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Minimizing the effect of TBI-related physical sequelae on vocational return

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Abstract—This article evaluated the common physical sequelae that affect return to work (RTW) after traumatic brain injury (TBI). We performed a Medline search and evaluation of current TBI rehabilitation texts. The information presented is a combination of published literature and clinical guidelines. The limitations faced by many patients with TBI can best be overcome through clever job search, job redesign, and community linkages with business and industry that are willing to partner in helping the patient with TBI regain employment. The physician plays a key role in communicating suggestions to the vocational specialist. The comorbidities described represent challenges to successful RTW. These problems are recurrent, long-term, and clearly affect job procurement, nature of job, level of required support, and likelihood of job retention. Conversely, these challenges should not be viewed as impenetrable obstacles. With appropriate supports such as compensatory strategies, job coaching, assistive technology, medical management, and job restructuring, successful RTW is viable option. Physicians must focus on employment outcomes in real jobs and not settle for volunteer work, sheltered work, or assessment and planning. Individuals should be placed in real work for real pay. Through close collaboration between the survivor of TBI, the physician, the vocational specialist, and community resources, successful employment for survivors of TBI is possible and must be prescribed a high value.

Key words: community reintegration, employment, functional restoration, rehabilitation, return to work, school reintegration, TBI, traumatic brain injury, vocational return, work reintegration.

INTRODUCTION

Traumatic brain injury (TBI) is a leading cause of worldwide disability, with one estimate showing a loss of 56 billion dollars in economic productivity [1]. Its economic impact is large because it has a predilection to affect young people on the verge of their most productive years. As understanding of TBI has increased, so has the focus on vocational return following injury [2]. Within the continuum of functional restoration, vocational return is regarded as the most durable sign of successful community return [3]. Employment becomes a challenging, if not unlikely, feat to attain because of a myriad of cognitive, physical, and psychosocial problems. Meaningful productive employment enhances self-esteem, financial status,

Abbreviations: CPT = computerized posturography testing, CRP = community rehabilitation program, GPB = grooved pegboard (test), HA = headache, HO = heterotopic ossification, NSAID = nonsteroidal anti-inflammatory drug, PTHA = post-traumatic HA, RTW = return to work, TBI = traumatic brain injury, UFOV = Useful Field of View (test).

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and one's overall quality of life, yet for many individuals with TBI, return to productive employment seems unlikely [4–5]. Furthermore, vocational return is increasingly viewed as a means of enhancing cognitive, behavioral, and physical recovery after the acute rehabilitation phase. Because of this and other factors, vocational rehabilitation strategies to assist survivors of TBI are of utmost importance.

The TBI literature has established reliable indicators of functional recovery, such as functional mobility, self-care, and community return [6]. Prognostication of vocational return is much more complex given the impact of personality characteristics and environmental, economic, and cultural factors. Several factors have been identified that add predictive value. These include injury severity, age at time of injury, number of neuropsychological deficits, level of physical disability, premorbid employment, and educational level [3,7–10].

A brief review of return to work (RTW) literature reveals contradicting findings regarding the effect of injury severity on RTW [11–13]. RTW rates have been tied to injury severity [9,13–14], although not all investigators have found a relationship between injury severity and RTW [12,15–16]. Despite the varying results, it is important to note that some individuals who sustained very severe injuries are able to return to employment relatively unhindered [7,13].

The specific types of cognitive, behavioral, physical, and psychosocial impairments associated with TBI of an individual may be more predictive of his or her RTW than the measure of injury severity [17]. For example, significant physical disability, psychosocial impairment, memory/reasoning deficits, and history of alcohol abuse have all been linked to poor work outcomes. An in-depth literature review by Chesnut et al. suggested that multiple issues besides cognitive and psychological factors, such as physical sequelae, impeded work [18]. Indeed, vocational specialists have known for some time that the array of different physical sequelae can be a problem for job retention [2,19].

