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Motor impairment after severe traumatic brain injury: A longitudinal multicenter study

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Abstract—Neuromotor impairment is a common sequela of severe traumatic brain injury (TBI) but has been understudied relative to neurocognitive outcomes. This multicenter cohort study describes the longitudinal course of neurological examination-based motor abnormalities after severe TBI. Subjects were enrolled from the four lead Department of Veterans Affairs and Defense and Veterans Brain Injury Center sites. The study cohort consisted of 102 consecutive patients (active duty, veteran, or military dependent) with severe TBI who consented during acute rehabilitation for data collection and completed all follow-up evaluations. Paresis, ataxia, and postural instability measures showed a pattern of improvement over time, with the greatest improvement occurring between the inpatient (baseline) and 6-month follow-up assessments. Involuntary movement disorders were rare at all time points. Two years following acute rehabilitation, more than one-third of subjects continued to display a neuromotor abnormality on basic neurological examination. Persistence of tandem gait abnormality was particularly common.

Key words: ataxia, brain injury, closed head injury, dyskinesias, measurement, neurological examination, outcomes, paresis, postural balance, rehabilitation.

INTRODUCTION

Neuromotor impairment is a common sequela of severe traumatic brain injury (TBI), yet the existing literature provides sparse observational data. Longitudinal reports on the rate and extent of recovery from neuromotor impairment are particularly limited. The few published human studies that examined rate of motor recovery focused on functional rather than impairment measures. Swaine and Sullivan showed that motor function improved markedly between the first and sixth weeks after TBI [1]. Others have reported the now well-established fact that motor function usually improves during acute rehabilitation [2–3]. In terms of the underlying motor impairment, investigators have focused on its functional prognostic significance rather than its longitudinal course [4–5].

Long-term neuromotor impairment is similarly understudied relative to other outcome domains. Published TBI outcome studies are skewed toward global measures and/or measures within the behavioral and cognitive rather than physical domains. A recent literature review of all TBI outcome studies from 1990 to 2004 by Williams et al. found only 175 articles that reported on mobility or physical outcome after TBI [6]. In virtually all of these studies, the mobility and physical domains were assessed by

Abbreviations: DVBIC = Defense and Veterans Brain Injury Center, FIM = Functional Independence Measure, LOC = length of coma, MMT = manual muscle testing, PTA = posttraumatic amnesia, TBI = traumatic brain injury, TBIMS = Traumatic Brain Injury Model Systems, VA = Department of Veterans Affairs, VAMC = VA medical center.

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functional or disability instruments, without the inclusion of impairment measures. The most commonly used measurement instruments were the Functional Independence Measure (FIM) [7–8], a structured interview, or a symptom survey. For example, Ponsford et al. used a structured interview 2 years after TBI and found frequent somatic symptoms, with 36 percent of patients reporting dizziness and 48 percent reporting visual symptoms [9]. Dombovy and Olek interviewed a sample of 48 patients with mild-moderate TBI over the telephone and found that most were physically independent on the FIM at 3 and 6 months postinjury [10]. Katz et al., in a retrospective study of 116 persons with severe TBI, determined that 73 percent were ambulatory <6 months postinjury [11]. Hillier et al. interviewed 67 subjects with TBI of mixed severity (mostly severe) 5 years postinjury and found that balance deficit was the second most frequent (45%) self-reported physical symptom after headache [12]. In a rare reporting, Hillier et al. also performed physical examination in a subset of patients and identified hemiparesis in 15 percent and quadraparesis in 6 percent [12].

In the few published TBI outcome studies that have reported physical impairment measures, neurological examination is rarely described. Several investigators used sophisticated postural stability measures to show persisting balance abnormalities after TBI. Lehmann et al. showed abnormal sway using force-plate measures [13]. Newton demonstrated abnormal response to perturbation in subjects with moderate and severe TBI [14]. Wöber et al. showed abnormal computer posturography in subjects up to 5 years after moderate and severe TBI [15]. They also reported that 43 percent of their sample had ataxia on neurological examination, although they did not describe the maneuver(s) used. Basford et al. showed abnormal Kix-Hallpike, caloric irrigation, and computer posturography in 10 subjects with mixed TBI severity 3 months postinjury [16]. These subjects reportedly had "normal" neurological examination, although the elements of the examination were not described for the reader.

