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Traumatic Brain Injury (TBI) Rehabilitation

JON A. MUKAND, MD, PhD

GUEST EDITOR



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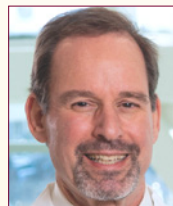
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The Complexities of Traumatic Brain Injuries

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.

References

1. Whyte J, Nordenbo AM, Kalmar K, Merges B, Bagiella E, Chang H, Yablon S, Cho S, Hammond F, Khademi A, Giacino J. Medical complications during inpatient rehabilitation among patients with traumatic disorders of consciousness. *Arch Phys Med Rehabil*. 2013 Oct;94(10):1877-83. PMID: 23735519.

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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 System² 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 Glasgow 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.

References

- Centers for Disease Control and Prevention. Traumatic Brain Injury and Concussion 2024 [updated 29 October 2024. Available from: <https://www.cdc.gov/traumatic-brain-injury/data-research/index.html>.
- Malec JE, Brown AW, Leibson CL, Flaada JT, Mandrekar JN, Diehl NN, et al. The Mayo classification system for traumatic brain injury severity. *J Neurotrauma*. 2007;24(9):1417-24. PMID: 17892404
- Lulla A, Lumba-Brown A, Totten AM, Maher PJ, Badjatia N, Bell R, et al. Prehospital Guidelines for the Management of Traumatic Brain Injury – 3rd Edition. *Prehosp Emerg Care*. 2023;27(5):507-38. PMID: 37079803
- ATLS Subcommittee; American College of Surgeons' Committee on Trauma; International ATLS working group. Advanced trauma life support (ATLS®): the ninth edition. The journal of trauma and acute care surgery. 2013;74(5):1363-6. PMID: 23609291
- Carney N, Totten AM, O'Reilly C, Ullman JS, Hawryluk GW, Bell MJ, et al. Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition. *Neurosurgery*. 2017;80(1):6-15. PMID: 27654000
- Cooper DJ, Rosenfeld JV, Murray L, Arabi YM, Davies AR, D'Urso P, et al. Decompressive Craniectomy in Diffuse Traumatic Brain Injury. *New England Journal of Medicine*. 2011;364(16):1493-502. PMID: 21434843
- Hutchinson PJ, Kolias AG, Timofeev IS, Corteen EA, Czosnyka M, Timothy J, et al. Trial of Decompressive Craniectomy for Traumatic Intracranial Hypertension. *New England Journal of Medicine*. 2016;375(12):1119-30. PMID: 27602507
- Arrich J, Holzer M, Havel C, Müllner M, Herkner H. Hypothermia for neuroprotection in adults after cardiopulmonary resuscitation. *Cochrane Database Syst Rev*. 2016;2(2):Cd004128. PMID: 26878327
- Griesdale DE, McEwen J, Kurth T, Chittock DR. External ventricular drains and mortality in patients with severe traumatic brain injury. *Can J Neurol Sci*. 2010;37(1):43-8. PMID: 26878327
- Nwachuku EL, Puccio AM, Fetzick A, Scruggs B, Chang YF, Shutter LA, et al. Intermittent versus continuous cerebrospinal fluid drainage management in adult severe traumatic brain injury: assessment of intracranial pressure burden. *Neurocrit Care*. 2014;20(1):49-53. PMID: 23943318
- François B, Cariou A, Clere-Jehl R, Dequin P-F, Renon-Carron F, Daix T, et al. Prevention of Early Ventilator-Associated Pneumonia after Cardiac Arrest. *New England Journal of Medicine*. 2019;381(19):1831-42. PMID: 31693806
- Dahyot-Fizelier C, Lasocki S, Kerforne T, Perrigault PF, Geeraerts T, Asehnoune K, et al. Ceftriaxone to prevent early ventilator-associated pneumonia in patients with acute brain injury: a multicentre, randomised, double-blind, placebo-controlled, assessor-masked superiority trial. *Lancet Respir Med*. 2024;12(5):375-85. PMID: 38262428
- Pease M, Mittal A, Merkaj S, Okonkwo DO, Gonzalez-Martinez JA, Elmer J, et al. Early Seizure Prophylaxis in Mild and Moderate Traumatic Brain Injury: A Systematic Review and Meta-Analysis. *JAMA Neurology*. 2024;81(5):507-14. PMID: 38587858
- Temkin NR, Dikmen SS, Wilensky AJ, Keihm J, Chabal S, Winn HR. A randomized, double-blind study of phenytoin for the prevention of post-traumatic seizures. *N Engl J Med*. 1990;323(8):497-502. PMID: 2115976
- Pingue V, Mele C, Nardone A. Post-traumatic seizures and antiepileptic therapy as predictors of the functional outcome in patients with traumatic brain injury. *Sci Rep*. 2021;11(1):4708. PMID: 33633297
- Casault C, Couillard P, Kromm J, Rosenthal E, Kramer A, Brindley P. Multimodal brain monitoring following traumatic brain injury: A primer for intensive care practitioners. *J Intensive Care Soc*. 2022;23(2):191-202. PMID: 35615230
- Chesnut RM, Temkin N, Carney N, Dikmen S, Rondina C, Viddetta W, et al. A trial of intracranial-pressure monitoring in traumatic brain injury. *N Engl J Med*. 2012;367(26):2471-81. PMID: 23234472
- Algattas H, Huang JH. Traumatic Brain Injury pathophysiology and treatments: early, intermediate, and late phases post-injury. *Int J Mol Sci*. 2013;15(1):309-41. PMID: 24381049
- Golub VM, Reddy DS. Post-Traumatic Epilepsy and Comorbidities: Advanced Models, Molecular Mechanisms, Biomarkers, and Novel Therapeutic Interventions. *Pharmacol Rev*. 2022;74(2):387-438. PMID: 35302046
- Wada T, Shiraishi A, Gando S, Yamakawa K, Fujishima S, Saitoh D, et al. Pathophysiology of Coagulopathy Induced by Traumatic Brain Injury Is Identical to That of Disseminated Intravascular Coagulation With Hyperfibrinolysis. *Front Med (Lausanne)*. 2021;8:767637. PMID: 34869481
- Böhm JK, Güting H, Thorn S, Schäfer N, Rambach V, Schöchl H, et al. Global Characterisation of Coagulopathy in Isolated Traumatic Brain Injury (iTBI): A CENTER-TBI Analysis. *Neurocrit Care*. 2021;35(1):184-96. PMID: 33306177
- Davison DL, Terek M, Chawla LS. Neurogenic pulmonary edema. *Crit Care*. 2012;16(2):212. PMID: 22429697
- Young VM, Hill JR, Patrini M, Negrini S, Arienti C. Overview of Cochrane Systematic Reviews of Rehabilitation Interventions for Persons with Traumatic Brain Injury: A Mapping Synthesis. *J Clin Med*. 2022;11(10). PMID: 35628818
- Harbinson M, Zarshenas S, Cullen NK. Long-Term Functional and Psychosocial Outcomes After Hypoxic-Ischemic Brain Injury: A Case-Controlled Comparison to Traumatic Brain Injury. *Pm r*. 2017;9(12):1200-7. PMID: 28512065
- Howe EI, Zeldovich M, Andelic N, von Steinbuechel N, Fure SCR, Borgen IMH, et al. Rehabilitation and outcomes after complicated vs uncomplicated mild TBI: results from the CENTER-TBI study. *BMC Health Serv Res*. 2022;22(1):1536. PMID: 36527074
- Horn SD, Corrigan JD, Beaulieu CL, Bogner J, Barrett RS, Giuffrida CG, et al. Traumatic Brain Injury Patient, Injury, Therapy, and Ancillary Treatments Associated With Outcomes at Discharge and 9 Months Postdischarge. *Arch Phys Med Rehabil*. 2015;96(8 Suppl):S304-29. PMID: 26212406
- Zarshenas S, Colantonio A, Horn SD, Jaglal S, Cullen N. Cognitive and Motor Recovery and Predictors of Long-Term Outcome in Patients With Traumatic Brain Injury. *Arch Phys Med Rehabil*. 2019;100(7):1274-82. PMID: 30605639
- Hoffman R, Spencer E, Steel J. A qualitative exploration of speech-language pathologists' approaches in treating spoken discourse post-traumatic brain injury. *Int J Lang Commun Disord*. 2024;59(2):608-22. PMID: 36918757
- Singh R, Dawson J, Mason PS, Lecky F. What are the functional consequences after TBI? The SHEFBIT cohort experience. *Brain Inj*. 2021;35(12-13):1630-6. PMID: 34711118
- Wilson L, Boase K, Nelson LD, Temkin NR, Giacino JT, Markowitz AJ, et al. A Manual for the Glasgow Outcome Scale-Extended Interview. *J Neurotrauma*. 2021;38(17):2435-46. PMID: 33740873
- Wilson CD, Burks JD, Rodgers RB, Evans RM, Bakare AA, Safavi-Abbasi S. Early and Late Posttraumatic Epilepsy in the Setting of Traumatic Brain Injury: A Meta-analysis and Review of Antiepileptic Management. *World Neurosurg*. 2018;110:e901-e6. PMID: 29196247