Importantly, cognitive and behavioral sequelae after TBI significantly affect both RTW and employment stability. Executive function deficits are quite common with all TBI given the predisposition to frontal lobe injury. There is a strong correlation between self-awareness and favorable vocational outcome [3]. Furthermore, issues with depression, anger, and impaired social pragmatics negatively affect successful community reentry [6]. Given the complexity of the deficits commonly seen post-TBI and the myriad of psychosocial confounders that accompany these injuries, a holistic approach to vocational return is paramount. A full review of the cognitive and behavioral barriers is beyond the scope of this article and has been addressed in detail in other sources [2–3,6–7].

There is a critical need to examine the multiple physical and sensory sequelae associated with TBI. We were unable to locate a succinct review in the literature detailing these deficits and their effect on RTW. This article reviews the literature on these sequelae. We discuss how these sequelae affect RTW. In turn, suggestions based upon clinical practice are offered to guide the clinician's treatment approach. It is important to note that the subset of physical sequelae is related to the degree of TBI severity. For instance, seizures are much more common in moderate to severe than in mild TBI. These clinical groupings are—

- Mild TBI:
  - Headaches.
  - Vision deficits.
  - Pain syndromes.
  - Dizziness.
  - Postural instability.
- Moderate to severe TBI:
  - Heterotopic ossification.
  - Hypertonicity.
  - Seizures.
  - Postural instability.
  - Fine motor deficits.
  - Visual deficits.
  - Insomnia.
  - Fatigue.

Hopefully, this article will improve TBI survivors' likelihood of successful vocational return.

SEIZURES

Posttraumatic seizures (or epilepsy) occurring after the first week following TBI affect 14 to 53 percent of survivors with moderate to severe TBI [20]. These seizures are extremely rare in individuals with mild TBI (or concussion). Although injury severity is predictive of seizures, the routine prophylaxis of patients with severe TBI has not been shown to be effective in reducing their late seizure risk [21]. In patients who will develop posttraumatic

seizures, most have initial onset within 1 year postinjury and about 76 percent within 2 years [22–23]. A multicenter study found that among various TBI-related imaging abnormalities, seizures most often followed biparietal contusion, dural penetration with metal fragments, multiple intracranial surgeries, subdural hematoma with evacuation, midline shift <5 mm, and multiple or bilateral cortical contusions [24]. Other factors have also been identified, including chronic alcoholism, age, intracranial hemorrhage, length of posttraumatic amnesia, depressed skull fracture, lesion location, and early glucocorticoid administration [24–29]. Prophylaxis for late seizures longer than the universally recognized standard of 7 days postinjury in response to a perceived elevated risk, such as the above mentioned factors, warrants further investigation and is not at present recommended.

The development of late seizures warrants treatment. Complex partial seizures (acute altered cognition with focal motor or sensory abnormalities) are the most common presentation, followed by simple partial seizures (focal motor or sensory abnormalities with no change in cognition) and then generalized or tonic-clonic seizures. Absence seizures (brief periods of altered cognition) are rare following TBI. The use of carbamazepine for the management of partial seizures and valproate for generalized seizures is advocated [20,30]. Phenytoin and carbamazepine are sedating and may be detrimental to cognition and long-term neurological recovery [31]. Newer agents, while often less sedating and easier to use, are less studied specifically for TBI-related late seizures [31].

The presence of posttraumatic epilepsy after TBI may affect the ability of individuals to return to and maintain work or schooling [32–34]. This effect is rarely due to the actual seizure activity, which is typically managed with medications, but more often due to the side effects (somnolence, mild decline in alertness and attention) of the medications, the limitations prompted by the risk of seizures (e.g., unable to drive for 6 months after a seizure, unable to work at heights or with heavy machinery until seizure-free for 6–12 months), and the negative bias brought on by a seizure disorder. Efforts at work and school reintegration must be closely integrated with the clinicians treating these clients so that medication adjustments can be judiciously made, specific limitations can be understood and negotiated, and the entire team (patient, physician, vocational specialist) can work from the same knowledge base.