Likewise, other investigators have used assessments that extend beyond the routine neurological examination to show persisting deficits in motor integrative function and fine motor skills after TBI. Haaland et al. showed impairment in finger-tapping 1 year after mild-moderate TBI [17]. Chaplin et al. showed impairment in integrative gross motor and speeded fine motor function in a sample of 14 children 16 months after severe TBI [18]. Kuhtz-Buschbeck et al. demonstrated deficits in fine motor skills, speed, and coordination in 23 children 8 months after TBI [19]. Gray et al. showed subclinical bradykinesia with impaired complex and simple reaction time in a sample of 24 persons 1 year after TBI [20]. Incoccia et al. showed residual motor programming deficits in 18 individuals with severe chronic TBI in the presence of good clinically assessed motor recovery [21]. Di Russo et al., measuring movement-related cortical potentials with electroencephalogram, showed selective deficit in motor preparation in seven subjects with severe TBI and clinically established good recovery by the time of testing [22]. Finally, Wiese et al. found altered movement-related potentials in a sample of 22 patients 8 weeks after TBI, which they posited to be a consequence of disturbed neuronal networks involved in the preparation and execution of voluntary movements [23].

Taken together, overwhelming evidence exists for persistent subtle neuromotor deficits after TBI, particularly severe TBI. But negligible data are available on the extent to which neuromotor impairments are present postacutely on the less-sensitive standard neurological examination. Our rationale for the current study was that better elucidation of neurological examination-based outcomes is important because routine neurological examination forms the basis for most medical impairment and disability evaluations. In addition, a fuller description of the degree of neuromotor (i.e., gait, balance, strength) impairment and improvement over time might add insight into the global recovery process and rehabilitation needs of persons with TBI. Our specific study objectives were to describe the longitudinal course of motor findings on routine neurological examination after severe TBI and determine the most frequent short- and long-term motor impairments.

METHODS

This multicenter study was conducted with acute rehabilitation and follow-up patient data from the Defense and Veterans Brain Injury Center (DVBIC). The four participating Department of Veterans Affairs (VA) medical center (VAMC) brain injury units were accredited by the Commission on Accreditation of Rehabilitation Facilities [24] and located in Richmond, Virginia; Tampa, Florida; Minneapolis, Minnesota; and Palo Alto, California. The DVBIC program was created in 1992 through Federal-agency collaboration between the Department of Defense and the VA, as well as the Brain Injury Association of America [25–26]. The DVBIC is an integrated disease-management system whose mission is to provide veterans and military beneficiaries who have sustained TBI with state-of-the-art assessment, rehabilitation, and follow-up. Each DVBIC VAMC brain injury unit delivers comprehensive interdisciplinary acute rehabilitation services, including physiatry and related medical services; nursing; physical, occupational, kinesio-, speech, and recreational therapies; psychological and neuropsychological services; and case management/social work.

The DVBIC prospectively collects and compiles standardized injury, assessment, treatment, and outcome data in order to evaluate the efficacy and cost of brain injury rehabilitation strategies. Patients are typically followed and data collected for 2 years postinjury. The local institutional review board of each participating center approved the data collection and analysis. Preselected data were extracted from this database for the current study. Subject inclusion criteria for the current study were (1) veteran or military beneficiary, (2) a severe TBI (as defined by definite traumatic injury with documented initial Glasgow Coma Scale score <9, length of coma [LOC] >24 hours, and/or posttraumatic amnesia [PTA] duration >7 days), (3) admittance to one of the four DVBIC acute brain injury rehabilitation centers within 6 months of injury, (4) no preinjury history of major neurological deficit, and (5) completed baseline evaluation by 2002 (i.e., only those due to complete 2-year follow-up by 2004).