32. Thompson K, Pohlmann-Eden B, Campbell LA, Abel H. Pharmacological treatments for preventing epilepsy following traumatic head injury. *Cochrane Database Syst Rev.* 2015; 2015(8):Cd009900. PMID: 26259048
33. Feinberg C, Carr C, Zemek R, Yeates KO, Master C, Schneider K, et al. Association of Pharmacological Interventions With Symptom Burden Reduction in Patients With Mild Traumatic Brain Injury: A Systematic Review. *JAMA Neurol.* 2021;78(5):596-608. PMID: 33464290
34. Giacino JT, Whyte J, Bagiella E, Kalmar K, Childs N, Khademi A, et al. Placebo-Controlled Trial of Amantadine for Severe Traumatic Brain Injury. *New England Journal of Medicine.* 2012;366(9):819-26. PMID: 22375973
35. Dams-O'Connor K, Juengst SB, Bogner J, Chiaravalloti ND, Corrigan JD, Giacino JT, et al. Traumatic brain injury as a chronic disease: insights from the United States Traumatic Brain Injury Model Systems Research Program. *Lancet Neurol.* 2023;22(6):517-28. PMID: 37086742
36. Ogonah MGT, Botchway S, Yu R, Schofield PW, Fazel S. An umbrella review of health outcomes following traumatic brain injury. *Nature Mental Health.* 2025;3(1):83-91. PMID: 39802934
37. Huang Y-q, Wu Z, Lin S, Chen X-r. The benefits of rehabilitation exercise in improving chronic traumatic encephalopathy: recent advances and future perspectives. *Molecular Medicine.* 2023;29(1):131. PMID: 37740180.
38. Hearn R, Selfe J, Cordero MI, Dobbin N. The effects of active rehabilitation on symptoms associated with tau pathology: An umbrella review. Implications for chronic traumatic encephalopathy symptom management. *PLoS One.* 2022;17(7):e0271213. PMID: 35862387.