SPASTICITY

Spasticity collectively refers to a host of motor overactivity syndromes stemming from upper motor neuron damage [35]. While focusing on the impact of hypertonicity, the clinician must recognize the detrimental functional impact of the accompanying symptoms of upper motor neuron syndromes: weakness and the loss of dexterity. In combination, these symptoms impair muscular control and affect functional independence [36–40]. Spasticity can have positive, but more frequently negative, effects on patient function [41–42].

The unique presentations of spasticity and its functional impact warrant individualized treatment plans and serial clinical monitoring. Individual assessment is important, with particular attention paid to the spasticity distribution (upper vs lower, unilateral vs bilateral, global vs focal). After an injury to the cerebral motor strip, the upper limbs typically respond in a flexion pattern. Lower limbs are more often in extension, though this pattern is more variable [43]. Treatment of spasticity should be multimodal and include therapeutic interventions and medications. A detailed assessment of the distribution (focal vs global), severity, and functional impact is crucial [44].

For global spasticity, a variety of oral medications are employed, including dantrolene, tizanidine, diazepam, and baclofen [45–46]. Unfortunately, all may have the common adverse effect of sedation, which can be quite problematic in the work setting. Dantrolene has the least sedative effect given its primary action at the peripheral muscles, so it is widely considered the first-line agent [6]. Intrathecal delivery of baclofen offers a higher concentration delivered to the spinal cord with lower circulating drug levels, thereby minimizing systemic side effects. Multiple studies show that intrathecal baclofen is safe, effective, and well tolerated over time [47–50]. Injection techniques such as intramuscular phenol and botulinum toxin are effective in the treatment of focal spasticity. Botulinum toxin seems particularly effective in the upper limbs [51–52], especially when complemented with ongoing therapies to maximize functional improvements.

Employment can be therapeutic for persons with spasticity after TBI because of joint motion associated with body movements and by facilitating neural recovery and latent abilities. However, hypertonicity represents a significant barrier to successful vocational return, particularly when it involves the upper limbs. Reach, grasp, and fine motor control form the basis of many work settings.

Furthermore, the other "negative" consequences (loss of dexterity and weakness) have additive detrimental effects. In combination, these decrease the radius of the workspace and have broad-ranging effects on efficiency and productivity. The lower-limb pattern can be somewhat beneficial and provide a stable base of support for standing. The increased energy expenditures with gait, though, must be taken into account.

A patient with spasticity should have a carefully tailored work space that takes into account his or her specific functional deficits. For those with significant upper-limb hypertonicity, the workspace area should be shortened to accommodate range of motion restrictions. Adaptive orthotics can assist in overcoming the deficits of grasp, discoordination, and hypertonicity. Particular emphasis should be placed upon the distribution because one side is commonly more affected. In this situation, tasks should be targeted to the preserved limb. Depending upon the pattern of lower-limb tone, appropriate seating modifications should be made. Further, it is important that the floor space be flat and free of impediments to allow for safe ambulation.

HETEROTOPIC OSSIFICATION

Heterotopic ossification (HO), or abnormal periarticular bone formation, occurs in 11 to 77 percent of individuals after TBI [53–57]. While the underlying pathophysiology of HO is unknown, a neuroendocrine alteration may be causative [58]. Risk factors for HO include prolonged coma (>2 months), ventilator support, spasticity, and fractures [59]. The symptoms of HO include pain, decreased range of motion, warmth, palpable mass, and tenderness [60]. The hips are overwhelmingly most affected, followed by the shoulders, knees, and elbows. Triple-phase bone scan allows early detection of HO, whereas plain X-rays are useful for diagnosis only after calcification [61]. Serum alkaline phosphate may be used to monitor maturity of lesions but has limited utility in diagnosis [61]. Complications include decreased range of motion, frank joint ankylosis, pain, and nerve compression. Not surprisingly, HO has been linked to poorer functional outcomes [53,62].

