggesting a high degree of correspondence (construct validity) between these two measures of neurological function. Additionally, items from the NOS-TBI compared favorably to the neurological examination items, with correlations ranging from 0.60 to 0.99 (all $p < 0.0001$). On formal neurological examination, some degree of neurological impairment was observed in every participant in this cohort of individuals undergoing rehabilitation for TBI, and on the NOS-TBI neurological impairment was evident in all but one participant. This study documents the presence of measurable neurological sequelae in a sample of patients with TBI in a post-acute rehabilitation setting, underscoring the need for formal measurement of the frequency and severity of neurological deficits in this population. The results suggest that the NOS-TBI is a valid measure of neurological functioning in patients with TBI.

Key words: Neurological Outcome Scale for Traumatic Brain Injury; outcome; traumatic brain injury; validity

Introduction

The Neurological Outcome Scale for Traumatic Brain Injury (NOS-TBI) is a measure adapted from the National Institutes of Health Stroke Scale (NIHSS; downloadable at http://www.strokecenter.org/trials/scales/nihss.html), and is intended to capture essential neurological deficits impacting individuals with traumatic brain injury (TBI). The scale contains domains similar to the NIHSS: level of consciousness, best gaze, visual field, facial palsy, and motor arm and motor leg, for which each domain is categorically rated by level of impairment to absence of abnormality (see Wilde et al., 2010 for a more detailed description of the modifications to the NIHSS). Although the NIHSS has undergone adequate validation in stroke populations through comparison with other stroke scales (D'Olhaberriague et al., 1996; Lyden and Lau, 1991; Lyden et al., 2001; Young et al., 2005), the Glasgow Outcome Scale, and imaging (Brott et al., 1989; Derex et al., 2004; Lyden et al., 2004; Meyer, 1998; Saver et al., 1999; Schiemanck et al., 2005), measures of neurological functioning such as the NIHSS are rarely validated against what is arguably the gold standard for neurological functioning: a clinical neurological examination performed by a neurologist. Accordingly, we evaluated the construct validity of the NOS-TBI using a quantified clinical neurological examination in a sample of patients with TBI currently undergoing post-acute rehabilitation. Construct validity is defined as the degree to which a scale measures an unobservable construct (e.g., fluid

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intelligence or motivation) that the scale purports to measure (Anastasi and Urbina, 1997; Nunnally and Bernstein, 1994), in this case neurological functioning.

Methods

Informed consent was obtained from the participant, a legal authorized representative, or a parent/guardian (for adolescents under 18 years of age) through an informed consent form, and the procedure was approved by the Institutional Review Board of Baylor College of Medicine and its affiliate institutions.

Participants

The same patient sample was used to test construct validity, as well as convergent validity and reliability for the NOS-TBI, which is detailed in the article by McCauley and associates (McCauley et al., 2010). Briefly, 50 participants (45 male and 5 female) who had sustained TBI, with a mean Glasgow Coma Scale (GCS; Teasdale and Jennett, 1974) score of 6 (SD = 3.2; range 3–15; median = 6) were enrolled in the study. The typical participant was injured as a result of a motor vehicle accident, and was a non-Hispanic Caucasian with a positive CT scan and a loss of consciousness. Inclusion criteria were patients between the ages of 15 and 65 years who sustained a TBI (mean age 33.3 ± 12.9 years, and mean time post-injury was 2.9 ± 2.4 months). Patients were excluded if they had evidence of a penetrating head injury, spinal cord injury, history of a premorbid neurological or major psychiatric disorder (e.g., schizophrenia or bipolar disorder), or if they were >18 months post-TBI. Participants were evaluated while participating in inpatient rehabilitation. Participants were selected with the intention of sampling the widest distribution of injury severity, time post-injury, and functional level.

Procedures

Administration of the neurological examination. The neurological examination consisted of procedures that would be performed as part of a standard clinical assessment, including testing of mental status, language, cranial nerves, strength and muscle tone, sensation, deep tendon reflexes, coordination, stance, and gait. Although under normal clinical circumstances the examination is performed after the physician obtains the patient's history, during the study, in the interest of time, the neurological examination was begun with very limited or no significant information about the patient's condition. The examination followed the same procedure in all patients. As part of mental status testing, the physician asked the patient the reason for his or her stay in the hospital. In the interest of time, mental status testing was limited to the evaluation of a very limited list of functions. As the examination progressed, information gathered from the patient and family members was used to explore selected aspects of patient functioning in more detail.

