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RHODE ISLAND MEDICAL JOURNAL

SPECIAL SECTION

# TRAUMATIC BRAIN INJURY REHABILITATION

GUEST EDITOR: JON MUKAND, MD, PhD

AUGUST 2025 VOLUME 108 • NUMBER 8 ISSN 2327-2228

RHODE ISLAND MEDICAL JOURNAL

J.A. Mukand, MD, PhD

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## Traumatic Brain Injury (TBI) Rehabilitation

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GUEST EDITOR



**On the cover:** In the physical therapy room, a young woman in her 30s who survived traumatic brain trauma from a train accident in 2024 works with her therapist. [iSTOCK PHOTO]

# The Complexities of Traumatic Brain Injuries

JON A. MUKAND, MD, PhD
GUEST EDITOR

Some of the most complex patients I have treated over the last four decades had brain injuries. They often had multifocal pathology that caused a variety of neurological problems. In addition, they usually had medical complications that compromised their rehabilitation and sometimes necessitated a transfer back to the acute care hospital.

A review of medical complications during inpatient rehabilitation for brain injuries found that there were 0.40 events per week per patient, and more than 80% had at least one adverse event. Hypertonia, agitation/aggression, urinary tract infection, and sleep disturbance were the most common (each more than 5% of all complications). The most severe problems included hydrocephalus, pneumonia, gastrointestinal conditions such as bleeding and obstruction, and paroxysmal sympathetic hyperactivity.[1]

Rehabilitation for patients with brain injuries requires an interdisciplinary approach, as reflected in this special issue of the *Rhode Island Medical Journal*. Neurologists are essential for the early care of these complex patients. **BRUNO MOURAO-PACHECO, MD**, and his co-authors discuss hypothermia, hyperosmolar therapy, and cerebrospinal fluid drainage as well as prevention of ventilator-associated pneumonia, deep venous thromboses, and seizures.

In the rehabilitation setting, **STEPHAN P. PIRNIE, MD, PhD,** describes a patient with a severe traumatic brain injury and highlights the neurocognitive, motor, and sensory abnormalities. **ALEXIOS G. CARAYANNOPOULOS, DO, MPH**, and his co-authors offer a review of rehabilitation strategies for aphasia, dysphagia, paresis, respiratory dysfunction, cognition, and behavior. Physical therapy for these patients is complicated by abnormal tone, balance, and cognition – as discussed by **KENNETH VINACCO, PT, DPT, NCS**, and his co-authors. **JOAN M. JORDAN, DHA, CCC-SLP**, and I review current approaches for cognitive-communication rehabilitation after brain injuries.

Even after returning home, patients with brain injuries are vulnerable to long-term complications. **JONATHAN LIU, MD**, and his co-authors review the pathophysiology, evaluation, and treatment of neurogenic heterotopic ossification. Spasticity and abnormal tone can impair the functional status of patients with brain injuries and lead to contractures. **MARY LOU, BS**, and her co-authors offer a comprehensive review of treatment strategies for spasticity, ranging from physical therapy to orthopedic surgery.

Patients with brain injuries require evidence-based and specialized care from an interdisciplinary team, such as the clinicians who have contributed to this issue. These patients should also receive long-term services for home and outpatient rehabilitation, counseling, cognitive rehabilitation, and vocational rehabilitation.

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# Traumatic Brain Injury – A Neurologist's Approach

BRUNO MOURAO PACHECO, MD; ANNA WHITHAM, MD; JONATHAN F. CAHILL, MD

Traumatic brain injury (TBI) is defined as brain injury caused by an external force. It remains a major global health concern, contributing significantly to death and disability across all age groups. In the United States, there were approximately 214,000 TBI-related hospitalizations in 2020 and 69,000 TBI-related deaths in 2021, averaging over 586 hospitalizations and 190 deaths per day. Individuals aged 75 years and older exhibited the highest rates of TBI-related hospitalizations and deaths. Males were nearly twice as likely to be hospitalized and three times more likely to die from a TBI than females.[1]

TBI is increasingly recognized not only as an acute insult but also as a chronic disease process that evolves over time. It carries the potential for long-term cognitive, emotional, and physical disability, underscoring the need for a comprehensive, multidisciplinary approach to care – from the point of injury through rehabilitation.

TBI is typically classified by severity into mild, moderate, and severe categories using the Glasgow Coma Scale (GCS). A GCS score of 13–15 indicates mild TBI, 9–12 indicates moderate, and 3–8 signifies severe injury. Further stratification involves structural imaging (CT/MRI), duration of loss of consciousness (LOC), alteration of consciousness, and post-traumatic amnesia (PTA).[2]

Contemporary classification schemes, including the Mayo Classification System[2] and the use of biomarkers and advanced imaging techniques, aim to provide a more nuanced characterization of injury severity and potential outcomes. Increasingly, the emphasis is shifting from static grading to dynamic, physiology-informed classification systems that account for evolving intracranial pathophysiology.

## PRE-HOSPITAL EVALUATION AND MANAGEMENT

The pre-hospital phase is critical in the management of TBI, as early interventions can significantly influence outcomes. Following primary injury, secondary insults from hypoxia, hypoperfusion, and/or ischemia may occur in the pre-hospital setting. Key priorities include ensuring airway patency, providing adequate ventilation to prevent hypoxia, and maintaining cerebral perfusion by avoiding hypotension. Cervical spine precautions should be implemented until spinal injuries are ruled out. Rapid transport to a facility equipped to manage TBI is essential. Pre-hospital providers should perform frequent pupillary response assessments and report GCS score every 30 minutes or with any change in mental status, which could indicate early signs of herniation or increased intracranial pressure (ICP). Administration of hyperosmolar therapy for prophylactic treatment of suspected elevated ICP, with or without signs of herniation, in the pre-hospital setting is not recommended.[3]

## IN-HOSPITAL INITIAL EVALUATION AND MANAGEMENT

Upon arrival at the hospital, patients with suspected TBI undergo a comprehensive assessment following Advanced Trauma Life Support (ATLS) protocols.[4] This includes a primary survey focusing on airway, breathing, circulation, disability (neurological status), and exposure. Neurological evaluation involves determining the GCS score and assessing pupil reactivity. A non-contrast head CT scan is the imaging modality of choice for detecting intracranial hemorrhages, contusions, and skull fractures. Laboratory evaluations may include coagulation profiles, blood glucose levels, and arterial blood gases. Early neurosurgical consultation is warranted for patients with mass lesions or deteriorating neurological status.[5]

The 4th Edition Guidelines for the Management of Severe Traumatic Brain Injury provide evidence-based recommendations for both treatment and monitoring strategies specific to adult patients with severe TBI.[5] Most treatment strategies are aimed at reducing intracranial pressure which can be elevated following severe TBI. Decompressive craniectomy (DC) is the most definitive and rapid means of reducing or relieving elevated intracranial pressure. Large fronto-temporo-parietal DC is recommended (Level II A) for improved mortality and neurological outcomes in select patients. However, early bifrontal DC, while effective in lowering intracranial pressure (ICP), reducing ICU days, and lowering mortality, was associated with more unfavorable outcomes at six months and did not show six-month functional improvement as measured by the Glasgow Outcome Scale–Extended (GOS-E).[6,7] Evidence supports the use of hypothermia as standard of care for neuroprotection after cardiac arrest from acute coronary syndromes.[8] When hypothermia is induced early after injury and prior to intracranial pressure elevation, it is termed "prophylactic". Prophylactic hypothermia lacks

sufficient evidence for a strong recommendation, as current studies are highly heterogeneous, preventing definitive conclusions.[5] Hyperosmolar therapy also reduces intracranial pressure and remains a mainstay of ICP management, but no single agent, such as mannitol or hypertonic saline, is clearly favored based on current data.[5] Cerebrospinal fluid (CSF) drainage is acknowledged for its utility in reducing ICP, though the evidence base is still developing.[5, 9,10]

Ventilation therapies (previously with emphasis on hyperventilation) are approached with caution. The emphasis is on tailored ventilation strategies that reduce ICP without compromising cerebral perfusion.[5] The use of anesthetics, analgesics, and sedatives in severe TBI remains guided largely by clinical judgment due to the low quality of available evidence.[5] Corticosteroids, particularly high-dose methylprednisolone, are not recommended (Level I), given strong evidence of harm.[5] Nutritional support should be initiated early – preferably within five to seven days post-injury – as evidence suggests a positive impact on recovery.[5] Infection prophylaxis now focuses on targeted strategies like oral care and management of ventilator-associated pneumonia (VAP). Prophylaxis against VAP has been previously supported by ANTHARTIC trial (patients after cardiac arrest)[11] and most recently by PROPHY-VAP which focused on patients with acute brain injury (including stroke, subarachnoid hemorrhage, TBI), this showed a decreased risk of VAP, decreased ventilation days, decreased prolonged ICU and hospital stay, and decreased mortality.[12]

For deep vein thrombosis (DVT) prophylaxis, a Level III recommendation supports the use of low molecular weight heparin (LMWH) or unfractionated heparin (UFH) in combination with mechanical prophylaxis, provided the hemorrhagic risk is acceptable. Finally, clinicians routinely prescribed antiseizure medications of post-traumatic seizure (PTS) prophylaxis despite lacking clear clinical evidence or supporting guidelines. There is modest effectiveness in PTS prophylaxis in mild to moderate TBI.[13] Phenytoin is recommended (Level II A) for early seizure prophylaxis for post-traumatic seizures (PTS) as it is effective in reducing seizures within the first seven days post-injury, though not for preventing late-onset seizures in severe TBI.[14] However, other antiseizure medications such as levetiracetam may pose less risks. It is important to mention that up to one-quarter of patients are inappropriately discharged with antiseizure medications after failure to stop prophylactic medications after seven days.[15] Prolonged and unnecessary antiseizure medication usage may also inhibit recovery from TBI, especially in moderate and severe TBI.[15]

Looking ahead, multimodal monitoring (MMM) represents a shift toward precision neurocritical care and is increasingly being employed with the goal to improve outcomes in patients with severe TBI.[16] ICP monitoring remains a foundational component of TBI management. The BEST:TRIP trial, however, highlighted the shortcomings of relying on ICP monitoring alone and emphasized the importance of using integrated monitoring strategies.[17] Cerebral perfusion pressure (CPP) monitoring is similarly supported with a Level II B recommendation. CPP-guided therapy has been shown to lower two-week mortality, although the overall quality of evidence remains limited.[5] Advanced cerebral monitoring (ACM) techniques are gaining interest as adjuncts to traditional methods.[16] Rather than applying a one-size-fits-all approach, MMM supports individualized treatment strategies based on real-time physiologic data. Future advancements include the development of validated multimodal algorithms, less invasive technologies like near-infrared spectroscopy, and the integration of artificial intelligence for real-time data interpretation and clinical decision support. While not yet standard practice, MMM offers a promising framework for improving outcomes in patients with severe TBI.

## EARLY COMPLICATIONS OF TBI

Early complications following TBI can significantly influence patient outcomes, so they require close monitoring and timely intervention. TBI experimental animal models are used to replicate human pathophysiology and clarify aspects of primary and secondary brain injury.[17]

Early damage in TBI often follows from an ischemic cascade and disruption of normal metabolic energy processes such as decreased glucose utilization, lactic acid accumulation, reduced ATP usage, excitotoxicity, and cellular death.[18] One of the most critical concerns is elevated ICP, which can progress to brain herniation – a life-threatening emergency that demands immediate treatment. Seizures are another common complication, with approximately 10% of individuals hospitalized for moderate to severe TBI experiencing post-traumatic seizures, most often within the first few days to weeks after injury.[19] Coagulopathy is also frequently observed, as TBI can disrupt the coagulation cascade and cause platelet dysfunction akin to disseminated intravascular coagulation (DIC), which increases the risk of both intracranial hemorrhage and cerebral ischemia.[20-21] Additionally, neurogenic pulmonary edema may develop because of acute brain injury, posing significant challenges for respiratory management.[22] Infections such as ventilator-associated pneumonia and surgical site infections are prevalent among TBI patients due to factors such as prolonged hospitalization, mechanical ventilation, and compromised immune responses.[5] Prompt recognition and management of these early complications are essential to improving short- and long-term outcomes in patients with severe TBI.