Treatment of HO is geared toward the complication with the most functional impact: restriction of joint range. At the minimum, daily full range of motion exercise to prevent joint contractures is indicated [63]. The effects of pharmacologic treatment and prophylaxis of HO, including bisphosphates, nonsteroidal anti-inflammatory drugs (NSAIDs), and radiation, are controversial [57,64–66]. Surgical excision is employed when lesions are causing a barrier to function, though recurrence may occur regardless of its timing (early vs late excision) [58].

In the work setting, the joint range restrictions must be accommodated. As with spasticity, accommodation includes shrinking the space to allow for the decreased range of motion of the shoulders and elbows. The workspace should also be clear of clutter that may affect safe ambulation.

POSTURAL INSTABILITY (BALANCE PROBLEMS)

Balance problems are frequent after TBI. Early balance deficits after severe TBI can indicate poorer long-term functional recovery [67–68] and can persist chronically. One study of severe TBI showed 34 percent of survivors with severe TBI reported balance impairments at 5 years [69]. In another study, 29 percent tested abnormal on tandem gait at 2 years [70]. TBI-related balance impairments are not exclusive to severe injuries. Investigations using computerized posturography testing (CPT) show that balance deficits are present acutely after mild TBI (e.g., concussion) [71–72]. Published data on balance deficits is lacking within the late postconcussive syndrome population.

Objective methods for quantifying balance impairment after TBI (CPT) can aid both the tracking of mobility recovery and the formulation of recommendations for disability, job restrictions, and leisure activities. CPT also provides data about the pattern of postural instability, which may be useful in guiding rehabilitation interventions [73]. More traditional quantitative balance tests include variations of the Romberg test [74], the test of sway [75], and the Berg Balance Scale [76].

Balance problems are important because of their potential impact on both basic (transfers, walking, etc.) and high-level (jumping, climbing, running, etc.) mobility skills. Even when grossly "independent," most individuals need some degree of high-level mobility skills for a complete and safe return to leisure and/or vocational activities. Safety risks from balance impairment are dramatically worsened when cognitive impairment coexists, which is common after severe TBI. Subsequently, individuals do not recognize physical limitations and fail to incorporate constructive strategies to safely compensate for their balance deficit.

Evaluation of patients with imbalance after TBI should include assessment for peripheral neuropathy, postural hypotension, limb ataxia, visual impairment, and dizziness symptoms. Patients with moderate or severe TBI should undergo a formal balance assessment, ideally with CPT, before returning to select work duties, military training, or combat. From a vocational rehabilitation standpoint, working at heights, on platforms, or around moving or sharp machinery is contraindicated in the presence of significant postural instability.

Despite the frequency of postural instability after TBI, scant published research addresses the effectiveness of interventions. Traditional approaches include physical therapy for specific exercises to promote postural stability, such as joint approximation, alternating isometrics, rhythmic stabilization, and practice of controlled mobility activities and functional movement transitions [77]. Biofeedback instruments can be added, such as a force platform or CPT. Individuals with falls or near falls while ambulating should also use a gait aid, especially on uneven terrain.

DIZZINESS

Posttraumatic dizziness affects 20 to 65 percent of patients with TBI [67,78–79] and remains disabling for many months [8,80–82]. Dizziness is among the top five postconcussive symptoms distinguishing patients with mild TBI from healthy controls [79,83–84] and appears to have important vocational implications. One study showed a 46 percent failure of RTW 5 years after mild to moderate TBI for patients with posttraumatic dizziness. A more recent study on RTW at 6 months postinjury (mild and moderate) showed 34 percent of individuals with dizziness were gainfully employed compared with nearly 75 percent of subjects without dizziness. Importantly, dizziness was also associated with increased psychological distress (including anxiety and depression), which may further hinder RTW efforts [85].