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Common Sequelae of Severe Traumatic Brain Injury: A Case Report

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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].

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 indomethacin² 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 1. Non-Contrast CT Brain demonstrating Intraparenchymal hemorrhage in the bilateral medial frontal lobe and parafalcine subarachnoid hemorrhage.

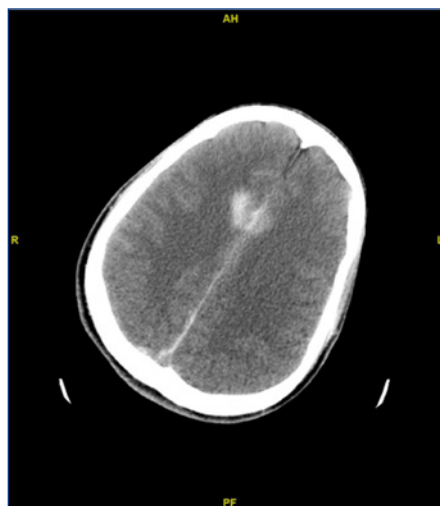


Figure 2. MRI brain demonstrating FLAIR hyperintensity

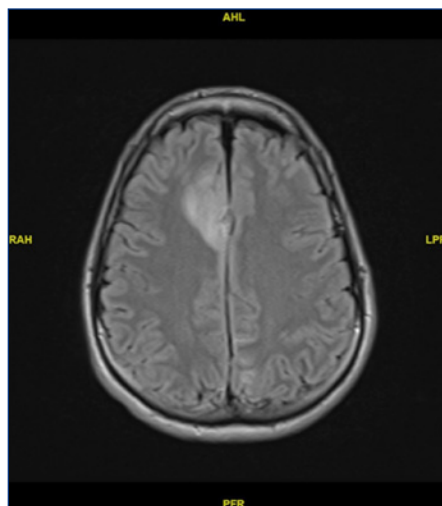


Figure 3. Intraparenchymal hemorrhage (SWI sequence which highlights areas of hemosiderin deposition) in the right medial frontal lobe.

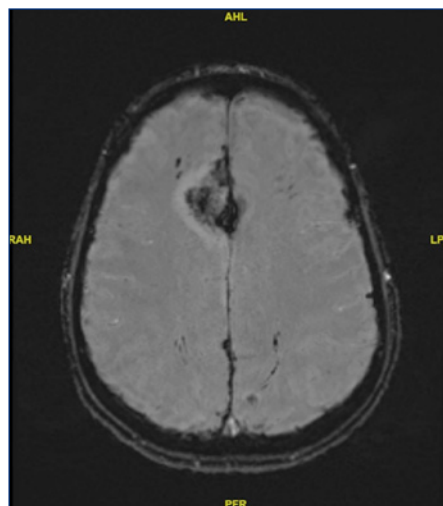
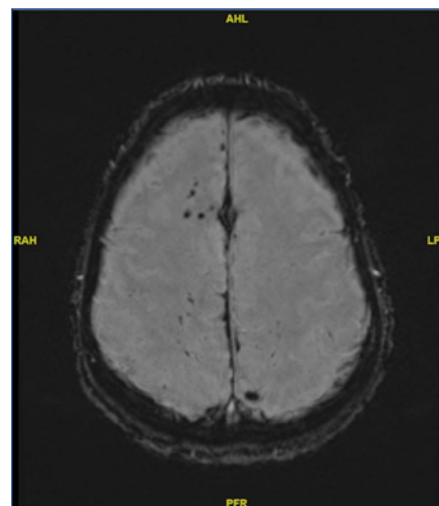


Figure 4. SWI MRI images with microhemorrhages in the bilateral cerebral hemispheres, consistent with diffuse axonal injury.



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

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



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.

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 dif-

ferent 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.

References

1. Giacino JT, Whyte J, Bagiella E, Kalmar K, Childs N, Khademi A, Eifert B, Long D, Katz DI, Cho S, Yablon SA. Placebo-controlled trial of amantadine for severe traumatic brain injury. *New England Journal of Medicine*. 2012 Mar 1;366(9):819-26.
2. Moreta J, Martínez-de los Mozos JL. Heterotopic ossification after traumatic brain injury. *Traumatic brain injury*. 2014 Feb 19;331-49.
3. Garland DE, Blum CE, Waters RL. Periarticular heterotopic ossification in head-injured adults. Incidence and location. *The Journal of Bone & Joint Surgery*. 1980 Oct 1;62(7):1143-6.
4. Smith DH, Meaney DF, Shull WH. Diffuse axonal injury in head trauma. *The Journal of head trauma rehabilitation*. 2003 Jul 1;18(4):307-16.

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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 sequelae 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.