Informed consent was obtained from each subject or, when lacking capacity, from a legally authorized representative. Patients were evaluated with standard DVBIC procedures during the course of their inpatient rehabilitation stay. Initial research assessment with the full DVBIC battery was generally initiated within 72 hours of receiving informed consent. Demographic information including age, sex, race/ethnicity, and traumatic etiology were obtained. Standard assessment protocols were performed across sites. Physicians performed a structured neurological examination that included measures of the following variables of interest: paresis (upper and lower limb with manual muscle testing [MMT]), movement disorders (tremor, choreic/athetoid, dystonic), ataxia (finger-nose-finger, heel-shin), and postural instability (Romberg, tandem gait). Each measure was categorized as normal, abnormal, or not testable (i.e., inability to follow instruction). For paresis, diffuse motor strength (<4/5 on MMT) or any detectable motor asymmetry (<5/5 on MMT) deemed caused by the current neurological injury was considered abnormal. Patients were reevaluated at approximately 6 months, 12 months, and 2 years after their baseline assessments with the same medical specialists administering the standardized assessment protocol. Subjects were excluded from the final sample if any of the three neurological follow-up evaluations were missing.

Demographic and injury severity characteristics of the final sample were calculated and compared with those of the subjects that were excluded because of one or more missing neurological evaluations. Ordinal variables were compared with independent *t*-tests. Nominal variables were dichotomized for 2 × 2 chi-square analyses. A significance level of 0.05 was defined for these comparisons. Descriptive statistics were used for the longitudinal analyses. McNemar nonparametric tests were used to assess change in each outcome measure between assessment periods, with a significance level of 0.05.

RESULTS

Of 240 eligible subjects, the final sample consisted of 102 consecutive participants who consented to research data collection and who had complete neurological examination data recorded at acute rehabilitation and 6-month, 12-month, and 2-year follow-ups. We excluded 138 subjects for incomplete neurological follow-up data. Demographic characteristics and injury severity measures for the final sample and excluded subjects are displayed in **Tables 1** and **2**, respectively. The final sample consisted of more nonwhite and more active duty subjects compared with those excluded for incomplete follow-up. Regarding injury characteristics, the final study participants also had shorter mean LOC.

Nested cross tabulation showed that the following proportions of participants had normal gross neuromotor examinations (every included variable was normal): 21.6 percent in acute rehabilitation, 54.2 percent at 6 months, 62.9 percent at 12 months, and 64.5 percent at 2 years. Longitudinal neuromotor examination data for each individual measure are displayed in **Table 3** (limb examination measures), **Table 4** (gait and posture measures), and **Table 5** (involuntary movement measures). Similar to the overall neuromotor examination, almost all individual measures showed a pattern of improvement over time, with the bulk of the improvement occurring by the 6-month follow-up. Specifically, statistical analysis

Table 1.
Demographics of study sample (*n* = 240). Data presented as percentages unless otherwise noted.

Variable	Final Sample (*n* = 102)	Excluded (*n* = 138)	*p*-Value
Age, yr (mean ± SD)	29.3 ± 11.3	32.3 ± 13.6	0.08
Male	93.1	93.5	0.92
Married	29.6	28.7	0.88
Race/Ethnicity (white)	60.6	72.8	0.049*
Education (HS graduate)	62.5	55.6	0.30
Employed Full-Time	85.0	86.0	0.82
Active Duty	75.0	61.0	0.02*

*$p < 0.05$.
HS = high school, SD = standard deviation.

Table 2.
Injury severity of study sample (*n* = 240). Data presented as mean ± standard deviation unless otherwise noted.

Variable	Final Sample (*n* = 102)	Excluded (*n* = 138)	*p*-Value
Initial GCS	6.11 ± 2.88	6.33 ± 3.27	0.64
LOC (d)	13.9 ± 18.0	26.5 ± 35.5	0.004*
PTA (d)	38.7 ± 36.0	48.5 ± 64.0	0.17
Ambulation Index	4.55 ± 3.14	4.65 ± 3.18	0.82
Disability Status Scale	5.03 ± 2.51	5.12 ± 2.69	0.80
Arm Paresis (%)	32.7	40.6	0.21
Leg Paresis (%)	31.7	42.8	0.08

*$p < 0.05$.
GCS = Glasgow Coma Scale, LOC = length of coma, PTA = posttraumatic amnesia.