## REHABILITATION STRATEGIES BEGINNING IN THE HOSPITAL

Early initiation of rehabilitation is essential for long-term recovery in almost all types of injuries. For TBI, hospital-based

cognitive rehabilitation offers little to no effect on return-to-work rates, but post-acute care becomes vitally important.[23] Compared to hypoxic-anoxic ischemic brain injury, better functional outcomes can be achieved after traumatic brain injury.[24] Post-acute therapy recommendations differ with severity of TBI, with mild severity needing minimal therapy for likely return to premorbid daily functioning while severe TBI patients have indeterminate and variable outcomes. Mild TBI (mTBI) can be further characterized into uncomplicated and complicated, the latter referring to patients with findings on CT. The Collaborative European NeuroTrauma Effectiveness Research in TBI (CENTER-TBI) showed that complicated mTBI had poorer outcomes than uncomplicated mTBI and that greater duration of therapy predicted poorer outcomes, as patients with more severe injuries needed more intensive treatment.[25] A greater number of transitions of care and pre-morbid psychiatric illnesses also predicted poorer outcomes for mTBI.[23]

In contrast to mild TBI, greater duration of therapies for severe TBI predicted a more favorable prognosis. Recommended therapies include physical (PT), occupational (OT), speech language pathology (SLP), psychiatric (PSY) and cognitive rehabilitation. In physical therapy, greater patient effort and more complex therapy, rather than length of therapy, are associated with improved functional outcomes.[26] Earlier and more intensive occupational therapy has been shown to improve outcomes.[27] Social communication approaches have been shown in a systematic review to be the most effective approach for SLP intervention for moderate to severe TBI.[28]

Overall, TBI functional outcomes are influenced more by patient and injury characteristics than time spent in therapies. For example, mechanism of TBI (assault), CT abnormalities, and premorbid alcohol use predicted worse outcomes on the Glascow Outcome Scale Extended (GOS-E).[29] The GOS-E is an assessment of physical, social and cognitive function that categorizes TBI patients in one of eight levels, from death to upper good recovery.[30] While the GOS-E is widely accepted for TBI outcomes, more complex, structured assessments are often used to quantify TBI outcome after rehabilitation to capture more of the nuanced improvements. One such outcome measure is the Functional Independence Measure (FIM) Cognitive score, which measures 13 motor and five cognitive items and rates patients from one (total assistance) to seven (complete independence).[26] A lower FIM score on admission to rehabilitation centers is associated with patients who had more in-hospital days prior to rehabilitation, Medicaid as primary payer, increased levels of agitation, and younger age.[26] Lower FIM scores on admission for rehabilitation were associated with a longer length of stay and decreased effort with OT/PT/SLP. Patient effort level (as rated by clinicians) during therapies was strongly associated with post-rehab placement, with those showing higher effort more likely to be discharged to a private residence.[26]

Current medical interventions in the post-acute TBI period affect outcomes less than therapies.[26] Though there is data to support anti-seizure medications (ASM) in prevention of early post-traumatic epilepsy, late seizures (greater than six months post-injury) and mortality are not modified by ASMs.[31] Similarly, neuro-protective agents such as magnesium sulfate did not show benefit.[32] In mild TBI, methylphenidate improves cognition, n-acetyl cysteine within 24 hours of injury helps with faster recovery, and galantamine improves episodic memory, but these findings cannot be extrapolated to those with moderate or severe TBI.[33] For severe TBI, amantadine may hasten recovery in the first four weeks after injury, but overall recovery at six weeks after a two-week washout period was not significantly different from placebo.[34]

## LATE COMPLICATIONS OF TBI

Most patients with severe TBI will have long-term disability in health, behavior and functional status. The United States Traumatic Brain Injury Model Systems of Care, which has followed individuals with moderate-to-severe TBI for over 30 years, has shown that TBI increases rates of hospitalization and decreases life expectancy compared to the general population.[35] Deficits may not be at peak at diagnosis, either, with evidence for decline overtime. For example, in the United States, the TRACK-TBI LONG study found that additional functional decline occurred in 29% of mild TBI and 23% of moderate to severe TBI to seven years post-injury.[35] Older age and lower acute functional status were associated with higher rates of post-injury decline. Rates of psychotic disorders, attention deficit hyperactivity disorder (ADHD), suicide, and depression are also increased post-TBI compared to a general population, with a relative risk of ADHD as high as 6.49 in the severe TBI cohort.[36]

Chronic traumatic encephalopathy (CTE) has been an increasingly researched entity, thought to occur from repetitive mTBIs. Official diagnosis requires demonstration of tauopathy on autopsy. Traumatic encephalopathy syndrome (TES) has been coined to describe the progressive symptoms associated with presumed CTE. Patient-specific targeted rehabilitation for cognition, executive functioning and emotional control in TES has been shown to improve patient-reported outcomes, with mixed objective significance.[37] Physical exercise has shown to be beneficial for motor function, balance and cognition in tauopathies,[38] and this has been extrapolated to treatment of TES.

Challenges with TBI rehabilitation research include the lack of standardized scoring as well as the observational and longitudinal nature of studies. As discussed previously, GOS-E is the most widely used outcome measure because of its simplicity and flexibility of administration including low administration time, but it may fail to capture symptoms and quality of life after TBI. Because long-term outcome

research requires the passing of time, studies are more logistically demanding and subject to error from patient loss to follow-up.

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## Disclosures

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# Common Sequelae of Severe Traumatic Brain Injury: A Case Report

STEPHAN P. PIRNIE, MD, PhD


## ABSTRACT

Severe traumatic brain injuries (TBI) are caused by external forces that damage brain tissue and result in prolonged cognitive, sensory and motor deficits. This case report highlights the neurocognitive, motor, and sensory results of severe traumatic brain injury as well as several sequelae that often complicate TBI.


**KEYWORDS:** Brain Injury; TBI; Diffuse Axonal Injury; Heterotopic ossification; Post-traumatic Amnesia

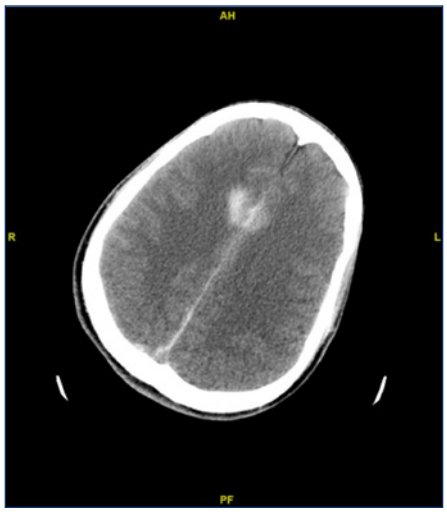
## INTRODUCTION

Traumatic Brain Injuries (TBI) exist on a spectrum of severity from mild TBI/concussion with time-limited symptoms to severe traumatic brain injury with long-term, significant cognitive, motor, and sensory deficits. Severe traumatic brain injury results from an external force applied to the brain and is defined by one or more of the following criteria: loss of consciousness for greater than 24 hours, initial Glasgow Coma Scale (GCS) of 3–8, and an episode of post-traumatic amnesia lasting greater than seven days. Additionally, the clinical course of these patients usually involves complications, including dysphagia, prolonged respiratory failure, heterotopic ossification, agitation and restlessness, and concomitant orthopedic injuries. The following case describes the complex medical management and complications of a patient with a severe TBI.

## CASE PRESENTATION

A 40-year-old man was the helmeted rider of a motorcycle that crashed into a car at highway speeds. His GCS in the field was 6–7, and he was intubated on arrival at the ED. Imaging demonstrated a subarachnoid hemorrhage, subdural hematoma, intraparenchymal contusions [**Figure 1**], facial fractures, bilateral rib fractures, pneumothorax, left hemothorax, left scapular fracture, right cervical ICA dissection, right scaphoid fracture, and left perilunate dislocation. Five days later, an MRI scan of the brain redemonstrated multifocal intraparenchymal hematomas, notably in the rostral corpus callosum, frontal lobes, and anterior (right greater than left) temporal lobes [**Figures 2**,**3**]. Additional findings included multifocal microhemorrhages and diffusion-restricting foci that were consistent with diffuse axonal injury (DAI) [**Figure 4**].

**Figure 1**. Non-Contrast CT Brain demonstrating Intraparenchymal hemorrhage in the bilateral medial frontal lobe and parafalcine subarachnoid hemorrhage.




He required a tracheostomy for prolonged endotracheal intubation. Due to severe dysphagia, a PEG tube was placed to allow enteral nutrition. His acute hospital course included multiple surgical procedures, including stabilization of facial fractures, open reduction and internal fixation (ORIF) of the right scaphoid fracture, and pinning of the left fifth metacarpal. His early acute course was notable for ongoing decreased alertness, and he was started on amantadine.[1] He developed significant psychomotor agitation, for which he started on propranolol. He had movement of the right upper and lower extremities but limited spontaneous movement on the left. With range of motion during therapy, he demonstrated significant pain with passive movement of his left upper extremity, especially at the elbow. Further evaluation revealed elevated alkaline phosphatase and heterotopic ossification (HO) in his left distal triceps on plain radiograph [**Figure 5**]. He started indomethacin[2] but did not tolerate a full course because of GI side effects.

Approximately 1.5 months following his injury, he was following simple commands but remained disoriented and had limited verbal output. Two months following his injury, he was able to vocalize spontaneously and follow simple one-step commands consistently. Approximately 2.5 months after his injury, he was transferred for acute inpatient rehabilitation to focus on intensive therapy with ongoing management of his medical sequelae.

The patient made slow but consistent progress, with improved movement of his left side, verbal output, carryover of commands, memory, and

**Figure 2.** MRI brain demonstrating FLAIR hyperintensity

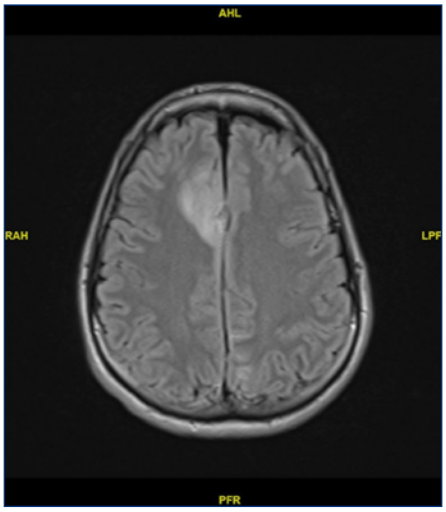



**Figure 3.** Intraparenchymal hemorrhage (SWI sequence which highlights areas of hemosiderin deposition) in the right medial frontal lobe.

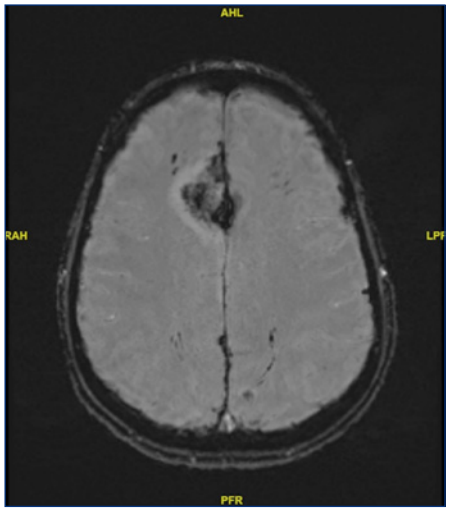
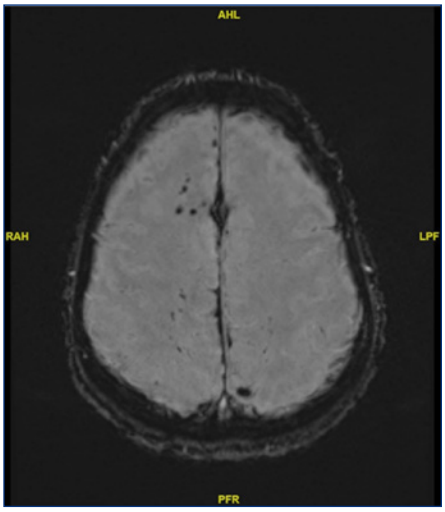



**Figure 4.** SWI MRI images with microhemorrhages in the bilateral cerebral hemispheres, consistent with diffuse axonal injury.