Mental status examination consisted of testing of orientation and the ability to answer simple questions, and to follow single and multi-step commands. Language and speech evaluations consisted of testing of both expressive (dysarthria and fluency in speech and confrontation naming), and receptive (comprehension and ability to follow complex commands) abilities. Cranial nerve testing was performed in a standard fashion for all cranial nerves. Coordination testing consisted of finger-nose-finger testing, heel-knee-shin testing, and rapid alternating movements. Testing of sensation included light touch, pinprick, temperature, vibration, and/or position sense in all four extremities. Muscle strength was recorded using the 0–5 scale of the Medical Research Council. Deep tendon reflexes (DTRs) were recorded using a traditional 0–4 scale, and graded as hypoactive (score of 0 or 1) or hyperactive (score of 3 or 4). When possible, other neurological abnormalities were graded using ratings of normal, mildly abnormal/impaired, and significantly impaired.

Although we did not anticipate a high frequency of cranial nerve (CN) abnormalities for the trochlear (CN IV), abducens (CN VI), glossopharyngeal (CN IX), vagus (CN X), spinal accessory (CN XI), and hypoglossal (CN XII) nerves, and the clinical testing of functions controlled by these nerves is limited, we included them in the neurological assessment because (1) they are part of the evaluation of CN and brainstem function, and (2) to formally verify the frequency of these abnormalities in this population. As a clinical examination also includes assessment of muscle bulk, resistance to passive manipulation strength, sensation, DTRs, and coordination, these were also retained in the clinical examination to determine the frequency of these findings in our sample. Stance and gait evaluation and the Romberg test were performed, again to document the frequency of abnormalities.

The neurological examination occurred within 48 h of administration of the NOS-TBI, and it was administered by a neurologist, generally in the patient's room or a nearby examination room in the unit. The same neurologist (P.M.) performed all neurological examinations, using a standard schedule to ensure inclusion of all elements that could be tested, and consistent scoring elements and procedures. Scoring of the examination was performed independently of the NOS-TBI raters. Depending on the schedule constraints of the patients, the neurologist, and the raters of the NOS-TBI, the neurological examination was performed either before or after the NOS-TBI. All items that could be safely and reasonably performed without causing the patient undue discomfort or risk were assessed. The neurological evaluation lasted approximately 30–45 min in most patients.

A standard set of assessment instruments was utilized for each examination: a Clark eye card for examination of visual acuity; a penlight for testing of pupillary response; disposable safety pins (pinprick), and a tuning fork (temperature and vibration) were used for examination of sensation; a reflex hammer was used for DTRs; and essential oils were used for olfactory testing. Everyday objects (e.g., pen, watch, and clothing parts) were used for testing of confrontation naming.

Scoring of the neurological examination

To calculate the neurological summary score, ratings from items were generally summed; however, some scores required recoding or other transformation before summing. Comprehension and articulation items were recoded as the highest level of impairment if the item was unable to be assessed. Strength ratings were reverse scored (1 = best strength, 5 = lowest strength) so that higher scores indicated higher levels of impairment, which conformed to the scoring of other test items. Muscle bulk and DTRs were not included in the scoring of the neurological examination total score, as

these are not tested in the NOS-TBI. DTRs were recoded from the original scoring so that degrees of hyper- and hyporeflexia were treated equally, and higher ratings reflected greater impairment. That is, "absent" and "mildly diminished" reflexes were recoded as 2 and 1, respectively, and "mildly increased" and "hyperreflexic" were recoded as 1 and 2, respectively. Although hyper- and hyporeflexia indicate upper and lower motor neuron disease and different probable lesion locations, for the purposes of this quantified examination, both were coded similarly as degrees of abnormality of reflexes. Items for stance and gait were coded as normal (0) or abnormal (1). Because of the large number of strength-related items, these test items were collapsed, forming four quadrants (right and left, and upper and lower extremity), each with a mean strength score (a reduction of 30 items to 4), corresponding to the same strength quadrants on the NOS-TBI. Only the items from "arousal" to "neglect/extinction" were summed to determine the quantified neurological examination score. In situations in which certain items were untestable, either due to the patient's degree of neurological impairment, failure to cooperate, or the presence of a condition or medical treatment device that would preclude testing, we applied the following rules: (1) for extinction/neglect, the patient received a 0 when there were sensory deficits, since extinction could not be reliably assessed in the presence of primary sensory modality deficits, with the rationale being that (2) for ataxia, a 0 was scored in the presence of profound weakness of the extremities, again with the rationale that for ataxia to be present, it would have to be outside the presence of deficits in strength, and (3) for dysarthria, a 0 was scored when speech could not be tested, using the rationale that the presence of dysarthria is impossible to determine in the absence of speech. Although this is not typical of clinical practice in neurology, we anticipated that these distinctions would be difficult for non-neurologists to make using the NOS-TBI.