References

1. Golden K, Borsi L, Sterling A, Giacino JT. Recovery after moderate to severe TBI and factors influencing functional outcome: What you need to know. *J Trauma Acute Care Surg.* 2024;97(3):343-355. DOI:10.1097/TA.0000000000004305
2. Nehra D, Nixon ZA, Lengenfelder C, Bulger EM, Cuschieri J, Maier RV, Arbabi S. Acute Rehabilitation after Trauma: Does it Really Matter?. *Journal of the American College of Surgeons.* December 2016;223(6):755-763. DOI:10.1016/j.jamcollsurg.2016.09.001
3. Bayley MT, Janzen S, Harnett A, et al. INCOG 2.0 Guidelines for Cognitive Rehabilitation Following Traumatic Brain Injury: Methods, Overview, and Principles. *J Head Trauma Rehabil.* 2023;38(1):7-23. DOI:10.1097/HTR.0000000000000838
4. Hintze T, Small CE, Montgomery J, Reveles KR, Hafeez S, Barthol CA. Comparison of Amantadine, Modafinil, and Standard of Care in the Acute Treatment of Disorders of Consciousness After Severe Traumatic Brain Injury. *Clinical Neuropharmacology.* 1/2 2022; 45(1):1-6. DOI: 10.1097/WNF.0000000000000487

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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: $((\text{dual task performance} - \text{single task performance}) / \text{single task performance} \times 100)$. 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.

References

1. Basford JR, Chou L, Kaufman KR, Brey RH, Walker A, Malec JF, Moessner AM. Therapeutic exercise during rehabilitation after traumatic brain injury. *J Head Trauma Rehabil.* 2003 Jan-Feb;18(1):76-87. PMID: 12544447.
2. Joyce K, Peters A, Jayaraman A. Balance, gait, and dual-task impairments in individuals with chronic TBI: A scoping review. *NeuroRehabilitation.* 2022;50(1):51-66. PMID: 35099435.
3. McIsaac TL, Lamberg EM, Muratori LM. Building a framework for a dual task taxonomy. *Biomed Res Int.* 2015;2015:591475. PMID: 25705628.
4. McCulloch K, de Joya AL, Hays K, et al. Outcome measures for persons with moderate to severe TBI: Recommendations from the Academy of Neurologic Physical Therapy. *J Neurol Phys Ther.* 2016 Jul;40(3):174-180. PMID: 27388073.
5. Shaffer SW, Harrison AL. Aging of the somatosensory system: A translational perspective. *Phys Ther.* 2007 Feb;87(2):193-207. PMID: 17261565.
6. Alashram AR, Padua E, Annino G. Effectiveness of balance training with virtual reality for patients with traumatic brain injury: A systematic review and meta-analysis. *J Clin Neurosci.* 2022 Mar;95:151-157. PMID: 34923288.
7. Moore JL, Potter K, Blankshain K, Kaplan SL, O'Dwyer LC, Sullivan JE. A core set of outcome measures for adults with neurologic conditions undergoing rehabilitation: A clinical practice guideline. *J Neurol Phys Ther.* 2018 Jan;42(3):174-220. PMID: 29957641.
8. Howe JA, Inness EL, Venturini A, Williams JI, Verrier MC. The Community Balance and Mobility Scale: a balance measure for individuals with traumatic brain injury. *Clin Rehabil.* 2006;20:885-95. doi: 10.1177/0269215506072183
9. Williams G, Pallant J, Greenwood K. Further development of the High-level Mobility Assessment Tool (HiMAT). *Brain Inj.* 2010;24(7-8):1027-1031. doi:10.3109/02699052.2010.490517
10. Jung J, Manosh-Zuniga D, Shapiro S. Cognitive-motor interference during dual-task gait and balance in individuals with traumatic brain injury: A review. *Brain Inj.* 2021;35(6):695-707. PMID: 33945320.
11. de Aquino Costa Sousa T, McIsaac TL, Lamontagne A. Gait and balance under dual-task in individuals with traumatic brain injury: A review. *Brain Inj.* 2022;36(9):1102-1115. PMID: 35935210.
12. Vallée M, McFadyen BJ, Swaine B, Doyon J. Effects of environmental demands on locomotion after traumatic brain injury. *Arch Phys Med Rehabil.* 2006 Sep;87(9):1299-1306. PMID: 16935074.
13. Rachal J, Reiss A, Modlesky C, et al. Reliability of cognitive dual-task tests in individuals with traumatic brain injury. *Phys Ther.* 2022 Aug 1;102(8):pzac059. PMID: 35472309.
14. Kelly VE, Eusterbrock AJ, Shumway-Cook A. A review of dual-task walking deficits in people with Parkinson's disease: Motor and cognitive contributions, mechanisms, and clinical implications. *Parkinsons Dis.* 2012;2012:918719. PMID: 23326763.
15. Lorenz LS, Charrette AL, O'Neil-Pirozzi TM, Doucett JM, Fong J. Healthy body, healthy mind: A mixed methods study of outcomes, barriers and supports for exercise by people who have chronic moderate-to-severe acquired brain injury. *Disabil Health J.* 2018 Jan;11(1):70-78. doi:10.1016/j.dhjo.2017.08.005.
16. Zotey V, Andhale A, Shegekar T, Juganavar A. Adaptive Neuroplasticity in Brain Injury Recovery: Strategies and Insights. *Cureus.* 2023;15(9):e45873. Published 2023 Sep 24. doi:10.7759/cureus.45873

