Mild Traumatic Brain Injury

***Epidemiology***

In the United States alone, approximately 1.4 million incidents of traumatic brain injury are reported each year, with 75 to 80% percent being classified as mild.1,2 While risk factors for the general population include non-white ethnicity and male sex, a large percentage of TBI-related hospitalizations in *older* adults occurs in whites.3 The majority of mild traumatic brain injuries (MTBI) are the result of falls, motor vehicle accidents, and assualts.1,2 Falls are responsible for a significant proportion of MTBI in the older adult population, while motor vehicle accidents account for most MTBI in adolescents and young adult males.1 Alcohol or other substances may be involved in as many as 50% of these injuries.2 In general, the incidence of TBI peaks in the second and third decades of life and again after the seventh decade.2 While men are injured two to three times more often than women in younger age groups, both sexes are equally at risk after age 65.2 In addition, those over 65 are at an increased risk for repetitive MTBI due to their increased risk for subsequent falls after an initial fall.3 Lastly, the highest suicide rate for all age groups is found in adults aged 65 and older. This is significant because firearms are the most common method used, which places the older adult at risk for a penetrating TBI if they survive.3

***Definition of MTBI***

There is no consensus definition of MTBI.1 In general, MTBI has been described as “a state in which there is an alternation of minimal duration and severity or no change from the patient’s baseline neurological or mental status after an injury.”1 The Centers for Disease and Prevention have developed a more specific definition for MTBI: “The occurrence of injury to the head resulting from blunt trauma or acceleration or deceleration forces with 1 or more of the following conditions attributable to the head injury during the surveillance time period: (1) any period of observed or self-reported transient confusion, disorientation, or impaired consciousness; (2) any period of observed or self-reported dysfunction of memory (amnesia) around the time of injury; (3) observed signs of other neurological or neuropsychological dysfunction; and (4) any period of observed or self-reported loss of consciousness lasting 30 minutes or less.”1 When using the Glascow Coma Scale to grade brain injury, a MTBI is typically defined by a score of 13 to 15, however some believe that a score of 13 should be excluded from the mild category.1 In addition to the above definitions, *concussion* is another term that is used interchangeably with MTBI.1 This term is often used in the context of sports and is classified by 3 grades. Grade I has no associated loss of consciousness or amnesia, grade II has amnesia but no loss of consciousness, and grade III is associated with loss of consciousness.1

***APTA Guide to Physical Therapist Practice Patterns***

Depending upon the age of injury, the following practice patterns are most appropriate for the evaluation and treatment of an individual with traumatic brain injury: (1) *Pattern 5C:* Impaired Motor Function and Sensory Integrity Associated With Nonprogressive Disorders of the Central Nervous System - Congenital Origin or Acquired in Infancy or Childhood; (2) *Pattern 5D:* Impaired Motor Function and Sensory Integrity Associated With Nonprogressive Disorders of the Central Nervous System - Acquired in Adolescence or Adulthood.

***Pathology and Pathophysiology***

The pathophysiology of traumatic brain injury is complex and there are many aspects that are not well understood.4 Aside from the initial trauma, there are many secondary injury processes, such as axonal and vascular injuries, ischemia, cerebral edema, and hemorrhage in or around the brain tissue that determine morbidity and mortality.4 After the initial injury, a complex pathophysiological cascade of changes occur, many of which are harmful.4

 While brain injury can result from a direct traumatic force, the majority of injuries result from indirect forces that set the head, skull, and brain in motion.4 Linear acceleration and rotational head movements are examples of these forces and they occur frequently during falls and high speed motor vehicle accidents.4 It is thought that linear acceleration forces are responsible for superficial brain lesions, while rotational movements are responsible for deeper cerebral lesions and concussions.4 Due to the viscoelastic properties of brain tissue, the brain is more susceptible to inertial forces and pressure gradients than more solid organs.4 It has been found that the cerebral gray matter is most affected by linear forces, resulting in cortical contusions and hemorrhage, while cerebral white matter is most affected by rotational forces, resulting in diffuse axonal injury.4