Items from "deep tendon reflexes" to "stance and gait" were not included in the total quantification score; instead, these were considered supplemental items. Not all patients were able to have these items evaluated, depending on level of consciousness or other contraindications, such as hip fracture, pain, or other reasons for non-weight-bearing status, a frequent occurrence in this population. When hearing, vision, or olfaction could not be tested secondary to diminished level of consciousness, these were scored with the highest possible score, consistent with the NOS-TBI, and the rationale that decreased consciousness assumes impairment in sensation.

Administration of the Neurological Outcome Scale for Traumatic Brain Injury

To examine the relationship of the NOS-TBI to the neurological examination, the rating of a non-physician (neuropsychologist) was used. The rater had completed NIHSS certification using the NIHSS certification training videotapes, and had received limited training by the study neurologist prior to this validation study. The NOS-TBI was performed using testing kits created for this measure, which included laminated cards from the Boston Diagnostic Aphasia Examination (Goodglass and Kaplan, 1983), which are the stimuli used in the NIHSS (i.e., the "Cookie Theft" picture and the object-naming picture), as well as laminated cards for the aphasia and dysarthria items. Disposable safety pins for testing sensation, penlights for testing pupillary response, and essential oils for olfactory testing were also included, as well as a laminated card with multiple-choice items for olfactory testing. The NOS-TBI was also administered in the participant's hospital room or in a nearby testing or conference room. Administration of the measure typically required 15–20 min.

Scoring of the Neurological Outcome Scale for Traumatic Brain Injury

In contrast to the scoring of the neurological examination, we applied a different scoring rule (i.e., maximum impairment ratings) for the items of extinction, ataxia, and dysarthria (in situations in which the items were untestable, either due to the patient's degree of neurological impairment or failure to cooperate), and for supplemental test items, including gait and limb ataxia (in situations in which the participant was unable to complete the measure due to pain, weight-bearing status, orthopedic injury, or medical devices that precluded administration of these items). The rationale for this procedure was to make the assignment of points on the NOS-TBI consistently decrease over the course of recovery, instead of occasional increases because an item could be assessed at a later time. Both methods of scoring these items were employed ("untestable" counted as either 0 impairment points or maximum impairment points), and neither method substantially changed the correlation between the neurological examination score and the NOS-TBI score. This suggests that the scoring method of these items did not substantially alter the measurement of the underlying construct of neurological impairment (the primary objective of this analysis).

Statistical analysis

All analyses were conducted using SAS software for Windows, version 9.2 (SAS Institute Inc., 2008). Statistical significance was defined as $\alpha = 0.05$ for all analyses unless otherwise specified. Data were screened for entry errors and necessary corrections were made. The Spearman rank-order correlation between the NOS-TBI score and the quantified clinical neurological examination score was used to evaluate construct validity.

We explored the use of a dichotomous scoring system (summing scores of 0-normal and 1-abnormal) for the quantified clinical neurological examination to determine if this procedure was preferable. Because the methodology for the quantification of a qualitative clinical neurological examination has not been presented in the literature to our knowledge, we explored both of the above quantification methods (summed graded and summed dichotomous scores). Neither method produced strikingly different correlations with the NOS-TBI; scores derived using the summed graded scoring procedures are reported below.

The NOS-TBI and the neurological examination assess similar types of neurological functioning, but through different means or with performance summed over wider body distributions. To demonstrate that the NOS-TBI conducted by non-neurologists fares well compared to expert neurological examination ratings, item-to-item correlations were calculated for items that were included in both examinations. Items from the neurological examination were grouped anatomically as appropriate.