17. Alashram AR, Padua E, Annino G. Effectiveness of physical therapy interventions on balance in individuals with traumatic brain injury: A systematic review. *Brain Inj.* 2020;34(7):913-921. PMID: 32588777.
18. Hornby TG, Henderson CE, Plawecki A, et al. Contributions of stepping intensity and variability to mobility in individuals with chronic stroke. *J Neurol Phys Ther.* 2019 Apr;43(2):115-123. PMID: 30789515.
19. Shick T, Perkins C, Paul A, et al. Randomized Controlled Trial: Preliminary Investigation of the Impact of High-Intensity Treadmill Gait Training on Recovery Among Persons with Traumatic Brain Injury. *Neurotrauma Rep.* 2025;6(1):82-92. Published 2025 Jan 24. doi:10.1089/neur.2024.0169
20. Plawecki A, Henderson CE, Lotter JK, et al. Comparative Efficacy of High-Intensity Training Versus Conventional Training in Individuals With Chronic Traumatic Brain Injury: A Pilot Randomized Controlled Study. *J Neurotrauma.* 2024;41(7-8):807-817. doi:10.1089/neu.2023.0494
21. Hornby TG, Reisman DS, Ward IG, Scheets PL, Miller A, Haddad D, Fox EJ, Fritz NE, Hawkins K, Henderson CE, Hendron KL, Holleran CL, Lynskey JE, Walter A. Clinical practice guideline to improve locomotor function following chronic stroke, incomplete spinal cord injury, and brain injury. *J Neurol Phys Ther.* 2020 Jan;44(1):49-100. doi:10.1097/NPT.0000000000000303.
22. Esquenazi A, Talaty M, Packel A, Saulino M. A randomized comparative study of manually assisted versus robotic-assisted body weight supported treadmill training in persons with a traumatic brain injury. *PM R.* 2013 Apr;5(4):280-290. doi:10.1016/j.pmrj.2012.10.009.
23. Kleffelgaard I, Soberg HL, Tamber A-L, et al. The effects of vestibular rehabilitation on dizziness and balance problems in patients after traumatic brain injury: a randomized controlled trial. *Clinical Rehabilitation.* 2019;33(1):74-84. doi:10.1177/0269215518791274
24. Tramontano M, Belluscio V, Bergamini E, et al. Vestibular Rehabilitation Improves Gait Quality and Activities of Daily Living in People with Severe Traumatic Brain Injury: A Randomized Clinical Trial. *Sensors (Basel).* 2022;22(21):8553. Published 2022 Nov 6. doi:10.3390/s22218553
25. Marcus HJ, Paine H, Sargeant M, et al. Vestibular dysfunction in acute traumatic brain injury. *J Neurol.* 2019;266(10):2430-2433. doi:10.1007/s00415-019-09403-z.
26. Alsalaheen BA, Mucha A, Morris LO, et al. Vestibular rehabilitation for dizziness and balance disorders after concussion. *J Neurol Phys Ther.* 2010 Sep;34(3):87-93. PMID: 20716972.
27. Bhattacharyya N, Gubbels SP, Schwartz SR, et al. Clinical practice guideline: Benign paroxysmal positional vertigo (update). *Otolaryngol Head Neck Surg.* 2017 Mar;156(3_suppl):S1-S47. PMID: 28238608.
28. Gordon CR, Levite R, Joffe V, Gadoth N. Is posttraumatic benign paroxysmal positional vertigo different from the idiopathic form? *Arch Neurol.* 2004 Jun;61(6):1590-1593. PMID: 15262741.
29. De Aquino Costa Sousa T, Gagnon I, Li K, McFadyen B, Lamontagne A. Exploring the challenges of avoiding collisions using a dual-task paradigm in individuals with chronic moderate to severe traumatic brain injury. *Journal of NeuroEngineering and Rehabilitation.* 2024
30. Fagan K, Howard K, Bruce J, et al. Using Bioness Integrated Therapy System (BITS) in visual and cognitive rehabilitation: A preliminary report. *NeuroRehabilitation.* 2020;46(4):541-548. PMID: 32538954.

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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

References

1. Center of Disease Control & Prevention. Mild traumatic brain injury and concussion: Information for adults. Traumatic Brain Injury & Concussion Discharge Instructions 2025.
2. Center of Disease Control & Prevention. CDC pediatric mild traumatic brain injury guideline recommendations. CDC pediatric mild traumatic brain injury guidelines 2025.
3. Morrow EL, Hereford AP, Covington NV, Duff MC. Traumatic brain injury in the acute care setting: assessment and management practices of speech-language pathologists. *Brain Injury*. 2020;34(12):1590–1609.