 In addition to the above primary injury forces, secondary biomechanical effects greatly contribute to the eventual pathology.4 The primary concern in regards to secondary injury is an increase in intracranial volume and pressure, which can be caused by multiple factors, including brain tissue edema, hemorrhage, and hematomas.4 Another concern is dysfunction of the blood brain barrier (BBB). The BBB separates the brain from the circulatory system and protects the central nervous system from potentially harmful chemicals while regulating transport of essential molecules and maintaining a stable environment.4  The BBB is formed by highly specialized endothelial cells and astrocytes that tightly regulate the composition of brain fluids by selectively controlling access of blood-borne ions, nutrients, and polypeptides to the brain.4  Both traumatic and non-traumatic brain injuries often disrupt this regulation and are associated with a dysfunction of the BBB, which greatly affects the progression of the injury.4

Animal studies have suggested that there is increased permeability of the BBB after TBI, leading to an infiltration of reactive oxygen species (ROS), proinflammatory cytokines, vascular endothelial growth factor, and matrix metalloproteinases (MMPs).4  These factors have the potential to open the BBB even further, which allows molecules to accumulate in the interstitial fluid and results in cerebral edema.1  Cerebral edema is dangerous after TBI because it increases cerebral volume, which increases tissue pressure, elevates ICP, and leads to brain ischemia.  Another hormone, arginine vasopressin (AVP), which maintains body water and electrolyte homeostasis, also promotes the disruption of the BBB and contributes to the formation of cerebral edema.4

Lastly, disruption of the BBB contributes to neuroinflammation.  Within hours after brain injury, there is a surge in the production of proinflammatory cytokines, which promotes neuronal death and interferes with the survival signals that are produced by growth factors.4  These cytokines lead to further dysfunction of the BBB, which is central to the progression of neuroinflammation after injury, as well as cerebral edema.4

***Effects on Systems***

Traumatic brain injury has the potential to affect multiple physiological systems, with the extent of impact being dependent upon the severity of the injury. In addition, each brain injury is unique, which further contributes to a variety of patient presentations. Due to the wide range of possible impairments, this paper will specifically discuss the effect of mild traumatic brain injury on cognition, neuromotor function, cardiorespiratory function, and vestibular function. The effect of mild TBI on cognition, neuromotor function, and cardiorespiratory function will be discussed in this section, while it’s effect on vestibular function will be discussed in the intervention section.

 According to Arciniegas et al., “at least 85,000 persons who experience TBI each year develop persistent cognitive, emotional, behavioral, or somatic disability,”2 with deficits in attention, memory, and executive function being the most common across all levels of severity.2 In addition to these deficits, comportment (“social intelligence”) and motivation are also frequently impaired after injury.2 While the majority of individuals with mild TBI recover rapidly, it has been reported that impairments in cognition and memory can persist for years after the initial injury, with 58% of patients experiencing deficits 1 month after injury and 15% at 1 year.1,5,6 Deficits in these areas, independent of their severity, can have a significant impact on daily function and have the potential to be a source of morbidity for the individual, their family, and society.2 Selective attention and sustained attention are required for the completion of simple tasks and for new learning; executive function is required for information retrieval, planning and organization of cognition and behavior, fluent shifting between information or behavior sets, and the use of language to guide behavior; comportment is required for appropriate interaction with others; and motivation is required for directing and sustaining goal-directed behavior.2

 In addition to cognitive impairment, neuromotor impairment is a common consequence of TBI.7 In general, it has been reported that neuromotor function rapidly improves within 1-6 weeks after TBI, especially for those who have experienced a mild TBI.7 While the majority of individuals with mild TBI are physically independent on the FIM at 3 and 6 months postinjury7, 30% of patients complain of impaired balance and altered coordination after the initial injury6 and many experience deficits in these areas up to 5 years postinjury.7 During gait and balance assessments, it is often found that individuals with TBI have an increased reliance on visual input and tend to sway more in all directions (anteroposterior and mediolateral).6 It has also been suggested that individuals with TBI may not use their vestibular systems as effectively as those who have not had an injury.6,7 In addition to balance impairments, persons with TBI also experience disturbances in gait, illustrated by a slower walking speed and a shorter stride length.6 These functional limitations result in reductions in whole-body center of mass (COM) displacement in both the sagittal and frontal planes.6 Research suggests that individuals with TBI reduce their COM displacement during gait in order to maintain their balance.6