Results

Neurological examination

Overall score. The mean quantified neurological examination score (which was the sum of all item scores for which a lower score indicates fewer and/or less severe deficits) was 67.6 (SD = 61.3, median = 50, range 6–247). In formal neurological testing, every participant demonstrated one or more types of neurological abnormality. The incidence of specific neurological deficits that were common to the NOS-TBI, and those that were not measured via the NOS-TBI is detailed below.

Frequency of neurological deficits not assessed with the NOS-TBI. As anticipated, the frequency of CN abnormalities for nerves formally tested as part of the neurological examination (but not assessed on the NOS-TBI) was indeed low, with no or few patients demonstrating abnormalities of the trochlear (CN IV; 2%), abducens (CN VI; 4%), glossopharyngeal (CN IX; 4%), vagus (CN X; 4%), spinal accessory (CN XI; 6%), and hypoglossal (CN XII; 6%) nerves. Visual acuity was diminished in 4% of patients. Muscle bulk was diminished in 20–30% of patients, particularly in the lower limbs (muscle groups including the quadriceps, gastrocnemius/soleus, and tibialis anterior). Detailed strength testing revealed abnormalities in 16–26% of patients, most notably in the upper arm (i.e., deltoids, biceps, and triceps), and also in leg flexion. Of all the DTRs evaluated, abnormalities were present in about 54–66% of patients. Abnormalities of stance or gait were present in 15.4–26.9% of weight-bearing patients who could undergo testing of these items (at least 48% of the total sample was non-weight-bearing). A positive Romberg test was present in only 7.6% of weight-bearing patients who could undergo this test item.

Neurological Outcome Scale for Traumatic Brain Injury

The mean NOS-TBI score was 12.7 (SD = 13.1, median = 7, range 0–56). Frequencies of normal, abnormal, and untestable items from the NOS-TBI are presented in Table 1. Not surprisingly, the most common abnormality was impaired olfaction, which was present in 76% of patients. Language impairment (46%) and disorientation (42%) were the next most common abnormalities, followed by impairment in gross motor functioning (30–38%), facial paresis (30–34%), and sensory functioning (14–28%).

Construct validity

The Spearman rank-order correlation between the NOS-TBI and the total neurological examination was $\rho = 0.76$, $p < 0.0001$ (57.8% shared variance), suggesting a high degree of correspondence between these two measures of neurological function. In terms of item-to-item correspondence, items from the NOS-TBI compared favorably to the neurological examination items, with correlations ranging from 0.60 to 0.99 (all $p < 0.0001$), as listed in Table 2. When correlations were not calculable (one scale rated all participants with the same level of functioning), the raw agreement in scores between the

Table 1. Frequencies for Normal and Abnormal Performance on Items Included in the Neurological Outcome Scale for Traumatic Brain Injury (NOS-TBI)

	NOS-TBI test items	*Normal*	*Abnormal*	*Untestable*
1a	LOC	46 (92.0%)	4 (8.0%)	0 (0%)
1b	LOC questions	29 (58.0%)	21 (42.0%)	0 (0%)
1c	LOC commands	43 (86.0%)	7 (14.0%)	0 (0%)
2	Gaze	42 (84.0%)	8 (16.0%)	0 (0%)
3a	Visual field right	42 (84.0%)	8 (16.0%)	0 (0%)
3b	Visual field left	46 (92.0%)	4 (8.0%)	0 (0%)
4	Pupillary response	42 (84.0%)	8 (16.0%)	0 (0%)
5a	Hearing right	44 (88.0%)	6 (12.0%)	0 (0%)
5b	Hearing left	43 (86.0%)	7 (14.0%)	0 (0%)
6a	Facial paresis right	35 (70.0%)	15 (30.0%)	0 (0%)
6b	Facial paresis left	33 (66.0%)	17 (34.0%)	0 (0%)
7a	Motor right upper extremity	31 (62.0%)	19 (38.0%)	0 (0%)
7b	Motor left upper extremity	35 (70.0%)	15 (30.0%)	0 (0%)
8a	Motor right lower extremity	35 (70.0%)	15 (30.0%)	0 (0%)
8b	Motor left lower extremity	34 (68.0%)	16 (32.0%)	0 (0%)
9a	Sensory right upper extremity	42 (84.0%)	8 (16.0%)	0 (0%)
9b	Sensory left upper extremity	37 (74.0%)	13 (26.0%)	0 (0%)
9c	Sensory right lower extremity	43 (84.0%)	7 (14.0%)	0 (0%)
9d	Sensory left lower extremity	36 (72.0%)	14 (28.0%)	0 (0%)
10	Best language	27 (54%)	23 (46.0%)	0 (0%)
11	Dysarthria	30 (60.0%)	13 (26.0%)	7 (14.0%)
12	Neglect	47 (94.0%)	6 (12.0%)	0 (0%)
13	Smell	11 (22.0%)	38 (76.0%)	1 (2.0%)
14	Gait ataxia	12 (24%)	4 (8.0%)	34 (68.0%)
15a	Limb ataxia right	29 (58.0%)	6 (12.0%)	15 (30.0%)
15b	Limb ataxia left	28 (56.0%)	5 (10.0%)	17 (34.0%)