4. Crook L, Riccardi JS, Ruddock HS, Ciccio A. Speech-Language Pathology Treatment of Cognitive-Communication Deficits in School-Aged Children with Traumatic Brain Injury: A Scoping Review. *Journal of Speech, Language & Hearing Research*. 2023;66: 1826–1841.
5. Hardin KY, Black C, Caldbick K, Kelly M, Malbotra A, Tidd C, Vallentin T, Turkstra LS. Current Practices Among Speech-Language Pathologists for Mild Traumatic Brain Injury: A Mixed-Methods Modified Delphi Approach. *American Journal of Speech-Language Pathology*. 2021;30:1625–1655.
6. Brown J, Kaelin D, Mattingly E, Mello C, Miller ES, Mitchell G, Picon LM, Waldron-Perine B, Wolf TJ, Frymark T, Bowen R. American Speech-Language-Hearing Association Clinical Practice Guideline: Cognitive Rehabilitation for the Management of Cognitive Dysfunction Associated with Acquired Brain Injury. *American Journal of Speech-Language Pathology*. 2022;31(6):2455–2526.
7. Togher L, Wiseman-Hakes C, Douglas J, et al. INCOG Recommendations for Management of Cognition Following Traumatic Brain Injury, Part IV: Cognitive Communication. *Journal of Head Trauma Rehabilitation*. 2014;29(4):353–368.
8. Feddermann-Demont N, Echemendia RJ, Schneider KJ, Solomon GS, Hayden KA, Turner M, Dvořák J, Straumann D, Tarnutzer AA. What domains of clinical function should be assessed after sport-related concussion? A systematic review. *British Journal of Sports Medicine*. 2017;51(11):903–918.
9. Ponsford J, Velikonja D, Janzen S, et al. INCOG 2.0 Guidelines for Cognitive Rehabilitation Following Traumatic Brain Injury, Part II: Attention and Information Processing Speed. *Journal of Head Trauma Rehabilitation*. 2023;38(1):38–51.
10. Mitchell JT, Covington NV, Morrow E, de Riesthal M, Duff MC. Memory and Traumatic Brain Injury: Assessment and Management Practices of Speech-Language Pathologists. *American Journal of Speech-Language Pathology*. 2024;33(1):279–306.
11. Frith M, Togher L, Ferguson A, Levick W, Docking K. Assessment practices of speech-language pathologists for cognitive communication disorders following traumatic brain injury in adults: an international survey. *Brain Injury*. 2014;28(13–14):1657–1666.
12. Fleeman JA, Stavisky C, Carson S, et al. Integrating cognitive rehabilitation: A preliminary program description and theoretical review of an interdisciplinary cognitive rehabilitation program. *NeuroRehabilitation*. 2015;37(3):471–486.
13. Brunner M, Hemsley B, Togher L, Palmer S. Technology and its role in rehabilitation for people with cognitive-communication disability following a traumatic brain injury (TBI). *Brain Injury*. 2017;31(8):1028–1043.
14. Anjum J, Johnson Krug R, Kindsvogel D. The role of AT-SLP collaborations in return to academics following mTBI: A scoping review. *Journal of Interprofessional Care*. 2022; 36(1):83–92.
15. Byom L, O'Neil-Pirozzi TM, Lemoncello R, MacDonald S, Meulenbroek P, Ness B, Sohlberg MM. Social Communication Following Adult Traumatic Brain Injury: A Scoping Review of Theoretical Models. *American Journal of Speech-Language Pathology*. 2020;29(3):1735–1748.
16. Meulenbroek P, O'Neil-Pirozzi TM, Sohlberg MM, Lemoncello R, Byom L, Ness B, MacDonald S, Phillip B. Tutorial: The Speech-Language Pathologist's Role in Return to Work for Adults with Traumatic Brain Injury. *American Journal of Speech-Language Pathology*. 2022;31(1):188–202.

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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 (IGF), 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 (VEGF), which stimulate angiogenesis and the migration of osteoprogenitor cells, which are stimulated by VEGF 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

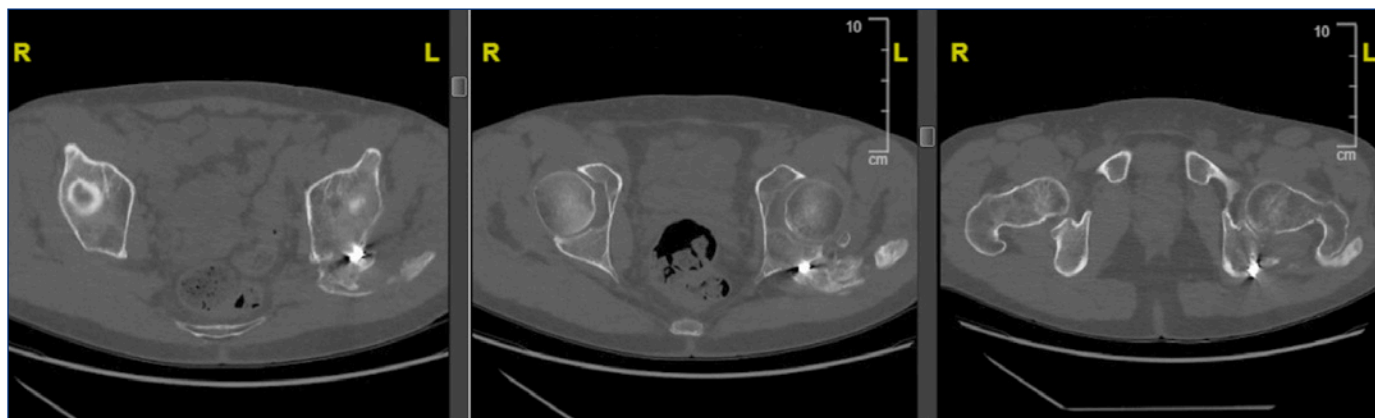


Figure 3. 3-view radiographs of the pelvis including AP [A], iliac oblique [B] and obturator oblique [C] at 4 months after surgical excision of prior NHO.



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.