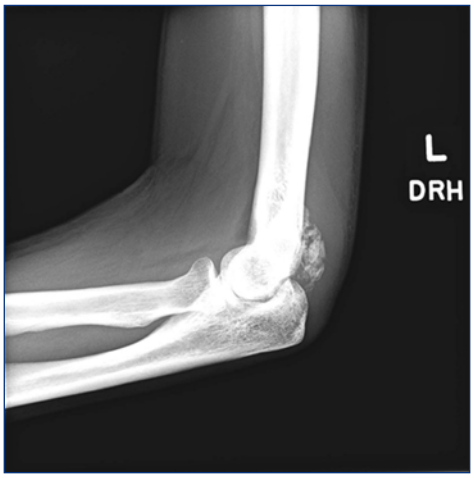



orientation. He emerged from post-traumatic amnesia approximately three months after his initial injury, evidenced by consistent orientation. His dysphagia was mainly characterized by oral apraxia, but he progressed to an oral diet; he improved to the point of meeting his nutritional needs, and his PEG tube was removed prior to discharge. Propranolol was weaned off as his psychomotor agitation improved with improving cognition. During his inpatient rehabilitation, he improved his ambulation; at the time of discharge, he needed contact guard for stand-pivot transfers and ambulation up to 150+ feet with a platform rolling walker. He continued to need minimal assistance for ADLs, including bathing and dressing. Throughout his rehabilitation course, he had significant support from his wife, who was able to provide assistance at home. She decreased barriers to his home discharge by installing a ramp at an entrance to their home and grab bars in the bathroom to improve his mobility and safety. He was eventually discharged home with his wife approximately five months following his injury.

Due to ongoing focal weakness in his left upper extremity and localized sensory loss in the ulnar aspect of the hand, there was concern for a peripheral nerve injury at the brachial plexus or ulnar nerve.

Electromyography and nerve conduction studies (EMG/NCV) demonstrated significant ulnar nerve injury, with evidence of axonal injury and demyelination localized at the elbow, which has been associated with heterotopic ossification in this location.[3] His home therapy focused on improving his independence with ADLs, fine motor function, balance, and ambulation.

**Figure 5.** Plain radiograph of the left elbow, with a focus of heterotopic ossification in the distal triceps.




## DISCUSSION

This case report highlights a typical recovery course of a patient with a severe TBI. His neurologic imaging displayed focal structural disruptions reflected in intraparenchymal hemorrhage/contusions as well as cellular neuronal damage, or diffuse axonal injury. These different primary neurologic injuries lead to different functional outcomes. For example, focal contusions in the parietal lobe may cause hemisensory loss and frontal lobe contusions typically lead to disinhibition. Conversely, diffuse axonal injury often leads to more global symptoms such as slowed processing time, post-traumatic amnesia, or disorders of consciousness including coma and minimally conscious state.[4] This case highlights the positive outcomes but prolonged recovery after TBI, as this patient's total hospitalization was approximately four months from admission to discharge home.

This case also highlights some common sequelae of severe TBI, including dysphagia, prolonged respiratory failure, agitation and restlessness, and concomitant orthopedic injuries. Rehabilitation for TBI focuses on symptom management to allow optimal participation in physical, occupational, and speech therapy as well as maximum functional recovery.

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# Rehabilitation Strategies for Traumatic Brain Injury: Insights and Innovations

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## INTRODUCTION

Traumatic brain injury (TBI) represents a significant public health challenge, manifesting in a spectrum of cognitive, physical, and emotional impairments that profoundly affect individuals' long-term functioning. In Rhode Island, where the prevalence of TBI is on the rise, addressing the complexities of rehabilitation is of paramount importance. This article is a review of current rehabilitation strategies for the evaluation and treatment of individuals with TBI, with a focus on aphasia, dysphagia, paresis, respiratory dysfunction, cognition, behavior, and long-term outcomes.

## ACUTE REHABILITATION GOALS AND TREATMENT STRATEGIES

Participation in interdisciplinary rehabilitation should begin in the intensive care unit to mitigate the complications of critical illness.[1] Acute inpatient rehabilitation after TBI is associated with improved long-term functional outcomes, lower mortality, and greater odds of regaining independence in the community.[2] Treatment teams consist of therapists, occupational therapists, and speech and language pathologists, a case manager, social worker, and a physiatrist as well as consultants such as psychiatry, neurology, psychology and nutrition.

## COMMON SEQUELAE AND MANAGEMENT STRATEGIES

Traumatic brain injury can lead to a variety of neurological impairments dependent on damage to specific brain areas or pathways, causing disruptions in the brain's ability to control and coordinate various bodily functions. The extent of these impairments can widely vary depending on factors such as the severity of injury, location, and the individual's health. Common sequalae of traumatic brain injury include pain, autonomic dysfunction, spasticity, aphasia, dysphagia, paresis, seizures, respiratory dysfunction, as well as cognitive and behavioral impairments.[3] Any of these complications alone or in aggregate can significantly limit a patient's function and quality of life.

## APHASIA AND DYSPHAGIA

Traumatic brain injury can damage language centers in the left hemisphere, where Broca's and Wernicke's areas are located. Broca's aphasia affects speech production and fluency, while Wernicke's aphasia impacts language comprehension. Dysphagia, or difficulty swallowing, can occur with damage to areas that control muscles in the swallowing process, including the brainstem and cortical regions such as the precentral gyrus. Aphasia and dysphagia significantly impact communication and feeding, affecting quality of life post-injury. Speech therapy targets improvement of language skills and swallowing capabilities through exercises to strengthen oral muscles and improve speech clarity. Technology aids such as speech-generating devices also assist communication. Percutaneous endoscopic gastrostomy (PEG) is used when oral feeding is unsafe; a feeding tube is inserted directly into the stomach, with careful monitoring to ensure proper nutrition and tube function.

## PARESIS

Paresis, or muscle weakness, results from injury to the corticospinal tract or motor cortex, which are responsible for voluntary muscle movement. Depending on the location, this can lead to unilateral or bilateral weakness. In severe cases, it can progress to paralysis of the affected muscles. Rehabilitation for paresis focuses on restoring movement using functional electrical stimulation (FES), task-specific training, and motor learning to promote strength and coordination. Severe TBI and resultant immobility can predispose to heterotopic ossification, which can be diagnosed through symptoms (pain, swelling, decreased range of motion) and imaging and managed with physical or occupational therapy, medications to halt ossification, or surgical intervention in severe cases.

## COGNITIVE IMPAIRMENT AND DISORDERS OF CONSCIOUSNESS (DOC)

While both cognitive impairment and DOC are consequences of brain injury, they differ in the severity and location of injury, which affects their pathophysiology and rehabilitation approaches. Cognitive impairment results from damage to brain regions like the prefrontal cortex, temporal lobes,

or hippocampus. Pathophysiological changes may include disrupted neural networks, neurotransmitter imbalances, and neuronal damage, which affect memory, attention, language, and executive function. DOC result from widespread or severe brain injury to the reticular activating system, thalamus, or cortex, leading to impaired arousal and awareness; these disorders include coma, vegetative state, and minimally conscious state.

Cognitive rehabilitation therapy (CRT) focuses on retraining cognitive processes through exercises, tasks, and compensatory strategies. CRT may incorporate pharmacotherapy to manage symptoms like attention deficit or depression as well as structured tasks like memory aids and puzzles. Environmental modifications are important to reduce distractions.

Cognitive-behavioral therapy (CBT) can address cognitive, emotional, and behavioral challenges to mitigate agitation, impulsivity, and emotional dysregulation. Personalized strategies include cognitive restructuring, behavioral activation, managing triggers, symptom management, goal-setting, and problem-solving to promote recovery and improve quality of life. CBT should include the patient, family, and caregivers to be most effective. For DOC, sensory stimulation therapy engages the patient with auditory, visual, tactile, and olfactory stimuli to enhance arousal and responsiveness. Medications like amantadine have been studied for their potential to improve outcomes.[4] Family and caregiver support training is important for those involved in day-to-day care of these complex patients. Emerging technologies include neuroimaging and brain-computer interfaces (BCI) for diagnosis and treatment options.

## RESPIRATORY ISSUES

Traumatic brain injury can cause irregular breathing or respiratory failure from damage to the brainstem; dysregulation of respiratory depth and rhythm from damage to neural pathways; aspiration from disruption of the autonomic nervous system; and sleep apnea from muscle weakness of the diaphragm. Respiratory therapy aims to improve ventilation, maintain airway clearance, and prevent complications such as pneumonia. Mechanical ventilation is often necessary in severe cases, which includes ventilators that either assist or take over the breathing process. Continuous positive airway pressure (CPAP) provides a steady flow of air to maintain airway patency. Tracheostomy is performed when long-term ventilatory support is required. Suctioning can remove secretions to prevent airway blockage and infections. Supplemental oxygen delivered via nasal cannula or mask can ensure adequate oxygenation; nebulization directly delivers bronchodilators and corticosteroids to the lungs to open the airways and reduce inflammation. Airway clearance techniques include chest therapy, coughing exercises, and postural drainage to clear mucus and prevent pneumonia. Finally, inspiratory muscle training (IMT) employs tools and exercises to strengthen the diaphragm and intercostal muscles to improve respiratory function.

## LONG-TERM OUTCOMES FOLLOWING TBI

Traumatic brain injuries can range in severity from mild to severe. Multiple factors including the patient's history and risk factors, mechanism/type of injury, extent of injury, and recovery timeline all impact the severity of the brain injury. While prognostication for both long-term recovery and overall functioning after traumatic brain injury is not an exact science, there are multiple tools that can provide guidance for patients, their families, and medical providers. Utilizing these predictors for prognosis allows for improved patient care and expectation setting for long-term management.

The Glasgow Coma Scale (GCS) is one of the first scores utilized during a trauma evaluation (especially when a possible brain injury is suspected). The GCS is made up of three parts to assess the severity of the brain injury, with a highest score of 15 and a lowest score of 3. The best motor response ranges from 1 (no response) to 6 (obeys verbal commands). The best verbal response ranges from 1 (no response) to 5 (able to converse, is alert, and oriented). Eye opening ranges from 1 (no eye opening) to 4 (opens eyes spontaneously). The more severe the injury, the lower the score, with a mild TBI being categorized by GCS of 13–15, moderate TBI from 9 to 12, and severe TBI from 3 to 8. While all three parts are important for assessment, the best motor response is the best predictor of outcome. Overall worse outcome is based on the lowest GCS in the first 24 hours of injury. The Glasgow Outcome Scale (GOS) illustrates the relationship between GCS and possible recovery/level of disability. It is divided into five categories, ranging all the way from death to good recovery. The Glasgow Outcome Scale Extended (GOSE) is a newer instrument, with an expanded 8-point scale for levels of disability after TBI. For example, while the GOS simply has severe disability, GOSE includes lower severe disability and upper severe disability, based on "frequent" vs. "infrequent" assistance for activities of daily living.

Other predictors of long-term outcome and recovery after TBI include duration of the coma and post-traumatic amnesia (PTA). PTA is described as the time when the patient can recall daily events after their injury. It is often assessed through the Galveston Orientation and Amnesia Test (GOAT) or the Orientation Log (O-log). A score of 75 or higher on the GOAT or 25 or higher on the O-log for two consecutive days indicate that the patient is no longer in PTA. Longer durations of coma and PTA are both associated with worse outcomes. The Disability Rating Scale and the Coma Recovery Scale are also used to assess early recovery and predict final functional outcome.

Younger age (specifically age >5 and <65) often predicts improved outcomes. The presence of significant neuro-

imaging findings (e.g., bi-hemispheric lesions) and neurological findings such as non-reactive pupils, decerebrate posturing, and oculocephalic signs are all associated with poor outcomes. Deficient or absent somatosensory evoked potentials (SSEPs) have also been associated with poor outcomes. Lastly, levels of proteins in the blood such as Glial Fibrillary Acidic Protein (GFAP), Ubiquitin C-terminal Hydrolase L1 (UCH-L1), neurofilament light chain (NfL), and S100B can be measured to further assess the severity of a traumatic brain injury.

Utilizing these prognostic scores to predict the extent of disability after TBI allow the patient and family to better prepare for the future. This creates time to arrange support within the home, whether from family caregivers (requiring teaching) or external help (home health aides). Home modifications can also be made to improve functional independence and decrease caregiver burden. For example, stair/chair lifts and ramps can be installed to make homes accessible. Durable medical equipment (DME) like commodes, shower chairs, and ambulation devices assist with improving a patient's independence with activities of daily living and mobility. Communication aids also allow patients to express their needs and interact without relying entirely on family members. During the acute rehabilitation stay, case managers, therapists, and social workers ensure the safest discharge plan, provide information on community resources, and order DME.