Studies of patients with dizziness after mild to moderate TBI show a rate of vestibular abnormalities of between 32 and 65 percent [86–87]. The large number of patients with dizziness who do not have objective findings of neural damage reinforces the connection with psychological factors [85–86]. Medication-based treatments, such as the anxiolytic agents buspirone or trazodone, may be aimed at these psychological factors and can have positive effects on symptoms. Commonly prescribed “sedating” medications, such as meclizine, are controversial in that their mechanism of decreasing the sensation of causative stimuli can theoretically slow recovery. Focused vestibular rehabilitation programs with purposeful exposure, habituation, and coping strategies have proven efficacy [87]. Benign paroxysmal positional vertigo is a condition that is caused by movement of otolithic debris or granules within the semicircular canals. Trauma can lead to the release of such debris. Specific therapies (Dix-Hallpike maneuver and Liberatory technique) can be used for both diagnosis and therapy [68].

Even in the face of disabling dizziness, progressive return to activity, including driving and work, is key to both reducing symptoms and preventing habituation to abnormal movement patterns (e.g., remaining lying down for activities). This “forced” activity is a necessary component of the rehabilitation efforts for vertigo. Intensive vocational rehabilitation services are indicated and invaluable in the early rehabilitation course [11,88]. Work site modifications and job accommodations are initially an integral aspect of vocational rehabilitation, but can usually be tapered away as the symptoms and tolerance improve. Gradated exposure to the necessary aspects of the job (e.g., repeated movements, visual distractions) is both therapeutic and facilitates RTW efforts. Medications, while occasionally helpful early in the treatment program, are limited in the work setting because of cognitive depression.

FINE MOTOR DEFICITS

Gross motor deficits do not occur after uncomplicated mild TBI and seldom persist chronically after severe TBI. One report showed hemiparesis in 15 percent and quadraparesis in 6 percent 5 years after severe TBI [69]. A more recent report showed arm weakness in 13 percent and leg weakness in 9 percent 2 years after inpatient rehabilitation [70].

Motor integrative function and fine motor skills are more common persisting deficits after TBI and often have occult presentations. Various investigations have shown impairment in finger tapping 1 year after mild to moderate TBI [89]; impairment in integrative gross motor and speeded fine motor skills 16 months after severe TBI [90]; deficits in fine motor skills, speed, and coordination 8 months after TBI [91]; subclinical bradykinesia with impaired complex and simple reaction time 1 year after TBI [92]; residual motor programming deficits in severe chronic

TBI despite clinically good motor recovery [93]; and selective deficit in motor preparation in severe TBI with good clinical recovery [94]. The grooved pegboard (GPB) test is a fine motor test included in many standard comprehensive neuropsychological batteries after TBI. In a large severe-TBI cohort, GPB was more commonly impaired than any of the predominantly cognitive performance tests at 5 years post-TBI, with 75 percent scoring under the 16th percentile for age-matched norms [95].

Fine motor impairment after TBI has important functional implications. Slower speed on the GPB is associated with poorer vocational outcome [96] and poorer Glasgow Outcome Scale scores [97]. From a vocational reentry standpoint, fine motor speed and dexterity should be formally assessed with appropriate modifications or restrictions for any tasks requiring high levels of fine motor functioning. Ideally, the tasks themselves should be directly assessed for speed and performance errors. Patients with deficits may also benefit from specific rehabilitation interventions. A recent evidence-based review article found "moderate" evidence to support the use of functional fine motor control retraining to improve motor coordination after acquired brain injury [98].