 Lastly, research suggests that individuals recovering from TBI have an increased risk for cardiovascular and pulmonary limitations during endurance activities.8 While mild TBI results in few physical impairments, the risk of cardiovascular and pulmonary limitations increases if the injury leads to physical inactivity or a sedentary lifestyle.8 The negative effects of inactivity are well established and include poor stamina, reduced muscle strength, and limited flexibility, putting the individual at increased risk for coronary heart disease, hypertension, thrombosis, osteoporosis, obesity, certain cancers, and diabetes.8 In addition to medical complications, inactivity can also lead to decreased employability, since peak aerobic capacity is associated with employment productivity in people recovering from TBI.8 More specifically, research suggests that individuals with TBI are less likely to achieve their age-predicted maximum heart rate during exercise and are more likely to breathe harder during physical activity to obtain adequate oxygen exchange.8 Due to the potential impact of mild TBI on cardiorespiratory fitness, individuals should be encouraged to participate in properly prescribed endurance training programs when appropriate after injury.8

***Activity, participation, and quality of life***

Limitations in activity, participation, and quality of life occur frequently after brain injury, including injuries that are categorized as mild. While each domain has it’s own meaning, there seems to be significant overlap between domains, making it challenging to differentiate how brain injury affects each domain independently. According to the International Classification of Functioning, Disability and Health (ICF), the domains of activity and participation are comprised of the following components: learning and applying knowledge, general tasks and demands, communication, mobility, self care, domestic life, interpersonal interactions and relationships, major life areas, and community, social, and civic life.9 Quality of life, on the other hand, is a multi-dimensional concept that often means different things to different people.10 According to Flanagan11, however, it appears that components of activities and participation often contribute to an individual’s quality of life.

In order to help define quality of life, Flanagan11 developed a list of 15 domains of life that are considered important to people.  These domains can be used to describe QOL in individuals with TBI and include the following:  material comforts, health and personal safety, relationships with relatives, having and rearing children, close relationship with a significant other, having close friends, helping and encouraging others, participation in public affairs, learning (school and other), understanding oneself, interesting work (job, at home), expressing oneself, socializing, passive recreation, active recreation.11  This list also confirms that activity, participation, and quality of life are often related, as limitations in activity and participation can significantly affect quality of life. Out of all of the above categories, however, interesting or satisfying work has been found to be the most significant determinant of QOL after brain injury.10

A study by Ruffolo et al. confirms the above findings and reports that only 12% of subjects with mild TBI returned to full pre-morbid level of employment, while 30% returned to modified work.12 While it is difficult to predict an individual’s ability to return to work, it is suggested that the following factors be considered: (1) severity of injury as measured by length of coma or post-traumatic amnesia, (2) specific measures of impairments resulting from the injury, (3) pre-injury occupational or educational background, and (4) age at time of injury.5 Based on these pre-morbid and injury factors, it has been found that individuals who sustain an injury after 40 years of age are less likely to return to work, while those with higher educational levels and whose pre-morbid occupation was managerial or professional are more likely to return to work.5 In considering those with mild TBI, 15% of individuals remain with post-concussive symptoms a year or more after injury.5 This is important to note because deficits are often subtle and may only become evident when the individual attempts to return to work.5 Although most individuals with mild TBI can manage their own return to work process, productive work is often challenging to achieve, even in cases of mild injury, due to the need for combined social, cognitive, and physical skills.5

While the ability to return to work has a significant impact on activities, participation, and quality of life after TBI, an individual’s sense of self has also been shown to contribute to impairments in the above domains.10 A loss of sense of self can manifest itself in different ways, including loss of personal expression.  Due to things like loss of employment and less opportunity for leisure activities, people with TBI have less occasion to express themselves.10  In addition, Duggan notes that patients often feel empty and lost after injury.13 Research suggests that findings such as these are not directly related to the brain damage itself, but rather to interactions with others.10  After brain injury, many individuals feel like they’re not understood by others both when they are “equated with a ‘damaged’ brain and when they [manage] to pass for ‘normal’.”10 As a result of such thinking, individuals may limit their activities and participation after brain injury, which may lead to further impairments in quality of life.

From the above information, it can be concluded that the effect of brain injury on activity, participation, and quality of life is both significant and complex. The impact of TBI is never isolated, as it affects all domains of life in one way or another.  In addition, brain injury is never the same from one individual to the next, which suggests that brain injury will impact each individual’s activities, participation, and quality of life differently. Therefore, as therapists, we need to recognize this and take the time to determine what’s important to our patients. We need to identify what defines these domains for our patients and do our best to help them find ways to maintain or adapt these definitions as they recover.