(McNamer tests) of time showed that all neurological impairment measures decreased (improved) between baseline and 6 months except for tremor ($p = 0.22$ for tremor, $p < 0.05$ for all others); only arm and leg strength continued to show improvement between 6 and 12 months ($p < 0.05$ for both arm and leg, $p > 0.05$ for all others), and no individual measures improved between 12 months and 2 years ($p > 0.05$ for all). At each time point, ataxia was more frequent than frank weakness, tandem gait was the most frequent abnormality, and involuntary movements were uncommon.

DISCUSSION

Several recently published investigations have examined how early residual neuromotor impairment may influence functional outcome after severe TBI. Englander and colleagues found that subjects who had motor strength <3/5 on MMT and moderate to severe incoordination at rehabilitation admission were more likely to need physical assistance for mobility and self-care both at rehabilitation discharge and 1-year postinjury [4]. Duong and colleagues subsequently replicated this finding in a larger sample [5]. Several investigators have reported that early balance impairment predicts worse outcome at rehabilitation discharge [27–29]. Early residual motor impairment may also be associated with poorer vocational outcome after severe TBI. This association is inferred from Cifu and colleagues' finding that physical function during rehabilitation as measured by FIM is associated with employment at 1-year postinjury [30].

Despite this evidence that early TBI-related neuromotor impairment is a prognostic indicator, the existing literature does not well describe its temporal evolution. In the present study, we tracked neurological examination longitudinally and showed a general pattern of neuromotor recovery that slowed after 6-month follow-up. Residual long-term neuromotor impairment detectable on routine neurological examination was quite common in this cohort with severe TBI. More than one-third of subjects

Table 3.
Longitudinal limb measures (n = 102). All values expressed as percent of participants.

Measure	Assessment Time Point			
	Rehab	6 Month	1 Year	2 Year
Arm Strength				
Normal	67.3	82.4	90.2	87.1
Paresis	27.7	16.7	9.8	12.9
Not Testable	5.0	1.0	0	0
Leg Strength				
Normal	68.3	81.2	89.1	91.1
Paresis	24.8	16.8	10.9	8.9
Not Testable	6.9	2.0	0	0
FNF & HS				
Normal	52.2	82.8	87.1	86.5
Ataxic	32.2	15.2	11.9	13.5
Not Testable	15.6	2.0	1.0	0

FNF = finger-nose-finger test, HS = heel-shin test, Rehab = during inpatient rehabilitation.

Table 4.
Longitudinal gait and posture measures (n = 102). All values expressed as percent of participants.

Measure	Assessment Time Point			
	Rehab	6 Month	1 Year	2 Year
Romberg Test				
Normal	42.6	90.0	84.8	88.0
Abnormal	17.8	8.0	11.1	8.0
Not Testable	39.6	2.0	4.0	4.0
Tandem Gait				
Normal	26.7	67.0	69.3	71.3
Loss of Balance	26.7	27.0	25.7	24.8
Not Testable	46.5	6.0	5.0	4.0
Hemiparetic Gait				
Normal	46.5	83.0	85.1	88.1
Circumduction	12.9	14.0	11.9	10.9
Not Testable	40.6	3.0	3.0	1.0

Rehab = during inpatient rehabilitation.

Table 5.
Longitudinal involuntary movement measures (n = 102). All values expressed as percent of participants.