VISION

Reports of visual changes (usually worsened visual acuity) occur in up to 20 to 40 percent of individuals with TBI [69,99]. True deficits in vision are difficult to differentiate from visual perceptual deficits (the brain's interpretation of what the eyes see) [99]. True field cuts (e.g., homonymous hemianopsia, quadrantonopsia, tunnel vision) are more common in ischemic damage to the brain but may also be seen with traumatic injuries [100–101]. Cortical blindness (Anton's syndrome) may be seen following occipital lobe injury but is uncommon [102]. The Useful Field of View (UFOV) test is a measure of the functional or useful range of peripheral vision under cognitive load conditions [103]. As cognitive load is increased by elevating task complexity, the functional range of peripheral vision (i.e., the degree of peripheral vision from which information is processed) becomes restricted [104]. Thus, the functional extent of peripheral vision under complex real-world conditions, such as detecting stimuli in cluttered backgrounds, is not always equivalent to the maximum extent of peripheral vision that can be measured with clinical perimetry techniques [105]. Individuals with TBI and strokes and older adults often have impaired UFOV [105–107]. Survivors with TBI exhibited decreased information processing time and mild impairment at all eccentricities, with the greatest impairment on the selective attention subtest of the UFOV. These findings suggest that survivors with TBI may need more time to locate stimuli in cluttered backgrounds and are less accurate than people without brain impairment. These results are consistent with previous reports of higher errors and task completion times on cancellation tests involving visual search [106].

Rehabilitation efforts for visual deficits focus on enriching the environments to increase appropriate stimuli to the areas of visual decline. This strategy should be applied both in the structured therapy setting and in the general treatment milieu. While a variety of specific visual rehabilitation programs have been described [100–101], there is no universal agreement on the efficacy of these protocols. In addition to enhancing visual stimuli and specialized interventions, rehabilitation efforts must also focus on enhancing the cognitive functioning. The use of these strategies and devices allows for greater adaptation to deficits and greater use of other senses. Many of the tactics used for newly blind individuals (from any cause) are used in a modified way for individuals with TBI.

Studies have demonstrated poorer overall functional outcomes with persistent visual deficits [69,106,108] and poorer RTW with visual perceptual deficits after TBI [109]. Vocational efforts for individuals who have visual deficits that are of clinical significance are typically introduced once an individual has returned to the community. Transportation to the work site may present significant challenges and must be addressed early in the course. Unique aspects of "blind rehabilitation" that are relevant to the individual with TBI are the overlying cognitive, behavioral, and physical impairments commonly seen. Safety concerns must be emphasized in individuals with TBI and impaired sight because of the decreased sensory feedback and, not uncommonly, decreased safety judgment.

HEADACHES AND PAIN

Pain is commonplace after TBI of all severity levels. In a study of hospitalized patients with TBI, 71 percent required narcotic pain medication prescription upon discharge [110]. In an adult tertiary care pain clinic investigation, 58 percent of persons with mild and 52 percent with moderate to severe TBI reported chronic pain [111].

Chronic pain is usually defined as 6 contiguous months of pain and is often intertwined with psychopathology and environmental factors [112]. In its persistent form, chronic pain typically imposes severe emotional, physical, economic, and social stresses on the patient and family.

After major trauma, potential etiologies of pain are numerous. The anatomical source of pain in individuals after TBI may be intracranial or extracranial, depending on the pattern of trauma [113]. Proper detection and diagnosis of pain is paramount for achieving optimal rehabilitation potential after TBI. With proper evaluation, pain can often be classified into one of the following clinical types: musculoskeletal, vascular, visceral, or neural tissue origin.

Headache (HA) is the most frequent pain reported after TBI, with a prevalence range from 18 to 93 percent depending on study methodologies [114]. HA that commences within 14 days of consciousness following TBI is termed posttraumatic HA (PTHA) [115]. Some investigations show higher prevalence of PTHA in milder TBI [111,116–118], but this finding remains controversial given sample selection bias concerns and conflicting reports [119–120]. PTHA is usually self-limited but may become chronic. A longitudinal study of patients with moderate and severe TBI showed daily PTHA in 30 percent during inpatient rehabilitation, declining to 10 percent 6 months later [120]. Similar to chronic pain in the general population, the evolution from acute to chronic PTHA is linked to emotional distress [119–120]. Chronic PTHA that persists beyond 6 months after injury will usually become permanent and disabling [120–122].