LOC, loss of consciousness.

Table 2. Item-to-Item Spearman Rank-Order Correlations for the Neurological Outcome Scale for Traumatic Brain Injury (NOS-TBI) and the Quantified Neurological Exam

NOS-TBI	*Neurological exam*	r_s	p *Value*
LOC	Arousal	0.99	<0.0001
LOC questions	Orientation	0.69	<0.0001
LOC commands	Commands	0.71	<0.0001
Gaze	Extraocular movements (sum left and right)	0.73	<0.0001
Visual field right	Visual field right	0.76	<0.0001
Visual field left	Visual field left	0.66	<0.0001
Pupil	Pupil direct light (sum left and right)	0.63	<0.0001
Hearing right	Hearing right	0.87	<0.0001
Hearing left	Hearing left	0.81	<0.0001
Facial paresis right	Facial muscles right	0.62	<0.0001
Facial paresis left	Facial muscles left	0.64	<0.0001
Motor RUE	Motor RUE (sum deltoid and interossei)	0.71	<0.0001
Motor LUE	Motor LUE	0.71	<0.0001
Motor RLE	Motor RLE (sum hip to foot)	0.67	<0.0001
Motor LLE	Motor LLE	0.72	<0.0001
Sensory RUE	Sensory RUE (sum deltoid and interossei)	0.64	<0.0001
Sensory LUE	Sensory LUE	0.67	<0.0001
Sensory RLE	Sensory RLE (sum hip to foot)	0.61	<0.0001
Sensory LLE	Sensory LLE	0.63	<0.0001
Best language	Comprehension	0.81	<0.0001
Dysarthria	Articulation	0.83	<0.0001
Neglect/extinction	Visual/tactile extinction (sum)	0.81	<0.0001
Smell	Olfaction	0.78	<0.0001
Gait ataxia	Tandem walk	0.88	<0.0001
Limb ataxia right	FNF and HKS	0.65	<0.0001
Limb ataxia left	FNF and HKS	0.60	<0.0001

r_s, Spearman correlational coefficient; LOC, loss of consciousness; RUE, right upper extremity; LUE, left upper extremity; RLE, right lower extremity; LLE, left lower extremity; FNF, finger-nose-finger test; HKS, heel-knee-shin test.

scales was 93.75%, suggesting excellent correspondence between the two scales.

Discussion

The present study documents the presence of measurable neurological sequelae in a sample of patients with TBI undergoing post-acute rehabilitation, underscoring the need for formal measurement of the frequency and severity of these deficits in this population. On formal neurological examination, some degree of neurological impairment was observed in every participant, and on the NOS-TBI neurological impairment was evident in all but one participant. While we acknowledge the composition of adult patients with primarily severe injury undergoing rehabilitation, 16% of our patient sample fell into the moderate and complicated mild TBI categories, and these patients also demonstrated some degree of neurological deficit.

To our knowledge, the NIHSS has never been validated against a formal and more extensive clinical neurological examination performed by a neurologist, and this study lends further support for the validity of the items that the NIHSS and NOS-TBI have in common. The original validation of the NIHSS was performed using examination of the relationship between computed tomography (CT)-based lesion areas and the NIHSS, but given the contribution of diffuse axonal injury and other non-focal forms of injury in TBI, as well as the historically poor relationship between day-of-injury CT and outcome in TBI (Bigler et al., 2006), we opted to perform a validation study of the NOS-TBI to include what is arguably the most important gold standard measure for neurological functioning: the clinical neurological examination. The NOS-TBI demonstrated excellent construct validity with the quantified neurological examination.