References

1. Ohlmeier M, Suero EM, Aach M, Meindl R, Schildhauer TA, Citak M. Muscle localization of heterotopic ossification following spinal cord injury. *Spine J Off J North Am Spine Soc*. 2017 Oct;17(10):1519–22.
2. Sakellariou VI, Grigoriou E, Mavrogenis AF, Soucacos PN, Pappagelopoulos PJ. Heterotopic ossification following traumatic brain injury and spinal cord injury: insight into the etiology and pathophysiology. *J Musculoskelet Neuronal Interact*. 2012 Dec;12(4):230–40.
3. Shehab D, Elgazzar AH, Collier BD. Heterotopic ossification. *J Nucl Med Off Publ Soc Nucl Med*. 2002 Mar;43(3):346–53.
4. Pape HC, Marsh S, Morley JR, Krettek C, Giannoudis PV. Current concepts in the development of heterotopic ossification. *J Bone Joint Surg Br*. 2004 Aug;86(6):783–7.
5. Cipriano CA, Pill SG, Keenan MA. Heterotopic ossification following traumatic brain injury and spinal cord injury. *J Am Acad Orthop Surg*. 2009 Nov;17(11):689–97.
6. Reznik JE, Biros E, Marshall R, Jelbart M, Milanese S, Gordon S, et al. Prevalence and risk-factors of neurogenic heterotopic ossification in traumatic spinal cord and traumatic brain injured patients admitted to specialised units in Australia. *J Musculoskelet Neuronal Interact*. 2014 Mar;14(1):19–28.
7. Bongetta D, Bua M, Bruno R, Colombo EV, de Laurentis C, Versace A, et al. Is Gender a Factor Affecting Long-Term Heterotopic Ossification Incidence After Single-Level Cervical Disc Arthroplasty? *World Neurosurg*. 2022 Sep 1;165:6–12.
8. Reznik JE, Biros E, Milanese S, Gordon S, Lamont AC, Galea MP. Prevalence of neurogenic heterotopic ossification in traumatic head- and spinal-injured patients admitted to a tertiary referral hospital in Australia. *Health Care Manag*. 2015;34(1):54–61.
9. Garland DE. Clinical observations on fractures and heterotopic ossification in the spinal cord and traumatic brain injured populations. *Clin Orthop*. 1988 Aug;(233):86–101.
10. Simonsen LL, Sonne-Holm S, Krashenninikoff M, Engberg AW. Symptomatic heterotopic ossification after very severe traumatic brain injury in 114 patients: incidence and risk factors. *Injury*. 2007 Oct;38(10):1146–50.
11. Kaplan FS, Glaser DL, Hebela N, Shore EM. Heterotopic ossification. *J Am Acad Orthop Surg*. 2004;12(2):116–25.
12. Lees-Shepard JB, Yamamoto M, Biswas AA, Stoessel SJ, Nicholas SAE, Cogswell CA, et al. Activin-dependent signaling in fibro/adipogenic progenitors causes fibrodysplasia ossificans progressiva. *Nat Commun*. 2018 Feb 2;9(1):471.
13. Kan C, Chen L, Hu Y, Ding N, Lu H, Li Y, et al. Conserved signaling pathways underlying heterotopic ossification. *Bone*. 2018 Apr;109:43–8.
14. Agarwal S, Loder SJ, Sorkin M, Li S, Shrestha S, Zhao B, et al. Analysis of Bone-Cartilage-Stromal Progenitor Populations in Trauma Induced and Genetic Models of Heterotopic Ossification. *Stem Cells Dayt Ohio*. 2016 Jun;34(6):1692–701.
15. Firoozabadi R, Alton T, Sagi HC. Heterotopic Ossification in Acetabular Fracture Surgery. *J Am Acad Orthop Surg*. 2017 Feb;25(2):117–24.
16. Sullivan MP, Torres SJ, Mehta S, Ahn J. Heterotopic ossification after central nervous system trauma: A current review. *Bone Jt Res*. 2013 Mar;2(3):51–7.
17. Wong KR, Mychasiuk R, O'Brien TJ, Shultz SR, McDonald SJ, Brady RD. Neurological heterotopic ossification: novel mechanisms, prognostic biomarkers and prophylactic therapies. *Bone Res*. 2020 Dec 9;8(1):42.
18. Almangour W, Schnitzler A, Salga M, Debaud C, Denormandie P, Genet F. Recurrence of heterotopic ossification after removal in patients with traumatic brain injury: A systematic review. *Ann Phys Rehabil Med*. 2016 Sep;59(4):263–9.
19. Chen S, Yu S yang, Yan H, Cai J yu, Ouyang Y, Ruan H jiang, et al. The time point in surgical excision of heterotopic ossification of post-traumatic stiff elbow: recommendation for early excision followed by early exercise. *J Shoulder Elbow Surg*. 2015 Aug;24(8):1165–71.