Measure	Assessment Time Point			
	Rehab	6 Month	1 Year	2 Year
Tremor				
Normal	94.1	99.0	99.0	97.1
Present	3.0	1.0	1.0	2.9
Not Testable	3.0	0	0	0
Choreic/Athetoid				
Normal	96.0	99.0	99.0	100
Present	1.0	1.0	1.0	0
Not Testable	3.0	0	0	0
Dystonic				
Normal	95.0	99.0	99.0	99.0
Present	2.0	1.0	1.0	1.0
Not Testable	3.0	0	0	0

Rehab = during inpatient rehabilitation.

continued to have at least one neuromotor examination abnormality 2 years after inpatient rehabilitation. The physical examination seemingly adds valuable information to the disability assessment in these cases. These still "abnormal" patients, even if they have no mobility symptoms or gross problems, probably do have motor underperformance relative to preinjury status, especially when high-level mobility skills are considered. And we must also consider that those normalizing within 2 years may still have postural stability or fine motor deficits detectable either qualitatively or quantitatively.

Our results also support the usefulness of assessing postural stability via the tandem gait. At each time point, tandem gait was the most frequent abnormality among the tested neuromotor variables. More than a quarter of the cohort continued to test abnormal 2 years after inpatient rehabilitation. Thus, residual postacute balance impairment after TBI, hitherto primarily reported via sophisticated testing, also can often be documented on routine physical examination. Of note, prior evidence suggests that vestibular dysfunction is an important contributor to postural instability after TBI [16,31–33]. Guskiewicz postulated that two possible mechanisms were responsible for vestibular dysfunction following TBI, peripheral (such as benign paroxysmal positional vertigo and perilymph fistula) versus central [34]. Basford et al. proposed that impairments in the vestibular system may result from tethering of the vestibular nerve as it passes through the internal acoustic meatus that is caused by shearing and the acceleration-deceleration forces often sustained during TBI [16].

Beyond their importance for assessment and identification, these results have implications for rehabilitation programs. Independence in basic mobility should not necessarily be the final stage of physical therapy. The treatment team should consider the effect that any residual neuromotor impairments may have on high-level physical activities and implement appropriate treatment strategies.

Unfortunately, research data to guide specific rehabilitation interventions are not available. The current study was uncontrolled regarding specific balance interventions and consequently not designed to assess their efficacy. Our literature search found no controlled studies of postacute postural instability treatments after TBI. But given current beliefs on the nature of balance impairment after TBI, interventions targeting the vestibular system appear especially appropriate. Brown and colleagues recently demonstrated the efficacy of physical therapy for central vestibular dysfunction in a case series that included five posttraumatic subjects [35]. Their customized program included vestibular adaptation exercises, substitution exercises for lost vestibular function, education in use of assistive devices and safety awareness techniques to avoid falls, and one or more of the following: balance and gait training, general strengthening and flexibility exercises, and use of varied senses to aid in maintaining balance. Regardless of the intervention chosen, progress should be monitored with quantitative outcome measures.

The major limitation of this observational study was that sample selection bias may limit generalization of the results. Recruitment originated in inpatient rehabilitation units, so results should not be generalized to patients who do not get selected for inpatient rehabilitation. This limitation has also been noted in studies of Traumatic Brain Injury Model Systems (TBIMS) cohorts. Unlike most TBIMS analyses, we further narrowed inclusion to only those with severe TBI by PTA criteria. Our rationale was that patients with moderate TBI are known to have more favorable outcomes and findings would be diluted by their inclusion. Our final sample was biased by more missed follow-ups among those with worse initial injury severity. Thus, the impairment rates reported here are likely underestimates for our entire eligible sample of patients with severe TBI. Another limitation was that treatment interventions were uncontrolled.

Nevertheless, these results add to the mounting literature on persisting neuromotor deficits after severe TBI. Physical examination should be included in any comprehensive evaluation of patients with severe TBI, even postacutely. Clinicians should routinely assess these patients for postural instability, particularly when decisions are needed about high-level physical activities. The degree to which specific therapeutic interventions might improve these deficits and/or related functional deficits warrants future investigation. These results should also serve as a useful benchmark owing to the shortage of published neuromotor information after severe TBI.

CONCLUSIONS

This study provides longitudinal neuromotor impairment data to complement the severe TBI outcome literature that has tended to focus on psychosocial, cognitive, and subjective outcomes. The cohort displayed a general pattern of neuromotor improvement over time that leveled off by the 12-month follow-up; more than a third had persistent impairment at 2 years. Persistence of tandem gait impairment was particularly common, suggesting that patients with severe TBI should receive postural stability assessment before returning to certain complex physical activities, such as working at heights.