Comparison of a brief standardized measure such as the NOS-TBI to a more fluid clinical examination is challenging, and we acknowledge the differences in the administration and procedures used in the measurement of some of these items. For example, two of the lower correlations (0.62 and 0.64) in the analysis were for facial paresis, which was tested only by observation for the NOS-TBI examiners, but by both observation and physical examination for the neurologist. Similarly, the correlation between the "LOC orientation" on the neurological examination and "LOC questions" on the NOS-TBI was lower than anticipated, as the examiners of the NOS-TBI used simple questions such as "What month is it?" and "What is your age?," while the neurological examination questions were more detailed, with questions about the patient name, age, detailed spatial and temporal orientation, and reason for being in the hospital. However, despite these differences, all item-to-item correlations were high and significant, suggesting that NOS-TBI items administered by non-neurologists fared well compared to expert neurological examination ratings.

Clearly, the most common neurological deficit present in our sample was decreased or absent olfaction. This was present in 76% of the sample, despite many patients failing to

report impaired olfaction when queried. Given the vulnerability of the olfactory nerves to damage during head trauma, this adaptation of the NIHSS to include this item is important in this population. Other items that are not part of the NIHSS such as hearing and pupillary response were also impairments detected in a substantial number of patients (12–14% and 16%, respectively) with TBI.

Undoubtedly, the NOS-TBI is not intended to replace clinical testing of neurological functioning by specialty-trained physicians. However, in actual practice many patients with TBI are not actively followed by neurologists during the post-acute and chronic recovery periods. Additionally, the cost and feasibility of including detailed neurological testing in natural history observational research or clinical trials are generally prohibitive, and given these issues, this scale provides a reasonable alternative to the measurement and monitoring of these deficits.

As anticipated, the frequency of CN abnormalities for the nerves formally tested as part of the neurological examination, but not the NOS-TBI, was indeed low (2–6%), and supports the exclusion of these items from the scale. Abnormal DTRs were found in only 10% of participants, with the notable exception of the Achilles tendon; since the NOS-TBI was designed to be administered by personnel that would not have the appropriate training to accurately perform DTR testing, and in view of the low number of DTR abnormalities in most body areas, these items were not included in the NOS-TBI. Decreased muscle bulk was found in 20–30% of patients, usually in the legs. Loss of muscle bulk would be expected from deconditioning during acute hospitalization, and likely could not be reliably assessed except by trained medical personnel, and thus these items were omitted from the NOS-TBI. Although a high percentage of participants presented abnormalities on expanded strength testing (32–40%), these additional items were not included in the NOS-TBI for the same reasons as muscle bulk items.

The supplemental NOS-TBI items appear to be relevant to patients with TBI, but we acknowledge the challenges in the assessment of these items in this population, due to orthopedic injury, non-weight-bearing status, and other safety considerations during the post-acute recovery period. These difficulties, however, are somewhat unique to the post-acute injury phase of the patients in this sample. We anticipate that these situations will be less frequent at more chronic intervals. In our sample, 30–68% of patients were unable to perform these items. However, inclusion of these items did not significantly change the overall correlation between the NOS-TBI and quantified neurological examination score (i.e., there was no serious threat to the measurement of the underlying construct of neurological functioning), as noted above.

There are some limitations in this study that warrant brief discussion. Complete or partial resolution of some neurological deficits is likely to occur during the course of recovery. Participants in our study were generally in the post-acute stage of recovery, and the frequency and severity of the deficits seen during acute or more chronic intervals requires further investigation. Nonetheless, we examined patients at less than 18 months post-injury, with a mean post-injury interval of 3.1 months, an endpoint that would be commonly examined in clinical and observational studies.

Conclusions

In conclusion, the current study has demonstrated strong construct validity of the NOS-TBI when matched against a quantified neurological examination; the NOS-TBI accounted for a substantial portion of the shared variance in the standard clinical neurological examination, even with a limited number of tests that were performed by non-neurologists. Overall, the preliminary data indicate that the NOS-TBI has excellent potential as a clinical and research instrument for measuring the neurological functioning of patients with TBI from acute through chronic recovery.