There is a shortage of treatment trials in PTHA to offer standard of care guidance for clinicians [123]. Clinicians should, after excluding serious intracranial pathology, attempt to classify PTHA and use medical treatment accordingly. Migraine HA type, diagnosed in 26 percent of one PTHA sample [120], appears to be overrepresented compared with its distribution in the general population [124]. Migraine-specific HA medications should be trialed for migraine-type PTHA. Cervicogenic HA, originating from cervical spine or surrounding soft tissues, is common after TBI [114] and may respond to physical therapy and modalities. PTHA with neuropathic quality may respond to neuropathic pain medications or peripheral nerve blocks. Narcotic medications and other medications with central effects such as muscle relaxants should be avoided if possible. Tension and cervicogenic HA may respond to NSAIDs and/or acetaminophen. Concomitant emotional disorders such as depression and anxiety should be treated with appropriate pharmacological and behavioral treatments. Even absent emotional disorders, psychological evaluation and behavior therapy, including relaxation training or biofeedback, may be beneficial. Contributing factors should also be sought out, including sleep disturbances, excessive caffeine intake, and sources of emotional distress.

From a vocational rehabilitation standpoint, the role of emotional stress in PTHA is paramount. Patients should be monitored for the onset of delayed PTHA or exacerbation of preexisting PTHA during work reentry or after changes in work routine. Whenever PTHA or other chronic pain syndromes worsen in employed patients with TBI, they should be medically examined. Their work schedule and duties should as well be reevaluated. Some PTHA syndromes (migraine) may be episodically disabling and necessitate intermittent work absence. Others may be chronically disabling (chronic daily HA), causing total work disability if refractory to treatment.

INSOMNIA

Insomnia following TBI is a common condition that requires close attention. Its effects on overall function and quality of life are well documented [125–128]. Following a TBI, 30 to 70 percent of survivors report difficulties with sleep [125,128–136]. In two separate studies, nearly 30 percent of TBI survivors met Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, criteria for insomnia syndrome [125,132]. In comparison, this rate is three times greater than the general population [137–138]. The most common features of TBI-related insomnia are sleep initiation and maintenance problems [125,139]. While some literature suggests the volume of sleep complaints decreases over time [130–131,134], the data suggest that a significant proportion develop a chronic course [130,136,140].

The literature on risk factors associated with post-TBI insomnia is scant [125,128]. Older age, female gender, depression, HAs, and alcohol abuse were negative prognosticators in one study [129]. Others show a similar association with depression, as well as less severe injuries [129,132]. Conversely, those with successful vocational return had less difficulty sleeping [129]. Given the limited information available, more research is needed to better understand the impact of other factors, including pain [141], psychological distress, and fatigue, on sleep after TBI.

Treatment of post-TBI insomnia is multifaceted, including sleep hygiene education, cognitive-behavioral therapy, and pharmacology. Behavioral approaches are more time-consuming but have been shown to produce more effective long-term results than use of medications alone [142]. Medications that promote the initiation and maintenance of sleep fall into four categories: benzodiazepine sedative hypnotics (diazepam, lorazepam), nonbenzodiazepine sedative hypnotics (zolipidem, etc.), antidepressants (trazodone, mirtazapine), and neutraceuticals (melatonin). These medications have all been shown to promote healthy sleep in the short-term, but effects of long-term use are poorly documented and the side effect profiles may be more problematic in the population with TBI. The benzodiazepines have a prominent hangover effect manifested by cognitive suppression and psychomotor agitation, and chemical dependency may occur [143–145]. The antidepressants are better tolerated but may carry anticholinergic and cardiac risks [146–151]. The nonbenzodiazepine sedatives carry the lowest side effect profile while maintaining similar efficacy on sleep parameters [128,152–160]. Further studies in the population with TBI are warranted to determine the most efficacious agents.