***Intervention***

 Dizziness and vertigo are two of the most common complaints after TBI14 and are symptoms that have the potential to persist long after the initial injury. One study suggests that 36% of patients report dizziness 2 years after injury7, while another study reports that headache and dizziness may persist for up to 1 year after mild TBI.1 Although these symptoms are common after TBI, their exact cause is not well understood, as they can originate from damage to the inner ear, cranial nerves, brain or other extra-cranial sources.14 Damage to cranial nerves is common after brain injury, including injuries categorized as mild.6 The vestibular nerve, in particular, is often involved and impairments in the vestibular system may result from the tethering of the vestibular nerve as it passes through the internal acoustic meatus.6 The acceleration and deceleration forces that are sustained during TBI often cause this type of injury.6

 While dizziness and vertigo are common complaints after TBI, many types of dizziness can be experienced after injury, depending upon the type of injury and the brain structures involved. For example, blunt mild TBI can produce positional dizziness, migraine associated dizziness, exercise induced dizziness, and spatial disorientation.15  On the other hand, blast mild TBI can be associated with positional dizziness, exercise induced dizziness, spatial disorientation and spatial disorientation with episodic vertigo plus fluctuating hearing loss, tinnitus, and ear pressure.15 In addition, symptoms can vary depending whether the injury is of peripheral (Benign Paroxysmal Positional Vertigo) or central origin.16 In order to best establish the origin of symptoms, each patient with TBI shoulder undergo a standardized vestibular assessment after injury.15

 Although vestibular pathology is well established after TBI, very little attention has been given to vestibular rehabilitation in this population. Medication is usually the first line of treatment for vestibular pathologies such as headaches and vertigo, while other forms of rehabilitation, such as physical therapy, often go overlooked.15 It is important that the public and other healthcare professionals are educated and aware that physical therapists are able to effectively and efficiently treat these vestibular pathologies, as dizziness and vertigo can have a very negative effect on rehabilitation outcomes for individuals with TBI.14

 When determining an appropriate treatment plan, it has been recommended that the goal of vestibular rehabilitation in this population is to decrease dizziness, increase balance function, and increase general activity levels.15 While research has shown that physical therapy improves functional dynamic visual acuity, standing balance, and gait in patients with mild TBI and vestibular pathology,15 it has been suggested that individuals diagnosed with central or mixed central and peripheral vestibular dysfunction do not recover as well as those with only peripheral dysfunction.17 Brown et al., however, report that individuals with central vestibular disorders demonstrate the ability to improve on measures of balance and postural control following therapy.17

In order to aid in the treatment of central vestibular dysfunction after TBI, Brown et al. suggest that a customized program be developed and include the following: vestibular adaptation exercises, substitution exercises for lost vestibular function, education in use of assistive devices, safety awareness techniques, balance and gait training, general strengthening, flexibility exercises, and balance training.17 When considering more specific treatment techniques for peripheral vestibular dysfunction, like BPPV, it has been shown that the majority of patients who are diagnosed with BPPV after TBI are successfully treated with an otolith repositioning maneuver.14

For more general treatment strategies for vestibular dysfunction after mild TBI, Gotshall suggests exercises that target the VOR, cervico-ocular reflex (COR), depth perception (DP), somatosensory retraining (SS), dynamic gait, and aerobic function.15 In his study, the VOR, COR, and DP exercises were progressed by changing the velocity of the head and the object in motion and by moving from sitting to standing.15 The SS exercises were progressed by narrowing the base of support, making the surface uneven, and changing the support surface from firm to soft.15 Walking exercises were progressed by changing direction, walking with eyes closed, increasing the speed of ambulation, walking on soft surfaces, and by navigating stairs.15

While intervention techniques for vestibular dysfunction vary after TBI, the most successful programs are individualized and have been shown to target gaze stabilization, dynamic visual acuity, static postural stability, dynamic postural stability, desensitization of head motion, and aerobic conditioning.15 In order to monitor a patient’s response to treatment, progress should be monitored with quantitative outcome measures, regardless of the intervention chosen.7 The broad selection of treatment strategies for vestibular dysfunction in this population further confirms the importance of thoroughly assessing each patient for specific impairments. In general, physical therapists need to be more consistent in screening patients with TBI for vestibular pathology, regardless of the severity of injury.  From the literature, it seems that this is something that often goes undetected, which is unfortunate considering the number of effective treatments we can provide to address this issue.

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