Early access to interdisciplinary rehabilitation care is essential for maximizing the possibility of independent living. This usually includes physical, occupational, and speech therapy along with cognitive rehabilitation and behavioral therapy in the acute care, post-acute care (inpatient rehabilitation), home, and outpatient settings. Early rehabilitation can also assess the need for adaptive equipment and family education. Brain injury organizations at both the local and national level are sources of education and support. For patients who wish to return to work, a gradual transition or engaging in vocational rehabilitation are recommended.

Impaired physical functioning, cognitive and behavioral changes, and increased psychosocial stressors after TBI can all be limiting factors to returning to work. Pre-injury factors such as employment status, education level, occupation, and demographics (age, marital status) are predictors for returning to work. Factors related to the brain injury – GCS, overall disability level/injury severity (similar to GOSE), and length-of-hospital stay – are also predictors for working after TBI. Workplace accommodations including modifications such as increased break time/frequency and access to vocational rehabilitation are associated with an increased likelihood to return to work.

## AREAS OF NEED IN RHODE ISLAND

The care of patients with traumatic brain injury is complex and requires multiple medical specialties, therapists, social workers, and community resources to properly care for patients and support their families. There are clearly areas of success as well as opportunities for improving access to these resources in Rhode Island. Trauma centers within the state have excellent neurologic and neurosurgical care, and several inpatient rehabilitation units provide post-acute care for patients with TBI. However, these patients often face difficulty with ongoing support following their inpatient rehabilitation.

Physical medicine and rehabilitation providers receive education and training on the care of patients with TBI during residency. Advanced fellowship training and board certification are available, but there are no brain injury specialists in Rhode Island. To access this specialized care, patients must travel to neighboring states (Connecticut and Massachusetts). Additionally, patients need home health agencies that can manage the behavioral challenges associated with TBI. There is limited long-term care for patients who are physically functional but cognitively impaired and unable to return home. Further challenges include limited availability of cognitive and neuropsychology programs for supporting patients in the outpatient setting. There are areas of improvement for the care of patients with TBI in Rhode Island. For instance, information packets with local and national resources for clinical care and community support of patients with TBI would be helpful at acute care hospitals and primary care clinics. Patients with TBI have complex care needs, and interdisciplinary care centers that include neurosurgery, physical medicine and rehabilitation physicians, therapists, psychologists, and social workers would lead to more efficient and comprehensive treatment. Finally, the state should invest in recruiting physicians, therapists, neuropsychologists, and social workers who specialize in caring for patients with TBI.

## CONCLUSION

The multifaceted nature of TBI rehabilitation calls for a collaborative approach that incorporates innovative practices and addresses the unique needs of these individuals. Effective assessment tools, personalized interventions, and ongoing support will help optimize long-term functional outcomes for TBI survivors in Rhode Island. Continued efforts to identify gaps in care and enhance rehabilitation practices will play a critical role in improving the trajectory of recovery for this vulnerable population.

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# Restoring Function After TBI: A Review of Physical Therapy Strategies for Balance, Gait, and Dual-Task Recovery

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## ABSTRACT

Individuals with moderate to severe traumatic brain injury (TBI) often experience impairments in balance, gait, and dual-task ability, limiting their functional independence and community reintegration. These deficits arise from disruptions across sensory, motor, and cognitive systems, requiring comprehensive physical therapy (PT) assessment and intervention. PT evaluations incorporate strength, coordination, and sensory integration measures using standardized outcome measures such as the Berg Balance Scale, Functional Gait Assessment, and dual-task assessments such as the Timed Up and Go (cognitive). Treatment strategies include high-intensity training, gait and balance training, and vestibular rehabilitation, each targeting specific deficits to promote neuroplasticity and recovery. Technological interventions like the LiteGait®, virtual reality, and the Bioness Integrated Therapy System enhance therapy outcomes by improving safety, cognition, and balance. PTs must address psychosocial barriers and collaborate across disciplines to support holistic recovery. Ongoing evaluation using outcome measures guides treatment progression and readiness for community reintegration, highlighting PT's critical role in restoring functional independence post-TBI.

## INTRODUCTION

Patients with moderate to severe traumatic brain injury (TBI) often have impaired balance, gait, and dual-task ability, which impacts their self-care, household, and community activities.[1] Balance deficits can be related to disruption of the complex integration between sensory, visual, and vestibular systems.[2] Individuals with TBI frequently demonstrate altered gait patterns, reduced walking speed, and impaired balance, particularly under dual-task conditions where cognitive and motor demands compete for attentional resources. Due to the multi-system effects that a TBI can have on cognition, sensory, and motor systems, there are limited available resources, leading to increased attentional demands while walking.[3]

Physical therapy (PT) plays an essential role in assessing balance and gait impairments, with quantitative and qualitative tools and outcome measures.[4] A physical therapy evaluation is multifaceted, including assessment of strength, range of motion, posture, balance, and gait while simultaneously considering cognition, sensory processing, and coordination. This assessment is the basis for treating balance, gait, and dual-task ability in people with TBI, which helps with re-integration back into the community.

## ASSESSMENT OF BALANCE AND GAIT DEFICITS

Patients with TBI may experience impairments of the visual, vestibular, and proprioceptive systems, which are the primary mechanisms for postural control and balance.[5] Impairments in the ability to integrate information from these systems may also affect postural control and balance. TBIs can be diffuse, so other brain regions that maintain postural control may be damaged, including the cerebellum, pre-motor and motor cortices, and involvement of the vestibular system and cranial nerves. This can result in dyscoordination, weakness, spasticity, and abnormal motor planning, which further impairs balance in patients with TBI. As such, clinicians should examine these systems to determine if there are problems with their function or integration.[2,6]

In 2016, the Academy of Neurologic Physical Therapy (ANPT) published recommendations for outcome measures, including balance, when assessing patients with TBI, followed by additional recommendations in 2018 for patients with neurologic impairments.[4,7]

For patients with limitations in static and anticipatory standing balance, the Berg Balance Scale (BBS) is helpful in assessing fall risk, identifying treatment goals, and tracking progress. Patients who can ambulate with or without a device should have evaluations of their dynamic balance with the Functional Gait Assessment (FGA) or the FGA-Advanced (FGA-A).[2] Patients with TBI who are engaged in high level functional mobility in the community should be assessed with the High-Level Mobility Assessment Tool (HI-MAT) or Community Balance Mobility Assessment Tool (CBMT).[8,9]

## ASSESSMENT OF DUAL-TASK DEFICITS IN TBI

Many daily activities involve concurrent motor tasks (walking while holding a box) or cognitive-motor tasks (conversing and driving).[10] Dual tasking requires the coordination

of multiple areas of the brain, including sensory, motor, and executive function, which can be disrupted in patients with TBI.[11]

A study demonstrated limitations in dual-task ability through use of the Stroop Word Task, which assesses attention and executive function based on the time to complete the task as well as the number of errors.[12] This study also demonstrated that people with a TBI exhibited greater difficulty, slower gait speed, and more caution when navigating obstacles, which may suggest increased reliance on attention for safety.

Although cognitive/communication impairments after TBI are usually evaluated by speech-language pathologists or occupational therapists, physical therapists also assess these deficits as they relate to balance and gait function. There are several tests that are recommended to assess dual-task ability across neurologic populations, including TBI. Standardized tests such as Walking While Talking (WWTT), Walking And Remembering (WART), and TUG-Cognitive (TUG-C) have shown excellent reliability and high inter- and intra-rater testing.[13] Dual-Task Cost (DTC) can be calculated with these outcome measures as follows: ((dual task performance-single task performance)/single task performance x 100). This provides a quantitative measure for changes in dual-task integration throughout the rehabilitation program.[3,14] Further research is needed to obtain data for dual-task cost related to TBI, but it remains a valuable assessment to monitor progress in dual-task ability.

## TREATMENT OF BALANCE, GAIT, AND DUAL-TASK DEFICITS

Exercise is beneficial for individuals with chronic moderate-to-severe acquired brain injury. After six weeks of a moderate-to-high intensity program, a study demonstrated significant improvements in endurance, advanced gait, and ambulatory status, which were maintained six weeks after the program ended.[15] Exercise also improved physical, cognitive, emotional, and social functioning as well as overall well-being. Encouraging participants to reconnect with their "athlete" identity, based on their past involvement in sports, was highly motivating and contributed to positive psycho-social outcomes. This approach helped bridge the gap between their "old self" (pre-injury) and "new self" (post-injury), promoting a sense of self-affirmation and boosting their confidence. Overall, increasing physical activity while incorporating salient social aspects kept participants engaged.[15]

Patients with TBI can improve their balance through treatment strategies that integrate principles of motor learning to induce functional neuroplasticity.[16] However, there is limited evidence for the effectiveness of balance interventions in people with moderate to severe TBI.[17] Current treatment strategies to address balance in this population include high intensity training (HIT), vestibular rehabilitation therapy (VRT), and virtual reality (VR).

HIT has been utilized for dynamic balance retraining in neurologic diagnoses including stroke, SCI, and TBI, but there is a paucity of literature on HIT for patients with TBI. Although strokes and TBI have different mechanisms of injury, both involve damage to the white matter, leading to similar functional deficits.[18,19] For this reason, the evidence for people with chronic strokes has been extrapolated to people with chronic TBI. Studies have demonstrated sustained improvements in transfers, balance confidence, and dynamic balance with HIT when compared to low intensity controls.[18,20] More studies are needed to explore the benefits of HIT training for patients with chronic moderate to severe TBI.

Treadmill walking with support harnesses, such as the LiteGait® system, can be used to assist patients through partial weight support or as a safety harness, depending on the patient's abilities. It allows for repetitive gait training and high intensity training while maintaining safety, which can enhance neuroplasticity and motor learning.[21] Although research has not demonstrated the benefits of body-weight-supported treadmill training for people with an acquired brain injury, it is an effective approach for improving walking capacity and gait quality. The repetitive nature of this training is thought to re-establish sensorimotor systems in individuals with moderate to severe chronic brain injuries.[22]

VRT is also utilized by physical therapists to improve balance for individuals with chronic moderate to severe TBI. VRT can include gaze stability training, habituation to dizziness, and balance exercises. Gaze stability training involves focusing on a target coupled with head motion, which recruits the vestibular ocular reflex (VOR). Habituation exercises induce moderate dizziness to help the brain adapt and reduce the intensity of dizziness. Balance exercises usually focus on sensory integration to improve vestibular and other sensory inputs.[23] The evidence for VRT on balance is mixed but favors VRT over conventional PT.[23,24] VRT may also include canalith repositioning maneuvers (CRM) for patients with Benign Paroxysmal Positional Vertigo (BPPV), which can affect 4–38% of people with TBI.[25,26] The effectiveness of CRMs ranges from 60–85% but recovery may be more prolonged in patients with TBI.[27,28] These maneuvers can cause neck/back injuries, nausea, and dizziness, so they should be performed by only well-trained therapists.

VR programs are emerging as treatments to augment balance therapy and even improve cognitive deficits for people with TBI.[29] Some of these VR systems immerse individuals in environments with obstacles and other pedestrians, so they have to focus on safe ambulation in the community. A small systematic review found no significant differences in balance outcomes between VR and conventional PT, but the authors noted that VR has promising effects on the visual, somatosensory, and vestibular systems; it also includes

motor learning principles such as repetition, feedback, and motivation.[6] Given that VR is more accessible in clinical settings, its utilization offers clinicians the ability to augment balance treatment and integrate motor learning principles.

A study on avoiding collisions with virtual pedestrians showed that patients with moderate to severe TBI have locomotor limitations as well as reduced cognitive task accuracy under dual-task conditions.[29] With VR they can safely practice interactions with virtual pedestrians and less risk of falls and injuries As expected, participants with TBI had alterations in their gait and balance for obstacle avoidance. They also had difficulties with dual-tasking and avoiding pedestrians from multiple directions. This study demonstrated that increased task complexity had a greater impact on gait and balance.