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References

Anastasi, A., and Urbina, S. (1997). *Psychological Testing*, 7th ed. Prentice Hall, Inc.: Upper Saddle River, NJ.

Bigler, E.D., and Ryser, D.K., Gandhi, P., Kimball, J., and Wilde, E.A. (2006). Day-of-injury computerized tomography, rehabilitation status, and development of cerebral atrophy in persons with traumatic brain injury. Am. J. Phys. Med. Rehabil. 85, 793–806.

Brott, T., Adams, H.P., Jr., Olinger, C.P., Marler, J.R., Barsan, W.G., Biller, J., Spilker, J., Holleran, R., Eberle, R., Hertzberg, V., et al. (1989). Measurements of acute cerebral infarction: a clinical examination scale. Stroke 20, 864–870.

Derex, L., Nighoghossian, N., Hermier, M., Adeleine, P., Berthezene, Y., Philippeau, F., Honnorat, J., Froment, J.C., and Trouillas, P. (2004). Influence of pretreatment MRI parameters on clinical outcome, recanalization and infarct size in 49 stroke patients treated by intravenous tissue plasminogen activator. J. Neurol. Sci. 225, 3–9.

D'Olhaberriague, L., Litvan, I., Mitsias, P., and Mansbach, H.H. (1996). A reappraisal of reliability and validity studies in stroke. Stroke 27, 2331–2336.

Goodglass, H., and Kaplan, E. (1983). The Boston diagnostic aphasia examination. Lea and Febiger: Philadelphia.

Lyden, P., Claesson, L., Havstad, S., Ashwood, T., and Lu, M. (2004). Factor analysis of the National Institutes of Health Stroke Scale in patients with large strokes. Arch. Neurol. 61, 1677–1680.

Lyden, P.D., and Lau, G.T. (1991). A critical appraisal of stroke evaluation and rating scales. Stroke 22, 1345–1352.

Lyden, P.D., Lu, M., Levine, S.R., Brott, T.G., and Broderick, J. (2001). A modified National Institutes of Health Stroke Scale for use in stroke clinical trials: preliminary reliability and validity. Stroke 32, 1310–1317.

McCauley, S.R., Wilde, E.A., Kelly, T.M., Weyand, A.M., Yallampalli, R., Pedroza, C., Schnelle, K.P., Boake, C., Levin, H.S.,

and Moretti, P. (2010). The Neurological Outcome Scale for Traumatic Brain Injury (NOS-TBI): II. Reliability and Convergent Validity. J. Neurotrauma 27, ???–???.

Meyer, M.A. (1998). Quantitative brain SPECT and the NIH Stroke Scale. Stroke 29, 1480.

Nunnally, J.C., and Bernstein, I.H. (1994). *Psychometric Theory*, 3rd ed. McGraw-Hill, Inc.: New York.

SAS Institute Inc. (2008). Statistical Analysis Software for Windows. SAS Institute, Inc.: Cary, NC.

Saver, J.L., Johnston, K.C., Homer, D., Wityk, R., Koroshetz, W., Truskowski, L.L., and Haley, E.C. (1999). Infarct volume as a surrogate or auxiliary outcome measure in ischemic stroke clinical trials. The RANTTAS Investigators. Stroke 30, 293–298.

Schiemanck, S.K., Post, M.W., Witkamp, T.D., Kappelle, L.J., and Prevo, A.J. (2005). Relationship between ischemic lesion volume and functional status in the 2nd week after middle cerebral artery stroke. Neurorehabil. Neural Repair 19, 133–138.

Teasdale, G., and Jennett, B. (1974). Assessment of coma and impaired consciousness. A practical scale. Lancet 2, 81–84.

Wilde, E.A., McCauley, S.R., Kelly, T.M.,. Levin, H.S., Pedroza, C., Clifton, G.L., Robertson, C.S., and Moretti, P. (2010). Feasibility of the Neurological Outcome Scale for Traumatic Brain Injury (NOS-TBI) in Adults. J. Neurotrauma 27, 975–981.

Young, F.B., Weir, C.J., and Lees, K.R. (2005). Comparison of the National Institutes of Health Stroke Scale with disability outcome measures in acute stroke trials. Stroke 36, 2187–2192.

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