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REFERENCES

1. Swaine BR, Sullivan SJ. Longitudinal profile of early motor recovery following severe traumatic brain injury. Brain Inj. 1996;10(5):347–66. [PMID: 8735665]
2. Bode RK, Heinemann AW. Course of functional improvement after stroke, spinal cord injury, and traumatic brain injury. Arch Phys Med Rehabil. 2002;83(1):100–106. [PMID: 11782839]
3. Cook L, Smith DS, Truman G. Using Functional Independence Measure profiles as an index of outcome in the rehabilitation of brain-injured patients. Arch Phys Med Rehabil. 1994;75(4):390–93. [PMID: 8172496]
4. Englander JS, Cifu DX, Wright J. The impact of acute complications, fractures and motor deficits on functional outcomes and length of stay after traumatic brain injury. J Head Trauma Rehabil. 1996;11(5):15–26.

5. Duong TT, Englander J, Wright J, Cifu DX, Greenwald BD, Brown AW. Relationship between strength, balance, and swallowing deficits and outcome after traumatic brain injury: A multicenter analysis. Arch Phys Med Rehabil. 2004;85(8):1291–97. [PMID: 15295755]
6. Williams G, Robertson V, Greenwood K. Measuring high-level mobility after traumatic brain injury. Am J Phys Med Rehabil. 2004;83(12):910–20. [PMID: 15624570]
7. Forer S. Functional assessment instruments in medical rehabilitation. J Org Rehabil Eval. 1982;2:29–41.
8. Granger CV, Hamilton BB, Keith RA, Zielesny M, Sherwin FS. Advances in functional assessment for medical rehabilitation. Top Geriatr Rehabil. 1986;1:59–74.
9. Ponsford JL, Olver JH, Curran C. A profile of outcome: 2 years after traumatic brain injury. Brain Inj. 1995;9(1):1–10. [PMID: 7874089]
10. Dombovy ML, Olek AC. Recovery and rehabilitation following traumatic brain injury. Brain Inj. 1997;11(5):305–18. [PMID: 9146836]
11. Katz DI, White DK, Alexander MP, Klein RB. Recovery of ambulation after traumatic brain injury. Arch Phys Med Rehabil. 2004;85(6):865–69. [PMID: 15179637]
12. Hillier SL, Sharpe MH, Metzer J. Outcomes 5 years post-traumatic brain injury (with further reference to neurophysical impairment and disability). Brain Inj. 1997;11(9):661–75. [PMID: 9376834]
13. Lehmann JF, Boswell S, Price R, Burleigh A, DeLateur BJ, Jaffe KM, Hertling D. Quantitative evaluation of sway as an indicator of functional balance in post-traumatic brain injury. Arch Phys Med Rehabil. 1990;71(12):955–62. [PMID: 2241541]
14. Newton RA. Balance abilities in individuals with moderate and severe traumatic brain injury. Brain Inj. 1995;9(5):445–51. [PMID: 7550216]
15. Wöber C, Oder W, Kollegger H, Prayer L, Baumgartner C, Wöber-Bingöl C, Wimberger D, Binder H, Deecke L. Posturographic measurement of body sway in survivors of severe closed head injury. Arch Phys Med Rehabil. 1993; 74(11):1151–56. [PMID: 8239952]
16. Basford JR, Chou LS, Kaufman KR, Brey RH, Walker A, Malec JF, Moessner AM, Brown AW. An assessment of gait and balance deficits after traumatic brain injury. Arch Phys Med Rehabil. 2003;84(3):343–49. [PMID: 12638101]
17. Haaland KY, Temkin N, Randahl G, Dikmen S. Recovery of simple motor skills after head injury. J Clin Exp Neuropsychol. 1994;16(3):448–56. [PMID: 7929712]
18. Chaplin D, Deitz J, Jaffe KM. Motor performance in children after traumatic brain injury. Arch Phys Med Rehabil. 1993;74(2):161–64. [PMID: 8431100]