Another technology, the Bioness Integrate System (BITS), has been utilized in the rehab setting to improve visual and spatial function. The BITS is a computer-based interactive tool that offers a variety of programs to improve visual, cognitive, and motor impairments in neurological populations. These activities are designed to improve reaction time, working memory, visuospatial perception, balance, and postural stability. These tasks can be measured for changes during a patient's rehabilitation treatment. There is limited research for its use in the TBI population, but other neurological conditions such as stroke have responded well to this training.[30]

## CONCLUSION

Physical therapists play a vital role in the assessment and treatment of balance, gait, and dual-task deficits following chronic moderate-to severe TBI. There is mixed evidence for some of the above treatments, but these interventions have improved balance, mobility, and safety. PTs should assess patients with TBI to determine which treatment strategies are best for balance, gait, and dual-task impairments. Psychological and social barriers should also be considered, given their prevalence in this population and their potential role in community re-integration.[23] More research is needed to determine which treatment strategies are most effective for treating patients with chronic moderate to severe TBI.

Overall, progress is tracked throughout the rehabilitation program to determine when patients are ready for reintegration into the community or their work life. This includes regular assessments of gait speed, balance and fall risk, and cognitive recovery. Standardized outcome measures should be used to assess cognition, dual-task function, balance, gait, and functional mobility. These tools can guide physical therapists in determining functional abilities and progress during the rehabilitation process. The goal of rehabilitation is to help patients return to their community and work environments through physical, cognitive, emotional, and social recovery. This ensures that patients can function as independently as possible. Physical therapists should actively work within an interdisciplinary team to meet these goals.

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## Disclaimer

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# Cognitive-Communication Rehabilitation after Brain Injuries

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## ABSTRACT

Speech-language pathologists (SLPs) provide communicative and cognitive rehabilitation for people with brain injuries, and this article describes current assessment and treatment after a brain injury. Cognitive problems can affect attention, concentration, and memory, as well as metacognitive skills to self-monitor, control, and adjust one's thinking. Speech-language pathology (SLP) rehabilitation is initiated in acute inpatient settings and then through a continuum of inpatient rehabilitation, skilled nursing facility, home care, and outpatient settings. Speech-language pathology helps adults with brain injury return to work; sports-related concussion care is provided in school settings. SLPs use assessment tools with normative comparisons to determine severity levels and treatment plans. Patient-centered approaches are used for assessment and treatment plans, to identify specific functional needs that may direct the treatment towards specific functional outcomes.

**KEYWORDS:** Speech-language pathology; communicative and cognitive rehabilitation; brain injuries

## INTRODUCTION

In the United States, there are more than 586 Traumatic Brain Injury (TBI)-related hospitalizations and 190 TBI-related deaths per day, according to the Centers for Disease Control and Prevention (CDC).[1] For those who survive a brain injury, rehabilitation and medical care can range from minimal to intensive levels. Depending on the location and severity of the brain injury, an individual's ability to walk, talk, and care for oneself can vary substantially. Mild to severe TBI can impact a person's abilities related to cognition and communication.[1,2] Speech-language pathology (SLP) has served on the forefront of communicative and cognitive rehabilitation for people with brain injuries.[3] This article describes current SLP treatment to restore cognitive-linguistic skills after a brain injury.

## COGNITIVE REHABILITATION

Cognitive problems caused by a brain injury can affect attention, concentration, and memory (especially short-term memory).[3] In addition, an individual's metacognitive abilities and skills can be affected.[4] These cognitive systems are the basis for communication skills with respect to one's ability to comprehend what is spoken as well as the ability to respond with a clear and understandable message. Cognitive-communication impairments are typically assessed and treated by speech-language pathologists (SLPs), who coordinate this aspect of rehabilitation for brain-injured patients.[6] There are several cognitive systems that the speech-language pathologist evaluates and treats, to improve cognition and communication after a brain injury.

Metacognition is described as systems that include the ability to self-monitor, control, and adjust one's thinking through self-awareness and self-regulation. Self-awareness is the ability to know one's own emotions, beliefs, and values, as well as recognize how they are being perceived by others. Self-regulation is the ability to manage one's behaviors and actions in the context of social rules and settings.

## CURRENT PRACTICE PATTERNS

SLP services vary depending on the degree and location of the brain injury.[5] SLP rehabilitation is initiated in acute inpatient settings,[4] and it progresses through a continuum of inpatient rehabilitation, skilled nursing facility, home care, and outpatient settings. Speech pathology serves on the forefront of return-to-work programming among adults with brain injury.[6] With the advent of sports-related concussion care, SLP is also provided in school settings.[6,9,12] Based upon the patient's needs and severity level, the speech-language pathologist determines the most appropriate model of assessment and intervention to achieve the best level of outcomes.

## ASSESSMENT

Multiple factors are considered in selecting specific tests and protocols to assess the cognitive skills of patients with brain injuries.[3,7] SLPs use assessment tools with normative comparisons to determine severity levels and treatment plans. Standardized testing also supports the basis for SLP services for insurance reimbursement. Criterion-referenced assessments for cognitive skills can provide effective measures that identify severity levels and specific systems of

cognitive impairment. Patient-centered approaches are used for assessment and treatment plans, to identify specific functional needs that may direct the treatment towards specific functional outcomes (e.g., financial management, cooking, return to work).

SLPs are a part of interdisciplinary teams, and they play a leadership role for the assessment and treatment of the patient's cognitive skills.[8] It should be noted, however, that other team members also assess the patient's cognitive skills that impact function. For instance, nurses assess cognitive skills related to remembering medications by name, dosage, and purpose. Physical therapists assess cognition in the context of safety with transfers and ambulation. Occupational therapists evaluate cognitive skills to improve safety and function with bathing, dressing, toileting, etc. SLPs collaborate with all disciplines to facilitate continuity of services and a consistent understanding of the patient's function and cognitive impairment. SLP assessment is based on the patient's level of severity as well as needs. Current models of assessment range from standardized testing to interviews and observations.[9]

## SERVICE DELIVERY PATTERNS

Currently, speech-language pathologists utilize a combination of restorative and compensatory treatments.[3] Restorative intervention is based on the premise of returning to baseline function, whereas compensatory treatment focuses on implementing alternate strategies or environmental aids. SLPs may also utilize a mixed-method approach to cognitive-communication treatment for patients with brain injuries. This approach provides the patient with strategies and tools to return to function during the early stages of rehabilitation while also addressing restorative intervention – with the goal of full recovery.[10] SLPs provide treatment based on the patient's needs, severity level, and pre-morbid status (family support, living arrangements, and work or school roles). Treatment models involve collaboration with the interdisciplinary team, including physiatrists, neurologists, neuropsychiatrists, physical and occupational therapists, and nurses. To determine the best treatments, the SLP seeks ongoing feedback and input from the patient, interdisciplinary team, and family. Taking all these factors into consideration, the SLP offers a variety of strategies and tools.

Current service delivery models range from cognitive exercises to family counseling to environmental aids. Spaced retrieval is an evidence-based technique used to build memory skills. [9,11] After introducing information that is recalled within a short timeframe (e.g., 10 seconds), the SLP asks the patient to recall the information in progressively longer timeframes. This technique for building memory skills has been proven to be effective among patients with brain injury as well as other brain impairments (e.g., dementia, aphasia). Cognitive rehabilitation is also delivered through immediate- and short-term memory exercises during face-to-face treatment and computerized programming.[13] The latter approach offers patients the ability to practice independently, which may lead to a faster recovery.[13]

SLPs include the family members in all components of the patient's recovery process.[12] Family members can provide insight into the patient's pre-morbid condition. The family can serve to substantiate or clarify the patient's functional-cognitive status within the home setting. The speech pathologist provides ongoing family education to facilitate continuity of cognitive therapy provided in the clinical setting. Family education will lead to a better understanding of the condition and therapeutic lifestyle changes. Family counseling also addresses the support required to facilitate the patient's progress and independence.

Compensatory strategies are useful in all stages of a patient's recovery process.[6,15] For instance, journal writing is used to improve short-term memory, episodic memory, and semantic memory skills. Placing schedule boards in the patient's living space can improve the ability to recall daily events. Checklists, alarms, and calendars are additional tools that lead towards independence and cognitive-communication recovery.

Byom and others identify social skills as viable goals that facilitate successful recovery for the adult brain-injured patient, for social interactions and return to work.[11,16] Social skills are commonly affected as a result of brain injury. Therefore, SLPs focus on social-pragmatic skills that are linked to social communication, social adjustment, and social cognition abilities. The importance of social-pragmatic skills is based on the premise of the utilization of functional use of language abilities that ultimately promotes communication.

The SLP's role in brain injury rehabilitation includes assessment and treatment across a continuum of clinical settings, as well as within schools for sports-related concussion care.[14] Speech-language pathologists can utilize formal as well as informal assessments that lead towards effective treatment. A variety of service delivery models are available and are based on the patient's needs. Computerized cognitive programs can provide fast recovery due to the increased opportunity for practice.[13] Family integration is an important component of cognitive-communication rehabilitation. The field of SLP continues to strive to support and improve the quality of life for people with brain injuries

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# Current Concepts in Neurogenic Heterotopic Ossification

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## ABSTRACT

Heterotopic ossification (HO) is abnormal bone deposition, most commonly in the hip or elbow, that can significantly impair patients due to pain, stiffness, and contractures, which prevents them from carrying out activities of daily living. A traumatic brain (TBI) or spinal cord injury may prompt the formation of heterotopic ossification, creating so-called neurogenic heterotopic ossification (NHO). The pathophysiology of this condition is not fully understood, but probably involves a complex interplay between a biological scaffold of demineralized bone, CNS signaling, and local tissue signal mediators that often result from trauma. This environment is conducive to HO formation. Diagnosis relies on laboratory testing, plain radiographs, and advanced imaging such as triple phase bone scan or computed tomography scan. Treatment involves medical management with anti-inflammatory drugs, bisphosphonates, radiation therapy, or surgical excision, although outcomes are variable both in regards to prevention and treatment. A mainstay of treatment is physical therapy to maintain range of motion. This paper also presents a case study of a poly-traumatized patient with a TBI and multi-level spinal column injury who developed NHO after acetabular fixation.

**KEYWORDS:** Heterotopic ossification; traumatic brain injury; bisphosphonates; indomethacin; radiation therapy; rehabilitation; physical therapy

## INTRODUCTION

Neurogenic heterotopic ossification (NHO) occurs in the setting of neurological disorders and is characterized by abnormal bone deposition in extraskeletal tissue.[1] While NHO is usually seen after traumatic injury to the brain (TBI) or spinal cord (SCI), it is also associated with disorders such as Guillain-Barre syndrome, cerebral anoxia, stroke, infections, and brain tumors.[2] It has been reported to occur in up to 20% of TBI patients and 30% of SCI patients.[3-5] Additional risk factors for NHO may include male gender, polytrauma, delayed rehabilitation, and prolonged hospital length of stay.[6,7]

Patients with NHO may present with pain, reduced joint range of motion, warmth, and swelling. Typical sites of NHO include the hips, knees, elbows, shoulders, hands, and spine.[8–11] Due to its location and associated symptoms, NHO can cause significant impairment of activities of daily living (ADLs). While the exact mechanisms behind the development of NHO are not completely understood, the complex relationship of traumatic injury, localized and systemic inflammation, and neural regulation are all thought to contribute to its development.[12–14]

This article will review the pathophysiology, diagnosis, and treatment of NHO in the context of a male patient who developed NHO after surgical fixation of a complex left acetabular fracture following a polytraumatic motor vehicle collision.

## PATHOPHYSIOLOGY

The pathophysiology of NHO is not entirely understood, but it is generally recognized as a complex interplay between traumatic injury, local and systemic inflammatory responses, and neuromodulation.[15,16] NHO affects approximately 20% of people with a spinal cord injury or TBI, so it is imperative to understand NHO and its associated morbidity. Generally, HO involves osteogenesis outside the appendicular or axial skeleton and instead within soft tissue (i.e., muscle).[16] It has been described as the formation of benign ectopic bone which undergoes osteogenesis through endochondral rather than intramembranous ossification.[17] Although ectopic bone formation can occur at any extraosseous site, the hip is the most common, followed by the elbow.[18-20] Specifically, the demineralized bone matrix that becomes embedded in muscle will undergo osteogenesis, which is contrary to that in other tissues (i.e., adipose).[15] Herein, we will further explore mediators of the local and systemic inflammatory responses as well as neuromodulatory responses that contribute to extraosseous bone formation.