When assessing patients with TBI, clinicians must continuously evaluate sleep patterns and daytime symptoms and pay close attention to daytime fatigue, HAs, and irritability. Insomnia leads to daytime fatigue, confusion, HAs, exacerbation of pain, and mood alterations [126–127]. These symptoms and side effects from pharmacological treatment compound the cognitive and behavioral changes associated with TBI. The impact of insomnia on work can be profound. Though challenging, reestablishing healthy and restorative sleep patterns after TBI is fundamental to the return of quality of life and vocational success.

FATIGUE

Fatigue is commonly observed and reported after TBI, regardless of severity. Following HAs and dizziness, it is the third most prevalent symptom of postconcussive syndrome [83,161–162]. Studies on fatigue in the general population have documented prevalence rates ranging from 7 to 45 percent [163]. The wide range of prevalence rates is likely attributable to differences in operationalizing a working definition of fatigue, as well as the variability of fatigue across different medical conditions and interventions. Some researchers have argued in favor of a multidimensional approach, such as defining fatigue in the dimensions of general, physical, and mental fatigue [163]. Typically, an assessment of fatigue is obtained via a detailed history [134], a structured interview [164], or questionnaires designed to measure broader conditions (e.g., Beck Depression Inventory) [165]. The most commonly recognized questionnaires in the literature are the Fatigue Impact Scale [164], the Fatigue Severity Scale [166], and the Barrow Neurologic Institute Fatigue Scale [167].

While fatigue is often clinically observed in brain-injured patients during the acute stages of recovery, there is a dearth of empirical studies on this topic. Fatigue has been shown to be a clinically relevant symptom up to 10 years postinjury [134,168–169], the Fatigue, both physical and mental, has been demonstrated to negatively affect functional performance [134,161,170–171] and return to vocational efforts [109,172]. Treatments focus on managing underlying physical (endocrine, infectious, cardiovascular) and psychological (depression, stress) factors and use of neurostimulant agents (e.g., methylphenidate) [83].

Vocational interventions need to address physical and cognitive conditioning to allow for successful employment. In addition, work accommodations should be made to account for decrease in functional abilities. As with many posttraumatic symptoms, a progressive program of reactivation of the body and the mind is both a specific treatment of the underlying symptom and a necessary vocational rehabilitation intervention. While scheduled rest breaks, adjusted work schedules, and job modifications may be needed initially to maintain the client in a vocational situation, the long-term goal is one of gradual weaning away of these adjustments. The partnership between the medical and rehabilitation team and the vocational specialist is vital to facilitate rapid feedback of the successes of interventions and the potential exacerbation of symptoms related to work.

CONCLUSIONS

The sequelae described in this article represent challenges to successful RTW. These problems are recurrent and long-term and clearly affect job procurement, nature of job, level of required support, and likelihood of job retention. Conversely, these challenges should not be viewed as impenetrable obstacles. With appropriate supports, such as compensatory strategies, job coaching, assistive technology, medical management, and job restructuring, successful RTW is a viable option. Most of the sequelae described

have solutions, not the least of which is searching for a more appropriate job. The astute medical professional will focus repeatedly on two questions:

- When will my patient be employed?
- What supports are needed for my patient to become employed?

The limitations faced by many patients with TBI can best be overcome through clever job search, job redesign, and community linkages with business and industry that are willing to partner in helping the patient with TBI regain employment. For example, the person who has periodic HAs may fair poorly in a stimulus-rich environment and flourish in a more controlled environment. The physician plays a key role in communicating suggestions to the vocational specialist.

Community rehabilitation programs (CRPs) provide supported employment services, frequently through agreements with state vocational rehabilitation agencies. The CRP staff members that provide employment services are called employment specialists. Well-trained employment specialists are skilled in a variety of activities necessary to identify, develop, implement, and monitor individualized employment supports.

Physicians must focus on employment outcomes in real jobs and not settle for volunteer work, sheltered work, or assessment and planning. Individuals should be placed in real work for real pay. Through close collaboration between the survivor of TBI, physician, vocational specialist, and community resources, successful employment for survivors with TBI is possible and must be assigned a high value.

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