19. Kuhtz-Buschbeck JP, Hoppe B, Gölge M, Dreesmann M, Damm-Stünitz U, Ritz A. Sensorimotor recovery in children after traumatic brain injury: Analyses of gait, gross motor, and fine motor skills. Dev Med Child Neurol. 2003; 45(12):821–28. [PMID: 14667074]
20. Gray C, Cantagallo A, Della Sala S, Basaglia N. Bradykinesia and bradyphrenia revisited: Patterns of subclinical deficit in motor speed and cognitive functioning in head-injured patients with good recovery. Brain Inj. 1998;12(5): 429–41. [PMID: 9591145]
21. Incoccia C, Formisano R, Muscato P, Reali G, Zoccolotti P. Reaction and movement times in individuals with chronic traumatic brain injury with good motor recovery. Cortex. 2004;40(1):111–15. [PMID: 15070006]
22. Di Russo F, Incoccia C, Formisano R, Sabatini U, Zoccolotti P. Abnormal motor preparation in severe traumatic brain injury with good recovery. J Neurotrauma. 2005; 22(2):297–312. [PMID: 15716635]
23. Wiese H, Stude P, Nebel K, Osenberg D, Völzke V, Ischebeck W, Stolke D, Diener HC, Keidel M. Impaired movement-related potentials in acute frontal traumatic brain injury. Clin Neurophysiol. 2004;115(2):289–98. [PMID: 14744568]
24. Commission for Accreditation of Rehabilitation Facilities. Standards manual and interpretive guidelines for medical rehabilitation. Tucson (AZ): Commission for Accreditation of Rehabilitation Facilities; 2002.
25. Salazar AM. Defense and Veterans Head Injury Program. Brain Inj Source. 1999;3:8–10.
26. Salazar AM, Zitnay GA, Warden DL, Schwab KA. Defense and Veterans Head Injury Program: Background and overview. J Head Trauma Rehabil. 2000;15(5):1081–91. [PMID: 10970929]
27. Black K, Zafonte R, Millis S, Desantis N, Harrison-Felix C, Wood D, Mann N. Sitting balance following brain injury: Does it predict outcome? Brain Inj. 2000;14(2): 141–52. [PMID: 10695570]
28. Juneja G, Czyrny JJ, Linn RT. Admission balance and outcomes of patients admitted for acute inpatient rehabilitation. Am J Phys Med Rehabil. 1998;77(5):388–93. [PMID: 9798829]
29. Wade LD, Canning CG, Fowler V, Felmingham KL, Baguley IJ. Changes in postural sway and performance of functional tasks during rehabilitation after traumatic brain injury. Arch Phys Med Rehabil. 1997;78(10):1107–11. [PMID: 9339161]
30. Cifu DX, Keyser-Marcus L, Lopez E, Wehman P, Kreutzer JS, Englander J, High W. Acute predictors of successful return to work 1 year after traumatic brain injury: A multicenter analysis. Arch Phys Med Rehabil. 1997;78(2):125–31. [PMID: 9041891]
31. Guerts AC, Ribbers GM, Knoop JA, Van Limbeek J. Identification of static and dynamic postural instability following traumatic brain injury. Arch Phys Med Rehabil. 1996; 77(7):639–44. [PMID: 8669988]

32. Guskiewicz KM. Postural stability assessment following concussion: One piece of the puzzle. Clin J Sport Med. 2001;11(3):182–89. [PMID: 11495323]
33. Radfar-Baublitz LS, Pickett TC, McDonald SM, Walker WC. Objectively assessing balance deficits after TBI: The role of computerized posturography. Am J Phys Med Rehabil. 2006;85(3):256.
34. Guskiewicz KM. Assessment of postural stability following sport-related concussion. Curr Sports Med Rep. 2003; 2(1):24–30. [PMID: 12831673]
35. Brown KE, Whitney SL, Marchetti GF, Wrisley DM, Furman JM. Physical therapy for central vestibular dysfunction. Arch Phys Med Rehabil. 2006;87(1):76–81. [PMID: 16401442]

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