Perhaps most critical for the development of HO is the biological scaffold that promotes bone formation, an environment that responds to an inducing agent (i.e., trauma) and contains osteogenic precursors.[16] Our understanding of the relationship between the central nervous system (CNS) and bone continues to evolve. Dense innervations of the periosteum provide a mechanistic route by which the CNS can modulate osteogenesis, through neurotransmitters including glutamate, calcitonin gene-related

protein, substance P, and catecholamines. Altogether, these transmitters upregulate osteoblastic activity while down regulating osteoclasts.[16]

Apart from the nervous system as a modulator for HO, local mediators are often further upregulated in the setting of TBI or SCI. Osteoprogenitor cells within skeletal muscle respond to the local environment, specifically to inflammatory mediators that create a hypoxic environment for osteogenesis.[17] Pro-inflammatory cytokines such as transforming growth factor-beta (TGF-β), interleukin-1 (IL-1), interleukin-6 (IL-6), insulin-like growth factor (ILGF), platelet-derived growth factor (PDGF) and fibroblast growth factor (FGF) contribute to the differentiation of osteoprogenitor cells.[1,3] Furthermore, the relatively hypoxic environment contributes to an influx of pro-inflammatory cells, including macrophages, neutrophils, and mast cells; this environment is pro-osteogenic. This leads to a cascade including upregulation of hypoxia inducible factor-1 and endothelial growth factor (FEGF), which stimulate angiogenesis and the migration of osteoprogenitor cells, which are stimulated by FEGF to differentiate into fibroblasts and chondrocytes. Ultimately, this process leads to an up-regulation of SOX-9 and the production of chondrocytes, which begin to form lamellar bone.[17] Overall, at the cellular level there are multiple contributors that lead to the up-regulation of chondrocytes and formation of heterotopic ossification.[15-17]

## DIAGNOSIS

Early NHO can manifest as joint stiffness, decreased range of motion, erythema, swelling, and pain.[3,5] Without clinical suspicion, the diagnosis of early NHO can easily be missed. NHO usually occurs 3–12 weeks after the injury/trauma, but it can take more than six months to present in some cases.[21] Common differential diagnoses that should be ruled out include deep vein thrombosis (DVT), tumor, and septic arthritis.

Laboratory studies can provide cost-effective and important information in the workup of NHO, especially in its early inflammatory phase. Non-specific inflammatory markers such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are often elevated in the inflammatory phase of NHO, though it is important to consider and rule out mimicking inflammatory or infectious pathologies.[22] Alkaline phosphatase and osteocalcin are also associated with NHO, though in a non-specific manner.[23] These markers increase in the first six to 12 weeks after trauma, with serum levels greater than 250 (IU/L) associated with early HO formation.[5,24-26] Additionally, increasing serum creatine kinase may be correlated with more severe disease and resistance to medical therapies, which may help further guide diagnosis and treatment.[3,27]

A venous duplex ultrasound can be ordered to quickly and easily rule out a DVT. Radiographs are specific but not sensitive in the early phase of NHO. Later on, it can be seen as circumferential bone formation at or around a joint. While triple-phase bone scan is the most sensitive test for NHO, as early as 2.5 weeks after injury, it has low specificity.[3] Computed tomography (CT) scans can determine the extent and three-dimensional structure of NHO, which is helpful to prepare for operative intervention but not as useful in diagnosis.[16,28] Magnetic resonance imaging (MRI) reliably detects NHO in a three-dimensional fashion, as early as one to two days after the onset of symptoms, but the specificity is low.[29] Other imaging techniques used are ultrasound and 3-dimensional stereolithography, but they are less popular. Early diagnosis is important because it allows for initiation of interventions that may halt its progression.

## TREATMENT

Treatment varies based on symptom severity and patient-specific risk factors for developing NHO. Studies vary in the reported incidence of asymptomatic NHO; patients without clinical symptoms from early-grade cases may be monitored closely.[30] Radiographic severity may not correlate directly with loss of function or range of motion, though high-grade cases are more likely to cause debilitating symptoms.[31] There is a wide range of treatment options, prophylactic and definitive, varying from NSAIDS and other oral medications to radiation therapy to surgical excision.[32]

The main medical therapy for NHO prophylaxis continues to be non-steroidal anti-inflammatory drugs (NSAIDs), which targets the early inflammatory phase.[30,33] Traditionally, indomethacin is considered the gold standard for prophylaxis following high-risk surgical procedures for the development of NHO. The recommended dose of indomethacin is 75 to 100 mg/day for seven to 14 days postoperatively, with monitoring for side effects such as ulcers, gastritis, or kidney injury.[32] Recent literature has suggested that less potent, nonselective NSAIDs such as ibuprofen or selective COX-2 NSAIDs such as celecoxib may be equally effective, with cost savings and a lower incidence of postoperative bleeding and side effects.[34] Bisphosphonate therapy is also effective in NHO prophylaxis, which can be especially useful in patients with contra-indications to NSAIDs.[35] As early prophylaxis, bisphosphonate regimens such as a three-day IV course of etidronate followed by a six-month oral course have effectively halted progression.[36] Limitations of bisphosphonate treatment include greater costs and treatment duration when compared to NSAIDs.[37] Additionally, bisphosphonates may be ineffective when started in the late stages of NHO (with positive radiographs) and have risks of severe associated side effects.[35]

Radiation therapy is also effective for NHO prophylaxis. This treatment involves the irradiation of pluripotent mesenchymal cells, which are thought to form heterotopic bone.[38] External beam radiation therapy is often prescribed at a 7 to 8

Gy fraction dose and typically given within 24 hours preoperatively or within 72 hours postoperatively.[30] Some studies have found radiation therapy to be superior to NSAIDs in preventing clinically significant NHO, while others report equivocal outcomes.[39,40] At present, there is no consensus on the most effective treatment, so either can be utilized based on provider preference, patient factors, and institutional protocols. It is important to consider radiation side effects such as wound healing delays, joint swelling, bony nonunion, and the rare incidence of secondary malignancy.[30]

Ultimately, high-grade NHO with functional impairment and pain may require surgical excision [**Figure 1A,B,C**]. Excision should be performed after the growth and maturation phases, as confirmed by serial radiographs, which can take over 1.5 years for TBI.[5] Surgeons should weigh the risks of prolonged debilitation and surgical complexity

**Figures.** 37-year-old male with neurogenic heterotopic ossification (NHO) of the left hip after surgical fixation of a complex left acetabular fracture in the setting of a polytraumatic motor vehicle collision with traumatic brain injury.

**Figure 1.** 3-view radiographs of the pelvis including AP [**A**], iliac oblique [**B**] and obturator oblique [**C**] views demonstrating extensive heterotopic ossification formation about the left hip in the setting of a prior posterior column acetabular fracture fixation four months post-operatively.

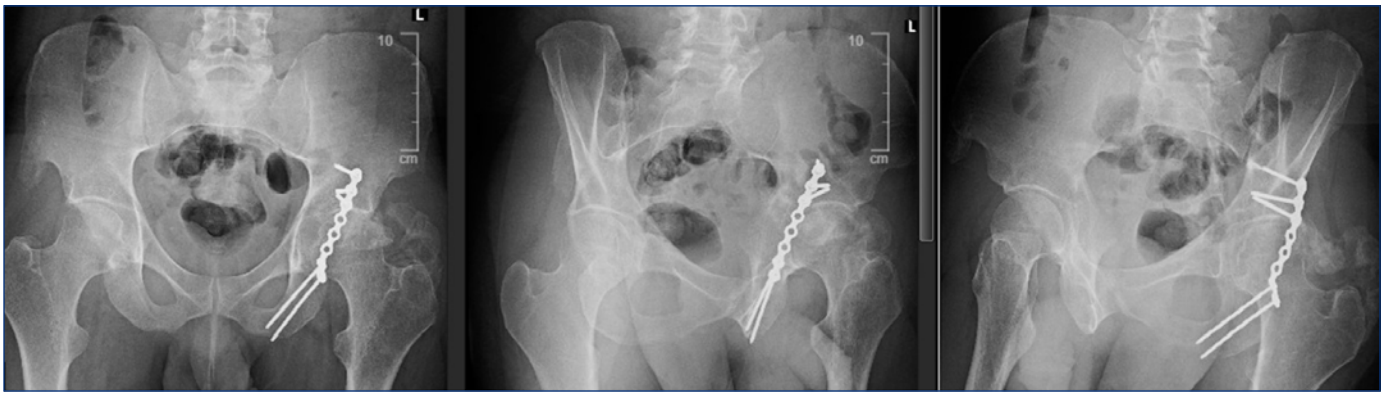



**Figure 2.** Computed tomography axial cuts at level of acetabulum [**A**], femoral head [**B**], greater trochanter [**C**] further characterizing the extent of heterotopic ossification at 4 months post-operatively

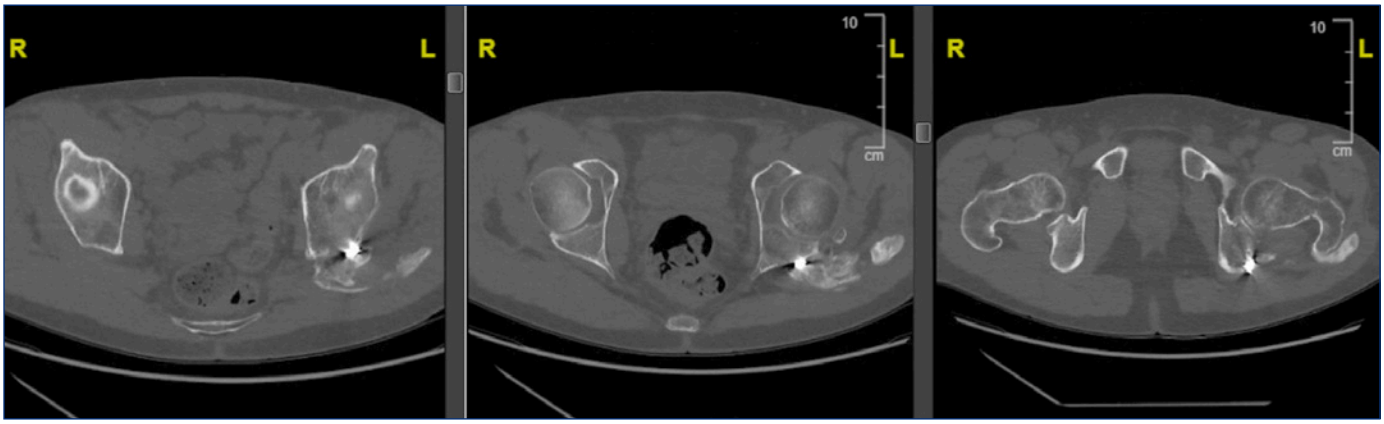
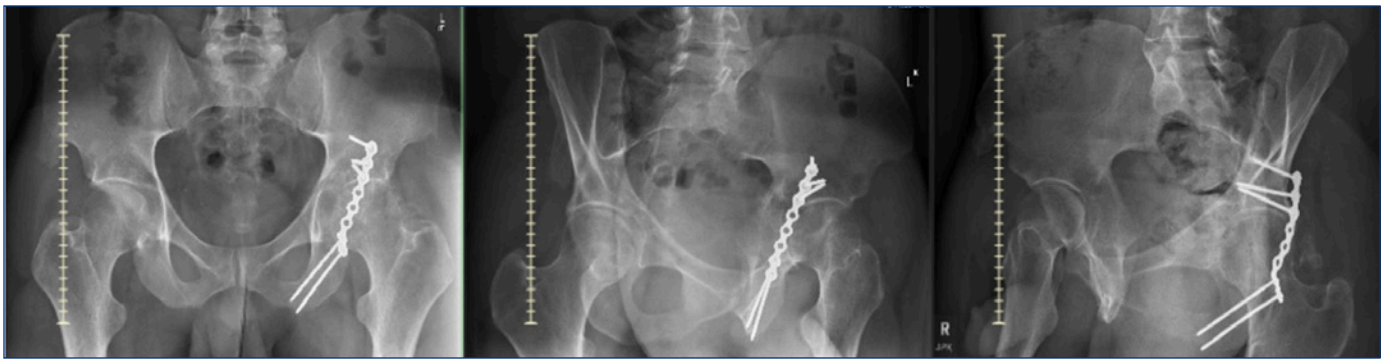



**Figure 3.** 3-view radiographs of the pelvis including AP [**A**], iliac oblique [**B**] and obturator oblique [**C**] at 4 months after surgical excision of prior NHO.



when considering surgical timing. Advanced imaging such as CT may be utilized for surgical planning, to further characterize the extent of heterotopic bone formation [**Figure 2A,B,C**]. Surgical management of NHO is challenging because excision may not fully address clinical symptoms and incomplete resection has recurrence rates as high as 33%.[41] Outcomes in the literature have been variable; many patients have improved pain and range of motion (ROM), but few achieve full resolution without recurrence.[42] While further high-quality investigations are required at this time, surgical excision is indicated in the patients with high-grade NHO and symptoms refractory to nonsurgical management. Additionally, due to high recurrence rates, surgical excision of heterotopic bone should be supplemented with medical treatments.[33] Traditional prophylaxis with NSAIDs, bisphosphonates, and radiotherapy is effective in reducing recurrence rates after surgical excision.[43] The role of additional medical management is further supported by findings in post-operative patients that new foci of ectopic bone are likely due to de novo formation rather than extension of unresected bone.[14] Therefore, the goal of resection, with medical prophylaxis for recurrence, is to improve range of motion while minimizing soft tissue trauma and surgical morbidity [**Figure 3A,B,C**].[44]

Regardless of the stage of NHO or treatment phase, physical therapy (PT) should be implemented throughout the disease course in conjunction with pharmacologic or surgical treatments.[33] While PT has not been shown to independently prevent NHO, range-of-motion exercises are crucial for preserving joint motion and preventing soft tissue contractures.[5] PT regimens may vary by institution and case-specific characteristics; however, early passive and active ROM of restricted joints in a controlled setting is recommended to preserve or optimize function prophylactically and post-treatment.[45,46]

## CONCLUSIONS

Neurogenic heterotopic ossification is a difficult problem to treat in poly-traumatized patients with neurologic injuries due to the significant functional limitations it can place on already debilitated patients. While prevention is effective, no consensus exists on optimal treatment and surgery has variable results. Physical therapy remains an important mainstay of treatment in order to maintain range of motion. Further studies on the pathophysiology of this condition are crucial in order to develop treatment and prevention efforts and minimize the negative impact of NHO on patients.

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## Disclosures

The authors have no disclosures.


**Funding:** None.


**Conflict of Interests:** The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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# An Orthopedic Perspective on the Management of Spasticity

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## ABSTRACT

Neurologic conditions such as brain injuries, cerebral palsy, stroke and multiple sclerosis involve injury of upper motor neurons, which can manifest as spasticity. The resulting hypertonicity and imbalance of forces between muscle groups leads to deformities that impair patient function and can cause significant long-term complications. Symptoms of spasticity can be managed with nonoperative techniques such as physiotherapy, bracing, or medications. Other approaches such as intrathecal baclofen pumps and selective dorsal rhizotomy have also demonstrated efficacy in controlling spasticity. Spasticity that has failed nonoperative management can be treated with orthopedic surgeries that correct deformities by either restoring joint anatomy or re-balancing the forces of spastic muscle groups. Improved mobility and reduced pain after these procedures can help patients with their rehabilitation, function, and independence.


**KEYWORDS:** Orthopedics; spasticity; brain injuries; cerebral palsy; upper motor neuron injury; spinal cord injury (SCI); multiple sclerosis (MS)

## INTRODUCTION

Spasticity is a common clinical sequela of neurologic conditions such as brain injuries, strokes, spinal cord injury (SCI), cerebral palsy (CP), and multiple sclerosis (MS).[1-3] Along with muscle weakness, hyperreflexia, clonus, hypertonicity, and a positive Babinski reflex, it is one of the characterizing signs of upper motor neuron (UMN) lesions.[1] Spasticity encompasses a broad range of presentations, ranging from reducible deformity to permanent contracture or joint instability, and thus, is a difficult condition to define.[2,3] Dressler et al recently revised the definition of spasticity as involuntary muscle hyperactivity – including one or more of rigidity, dystonia, spasms, spasticity, or spasms – in the context of central paresis.[4] The resulting deformities and motor impairments caused by spasticity generate significant physical, psychological, and social burdens for patients.[3] Treatment of the condition through nonoperative or surgical means is a critical component of neurorehabilitation, and can significantly improve quality of life, help patients regain independence, and prevent long-term complications.[5]

## NONOPERATIVE MANAGEMENT

### Physiotherapy

Rehabilitation through modalities such as exercise, cryotherapy, and stretching plays a significant role in managing spasticity, and research suggests that maximum benefit is achieved by early intervention. Physical therapy typically focuses on stretching of spastic muscle groups and strengthening of muscle antagonists to maximize passive and active range of motion.[6] The improved motor control and strength aids in controlling distal movements, thereby improving overall motor function. Exercises that involve weight-bearing have also been shown to increase bone mineral density, improve bowel function, and enhance mobility.[5,7] Physical therapists will also guide and support decision-making around adaptive and assistive devices, based on a patient's abilities and long-term goals for mobility. Furthermore, if a patient undergoes operative management, physical therapy plays a significant role in maximizing postoperative benefits.[8]

### Casting and Bracing

Stretching consistently at home to reinforce physical therapy gains may be challenging for patients and families. Therefore, bracing can be a useful supplement to therapy, with positioning to correct contractures, improve flexibility, and increase range of motion. Bracing may also improve balance, transfers, and ambulation as well as help maintain a comfortable position in a wheelchair. Proper fitting of orthotics is necessary to maximize stretching and positioning benefits while avoiding skin breakdown and irritation.[9]

### Extracorporeal Shockwave Therapy

Extracorporeal shock wave therapy (ESWT) is a procedure for the management of spasticity that can be done in outpatient clinics. While the exact mechanism is unknown, ESWT involves the application of focused, singular acoustic pulses to create pressure waves that induce cellular changes. For patients with spasticity, ESWT has been shown to reduce spasticity and pain while improving range of motion and function.[10] This technique is painless, safe, and non-invasive, with improvements seen as early as after one session. This technique can be combined with other treatments of spasticity, with evidence to suggest that ESWT may enhance the effects of Botulinum neurotoxin (Botox).[11]

**Table 1.** Common medications used for the treatment of spasticity.[12,14]

| Drug Name | Administration | Mechanism | Dosage | Side Effects |
|---|---|---|---|---|
| Baclofen | Oral, Intrathecal | GABA-B Agonist | Maximum 40 mg/day if age less than 8 years<br>Maximum 60 mg/day if age older than 8 years | Sedation, fatigue, constipation, hepatotoxicity, weakness, drowsiness, withdrawal, seizures, altered mental status |
| Benzodiazepines | Oral, intravenous | GABA-A Agonist | For Diazepam, 6 months and older 1–2.5 mg orally 3-4 times daily | Sedation, fatigue, constipation, confusion, respiratory depression, dependency, withdrawal |
| Clonidine/ Tizanidine | Oral | Alpha-2 Agonist | **Clonidine:** 0.02 ± 0.03 mg/kg/day<br>**Tizanidine:** Children aged > 2 years, 0.3–0.5 mg/kg/day in 4 divided doses | Hypotension, bradycardia, muscle weakness, sedation, xerostomia, hallucinations, QT interval prolongation |
| Gabapentin | Oral | Alpha-$2_{\delta 1}$ subunit binding to inhibit Ca2+ currents | **Infants:** 5 mg/kg/day, titrate by adding up to 3x day or increasing dose<br>**3–11 years:** 10–15 mg/kg/day in 3 divided doses<br>**>11 years:** 300 mg 3 times daily, titrate up to 3600 mg/day | Somnolence, tremor, nystagmus, mood changes such as anxiety or aggression, fatigue, weakness, nausea/vomiting, headache, challenges with concentration |
| Dantrolene | Oral | Inhibition of Ca+ release from sarcoplasmic reticulum to prevent muscle contraction | 0.5 mg/kg once daily for 7 days, then 0.5 mg/kg 3 times a day for 7 days, then 1 mg/kg 3 times a day for 7 days, then 2 mg/kg 3 times day<br>Maximum 100 mg 4x per day | Liver failure, weakness, fatality associated with high dosing |
| Botulinum Toxin | Injection | Acetylcholine release inhibition | **Botox:** 8 u/kg or 300 u total for lower limbs<br>10 u/kg or 340 u for total body | Dry mouth, dysphagia with use in upper limbs/neck muscles, double vision, weakness, respiratory difficulties, spread to other muscles, resistance with increased usage |
| Phenol/Alcohol | Injection | Chemical Neurolysis | **Phenol:** concentration of 3–7% (50mg/mL-70 mg/mL), pediatric dosing 30mg/kg<br>**Alcohol:** concentration typically 40–50% | Flushing, cardiac dysrhythmia neuropathic pain, paresthesia, bowel/ bladder incontinence, sexual dysfunction, muscle fibrosis, vascular sclerosis |

### Medications

Several oral medications can be used in the management of spasticity. These are usually GABA receptor agonists, though alpha-2 agonists, gabapentin, and dantrolene are also helpful (**Table 1**). Injectable medications such as Botox, alcohol, or phenol can also achieve a more targeted effect on spastic muscle groups. The effectiveness of these medications can decrease over time, so using them in combination with other medications and maximizing the time between injections may prolong their effectiveness.[12] Cannabis is also an increasingly popular treatment option for pain control in spasticity, particularly for those with MS.[13] The benefit of cannabis to treat spasticity in other neurologic conditions or in pediatric conditions is less well-established.

## PROCEDURAL MANAGEMENT

### Intrathecal Baclofen

Intrathecal baclofen is useful in reducing spasticity while avoiding systemic adverse effects of oral medications such as sedation, or in patients who do not benefit from the maximum oral dose.[15] Intrathecal baclofen can reduce pain and improve general functional status, notably with gait and mobility.[16] Patients should have a trial injection of intrathecal baclofen to determine its efficacy. Following implantation of the pump, dosing can be titrated to achieve the best muscle tone for a patient's comfort and functional ability. Complications of pump placement include infection and catheter obstruction. The pump must be refilled, but the time between refills can be increased with gradual increases in the medication concentration.[17]

### Selective Dorsal Rhizotomy

Selective dorsal rhizotomy (SDR) is a procedure that involves sectioning afferent nerve rootlets that innervate affected muscle groups, which reduces the exaggerated stretch reflexes of spasticity, leading to improvements in gross motor function and gait.[18] Additionally, patients who undergo SDR may be less prone to long-term complications of spasticity and thus require fewer surgeries.[19] The results of SDR also appear to be long-lasting; at 20-year follow-up, 91% of respondents reported that the procedure improved their quality of life and 88% said they would recommend the procedure to others.[20] Proper patient selection for this

procedure is crucial to maximizing outcomes. Patients who benefit most from the procedure are ambulatory with primarily lower extremity involvement, have good core and abdominal strength to support the trunk while walking, and the cognitive function and resources to participate in intensive therapy postoperatively.

## ORTHOPEDIC SURGERIES FOR SPASTICITY

Orthopedic procedures for the treatment of spasticity can be grouped into the categories of tendon transfer, tendon lengthening or release, osteotomy, and arthrodesis.[3,5] Indications for surgery vary depending on the patient's age and goals, but they generally include fixed contractures, joint deformity, or joint dislocation that affects function, impairs hygiene, or causes significant pain.[2,21] Surgery aims to address deformities by stabilizing the contracted joint or correcting the muscle strength imbalance. Van Heest et al showed that children with spastic upper extremities who were suitable candidates for tendon transfers had greater improvement in function than repeated botulinum toxin injections or regular therapy sessions at 12 months of follow-up; this suggests that the surgical management of spasticity improves quality of life, provided that patients are suitable candidates for a procedure.[22]

### Upper extremity

The shoulder's positioning in patients with spasticity is due to the unbalanced forces of the internal rotators: latissimus dorsi (LD), teres major (TM), pectoralis major (PM), and subscapularis.[3] For non-functional shoulders, complete release of the subscapularis, LD, and PM tendon insertions, while preserving the shoulder joint capsule, improved passive range of motion in flexion, extension, abduction, and external rotation. For patients with remaining shoulder function or some voluntary movement, fractional tendon lengthening of hypertonic muscle groups (PM, LD, and TM), is the preferred surgical procedure.[3,23] In shoulders with severe internal rotation and chronic posterior dislocation, proximal humerus derotational osteotomy helps to stabilize the joint and increase range of motion, especially in younger patients.[6,24]

Surgical treatment of elbow flexion deformities depends on their angles. Fixed contractures of less than 45 degrees are treated with fractional tendon lengthening of the elbow flexors, while those with worse contractures may require total tendon releases.[23] Fractional tendon lengthening involves the Z-lengthening approach, in which a tendon is split longitudinally and one of the resulting tendon limbs is reflected.[23,25] Both fractional tendon lengthening and complete tendon release of elbow flexors will increase range of motion and decrease pain in patients with spasticity.[23]

Contributions by the pronator teres and pronator quadratus make forearm pronation deformities far more common than supination ones.[26] Surgical correction is done via tendon lengthening of the pronator teres; full tendon release is done for patients without volitional activity of the muscle. Similarly, the pronator quadratus can also be partially or fully transected, depending on forearm function; in most cases, lengthening of the pronator teres is sufficient to improve supination.[5,26] In the rare case of spastic supination, the biceps tendon can be rerouted distally to wrap around the radius in order to restore the forearm to a neutral position.[26] Transfer of the flexor carpi ulnaris (FCU) dorsally to the extensor carpi radialis brevis (ECRB) or extensor digitorum communis muscles is often combined with pronator release or transfer to increase supination in patients with concomitant ulnar deviation deformities.

Flexed wrist, clenched fist, and thumb-in-palm deformity are classic deformities from spasticity, caused by hyperactivity of flexor muscles.[23,27] The most common procedure for surgical correction of wrist deformity is FCU to ECRB tendon transfer, which allows for increased extension and decreased ulnar deviation.[23] The tendon transfer can be passed ulnarly to aid in supination or radially to aid in pronation.[27] In patients with functional upper extremities, fractional lengthening of the flexor digitorum profundus (FDP), flexor digitorum superficialis (FDS), or flexor carpi radialis (FDR) via Z-lengthening can also improve hand function.[23,27] Patients who are skeletally mature can also have total wrist fusion for a severe flexion contracture [**Figure 1**].[6] Thumb-in-palm deformity can be addressed in a variety of surgical options, including tenotomy of the adductors, lengthening of the flexor pollicis longus (FPL), Z-plasty of the first web space, or tendon transfers of the extensors.[6] The surgical procedure depends on the specific tendinous structures that are abnormal and whether the first web space in implicated in the deformity.[27]

**Figure 1.** Twenty-year-old woman with left wrist contracture and a painful bunion deformity on left foot due to spasticity related to an intracranial bleed at age eight. [Radiographs are courtesy of Dr. Craig Eberson.]

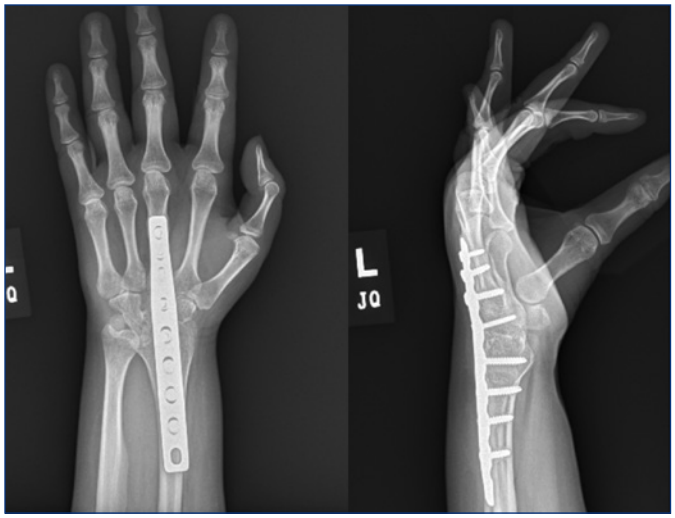
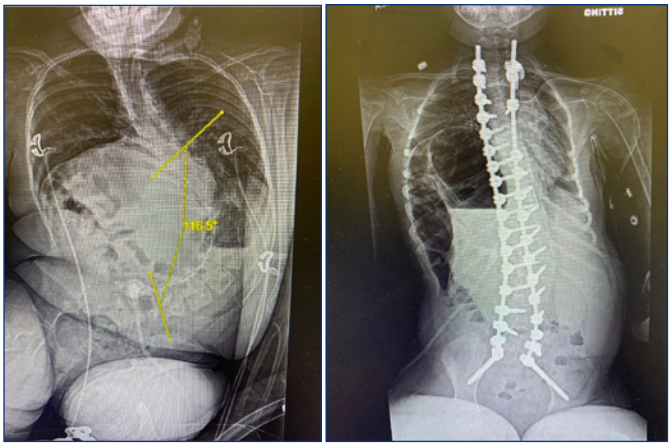


**Figure 2. [A]** Preoperative radiograph of a patient with severe neuromuscular spinal deformity secondary to cerebral palsy. **[B]** Postoperative radiograph after posterior spinal fusion from T2 to the pelvis, improving pulmonary function and allowing this patient to sit more comfortably.



## Spine

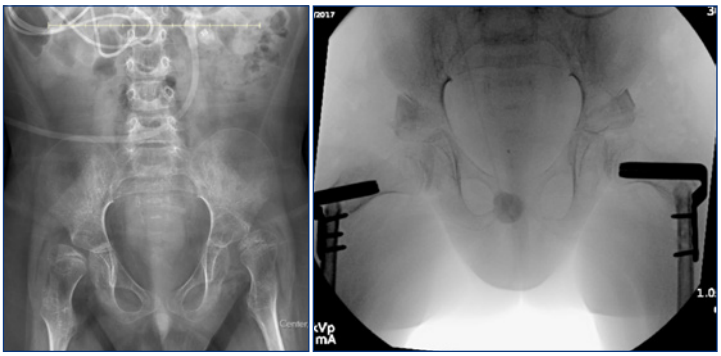
Cerebral palsy (CP) is the leading cause of neuromuscular scoliosis (NMS).[28] The progression of scoliosis curvature in CP patients can be rapid, particularly in those that are non-ambulatory, due to persistent muscle weakness, imbalance of forces, and decreasing bone density.[29] The resulting severe spinal deformity [**Figure 2A**] can cause adverse sequelae such as respiratory or cardiac compromise, pelvic obliquity, skin infections, poor nutritional status, and pain.[28,30] Nonoperative management with spinal bracing can delay surgical intervention, but has less efficacy in halting curve progression than in patients with adolescent idiopathic scoliosis.[29] Indications for surgery, traditionally a posterior spinal fusion, vary depending on the etiology of the deformity and patient circumstances, but is usually recommended in patients with progressing curves that are impacting balance or positioning with sitting or standing.[29,30] The extent of the spinal deformity determines the levels of spinal fusion; long fusions extending from T2 to the pelvis may be necessary in non-ambulatory children with CP [**Figure 2B**].[29,30] Posterior spinal fusion was previously combined with anterior procedures in patients with rigid spinal deformities or severe pelvic obliquity, but advances in implants and increased usage of traction devices have decreased the necessity of the combined anterior and posterior approach.[30] While surgical correction of neuromuscular scoliosis significantly improves quality of life, it also entails the highest surgical complication rate among all types of scoliosis. Seaver et al demonstrated a 10-year reoperation rate of 21.7%, most commonly due to implant failure and surgical site infections, in children with non-ambulatory CP who underwent posterior spinal fusion.[29,31] This is particularly notable as children who undergo posterior spinal fusion carry the implants within them as they transition to adulthood.[28] Thus, the final decision to pursue surgery requires detailed discussion of risks and benefits between providers and families in order to arrive at the optimal treatment plan.

## Hip

Patients with spasticity are prone to hip abnormalities such as acetabular dysplasia, coxa valga, and femoral anteversion. Hypertonic hip flexors and adductors overpower the weaker hip extensors and abductors, shifting the center of rotation of the hip from the femoral head to the lesser trochanter and leading to pathologic remodeling of the femoral head and acetabulum.[32] Untreated deformities can cause subluxation and eventual dislocation of the hip (**Figure 3A,B**), along with early osteoarthritis due to the loss of joint integrity.[33]

Hip dislocation often progresses silently, especially in non-ambulatory children with CP. As a significant percentage of these patients are intellectually disabled or nonverbal, it is challenging for parents to identify hip pathology from observation of patients alone. Periodic surveillance with both clinical and radiographic examination is recommended, accompanied by hip joint measurements such as the Reimer migration index (RMI) and the acetabular index (AI) to allow for prompt recognition and surgical intervention.[33] In children with cerebral palsy, orthopedic surgeries of the spastic hip can be categorized into preventive, reconstructive, and salvage procedures, with differing goals, indications, techniques, and success rates.[34] (**Table 2**)

**Figure 3.** Eight-year-old male patient with spasticity and hip subluxation after spinal cord injury and traumatic brain injury at age one. [**A**] Preoperative radiograph demonstrating bilateral hip dislocation and deformity. [**B**] Postoperative radiograph after soft tissue release and femoral and pelvic lengthening for hip relocation. The operation reduced the difficulties the patient had with posture and sitting.



## FOOT AND ANKLE

Equinovarus is the most common foot and ankle deformity seen in patients with strokes and cerebral palsy.[35] Other deformities in patients with spasticity include plano-valgus, toe flexion, and equinovalgus. Surgical management is recommended for patients who are older or unresponsive to

**Table 2.** Goals, indications, and descriptions of preventive, reconstructive, and salvage procedures in the correction of hip deformities.[2,34]

| | Goals | Indications | Techniques | Success |
|---|---|---|---|---|
| Preventive | Delay hip subluxation or dislocation | 30% ≤ RMI < 60%<br>Limited hip abduction (<30°) | **Soft tissue procedures:** adductor tenotomy, iliopsoas release, semitendinosus lengthening | Dependent on pre-operative severity of hip abnormality. Increased RMI associated with decreased success. |
| Reconstructive | Maintain or rebuild a located hip with preserved mobility | RMI > 40% | **Bony procedures:**<br>**Femur:** proximal femoral varus derotation osteotomy<br>**Acetabulum:** Osteotomies (Dega, Periacetabular, Pemberton, Pericapsular, Chiari pelvic, Salter innominate) | Positive outcomes after long follow-up. Complications include dislocation recurrence, osteonecrosis of femoral head, heterotopic ossification. |
| Salvage | Reduce pain or increase abduction to maintain hygiene when hip cannot be reconstructed to be mobile or located anymore | Severe dysplasia and degenerative changes that are beyond the scope of reconstructive surgeries | **Resection:** Castle procedure (remove proximal femur, reattach rectus and vastus lateralis to femoral shaft, redirect gluteal muscles to between femur and acetabulum)<br>**Redirection:** subtrochanteric valgus osteotomy<br>**Arthroplasty:** total hip replacement | Inferior outcomes to reconstructive procedures due to high rates of complications. |

nonoperative approaches, and the procedure depends on the severity and degree of involvement of specific muscle groups. Hoke lengthening (percutaneous triple hemi-section tenotomy) can be performed for patients with an Achilles tendon contracture.[2] For equinus deformities that are dynamic and combined with other abnormalities, gastrocnemius-soleus lengthening can be considered as well. Spastic equinovarus can also be approached with the split anterior tibialis tendon transfer (SPLATT) procedure, a rerouting of half of the tibialis anterior, which has demonstrated marked success in correcting the varus part of the deformity. Alternatively, fractional lengthening of the posterior tibialis tendon and transfers of the flexor hallucis longus and flexor digitorum longus may be done for varus correction.[35] In children, intramuscular lengthening of the posterior tibial tendon, Achilles lengthening, and SPLATT or split posterior tibial tendon transfer (SPTTT) anterior to the Achilles tendon into the peroneus brevis tendon are all effective procedures to reduce equinovarus deformities that are flexible. In more rigid deformities, osteotomy of the midfoot/calcaneus or triple arthrodesis (in skeletally mature patients) may be needed.

## CONCLUSION

Spasticity results from many neurologic conditions and poses a significant challenge to the independence and quality of life of patients. Depending on the goals of care and patient function, a variety of combinations of nonoperative and operative approaches can be utilized for managing this condition. Orthopedic surgeries that treat spasticity aim to restore mobility, decrease long-term complications, and reduce pain, thereby improving the quality of life for these patients.

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