20. Bedi A, Zbeda RM, Bueno VF, Downie B, Dolan M, Kelly BT. The incidence of heterotopic ossification after hip arthroscopy. *Am J Sports Med.* 2012 Apr;40(4):854–63.
21. Tao MJ, Probyn L, Poon M, Kreder H, Nousiainen M, Jenkinson R, et al. Potential discrepancy between plain films and CT scans in Brooker classification of heterotopic ossification. *Br J Radiol.* 2017 Dec;90(1080):20170263.
22. Estrores IM, Harrington A, Banovac K. C-reactive protein and erythrocyte sedimentation rate in patients with heterotopic ossification after spinal cord injury. *J Spinal Cord Med.* 2004;27(5):434–7.
23. Mysiw WJ, Tan J, Jackson RD. Heterotopic ossification. The utility of osteocalcin in diagnosis and management. *Am J Phys Med Rehabil.* 1993 Aug;72(4):184–7.
24. Vanden Bossche L, Vanderstraeten G. Heterotopic ossification: a review. *J Rehabil Med.* 2005 May;37(3):129–36.
25. Wittenberg RH, Peschke U, Bötzel U. Heterotopic ossification after spinal cord injury. Epidemiology and risk factors. *J Bone Joint Surg Br.* 1992 Mar;74(2):215–8.
26. Kjaersgaard-Andersen P, Pedersen P, Kristensen SS, Schmidt SA, Pedersen NW. Serum alkaline phosphatase as an indicator of heterotopic bone formation following total hip arthroplasty. *Clin Orthop.* 1988 Sep;234:102–9.
27. Sun E, Hanyu-Deutmeyer AA. Heterotopic Ossification. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 [cited 2025 Mar 30]. Available from: <http://www.ncbi.nlm.nih.gov/books/NBK519029/>
28. Genet F, Marmorat JL, Lautridou C, Schnitzler A, Mailhan L, Denormandie P. Impact of late surgical intervention on heterotopic ossification of the hip after traumatic neurological injury. *J Bone Joint Surg Br.* 2009 Nov;91(11):1493–8.
29. Argyropoulou MI, Kostandi E, Kosta P, Zikou AK, Kastani D, Galiatsou E, et al. Heterotopic ossification of the knee joint in intensive care unit patients: early diagnosis with magnetic resonance imaging. *Crit Care Lond Engl.* 2006;10(5):R152.
30. Lee A, Maani EV, Amin NP. Radiation Therapy for Heterotopic Ossification Prophylaxis. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 [cited 2025 Mar 30]. Available from: <http://www.ncbi.nlm.nih.gov/books/NBK493155/>
31. Vavken P, Castellani L, Sculco TP. Prophylaxis of Heterotopic Ossification of the Hip: Systematic Review and Meta-Analysis. *Clin Orthop.* 2009 Dec;467(12):3283–9.
32. Fransen M, Anderson C, Douglas J, MacMahon S, Neal B, Norton R, et al. Safety and efficacy of routine postoperative ibuprofen for pain and disability related to ectopic bone formation after hip replacement surgery (HIPAID): randomised controlled trial. *BMJ.* 2006 Sep 9;333(7567):519.
33. Gil JA, Waryasz GR, Klyce W, Daniels AH. Heterotopic Ossification in Neurorehabilitation. *R I Med J* 2013. 2015 Dec 1;98(12):32–4.
34. Miglioni F, Trivellas A, Eschweiler J, Driessen A, Tingart M, Maffulli N. NSAIDs for Prophylaxis for Heterotopic Ossification After Total Hip Arthroplasty: A Bayesian Network Meta-analysis. *Calcif Tissue Int.* 2021;108(2):196–206.
35. Tumminelli P, Shapiro S, Cooper V, Thomas C, Sampathkumar H, Islam M. Management of Heterotopic Ossification with Bisphosphonates after Hip Hemiarthroplasty in Patients with Contraindications to Standard of Care Prophylaxis. *Quill Scope [Internet].* 2017 Jan 1;9(1). Available from: https://touro scholar.touro.edu/quill_and_scope/vol9/iss1/11
36. Banovac K, Gonzalez F, Renfree KJ. Treatment of heterotopic ossification after spinal cord injury. *J Spinal Cord Med.* 1997 Jan;20(1):60–5.
37. Vasileiadis GI, Sakellariou VI, Kelekis A, Galanos A, Soucacos PN, Papagelopoulos PJ, et al. Prevention of heterotopic ossification in cases of hypertrophic osteoarthritis submitted to total hip arthroplasty. Etidronate or Indomethacin? *J Musculoskelet Neuronal Interact.* 2010 Jun;10(2):159–65.
38. Chao ST, Joyce MJ, Suh JH. Treatment of heterotopic ossification. *Orthopedics.* 2007 Jun;30(6):457–64; quiz 465–6.
39. Georhakopoulos I, Kouloulis V, Kougiountzopoulou A, Platoni K, Antypas C, Liakouli Z, et al. Radiation therapy for the prevention of heterotopic ossification: Efficacy and toxicity of single fraction radiotherapy. *Orthop Rev.* 2020 Aug 18;12(2):8577.
40. Pakos EE, Ioannidis JPA. Radiotherapy vs. nonsteroidal anti-inflammatory drugs for the prevention of heterotopic ossification after major hip procedures: a meta-analysis of randomized trials. *Int J Radiat Oncol Biol Phys.* 2004 Nov 1;60(3):888–95.
41. Garland DE, Hanscom DA, Keenan MA, Smith C, Moore T. Resection of heterotopic ossification in the adult with head trauma. *J Bone Joint Surg Am.* 1985 Oct;67(8):1261–9.
42. Lachiewicz PF, Skalla LA, Purcell KF. Surgical Treatment of Severe Heterotopic Ossification After Total Hip Arthroplasty Over the Last 25 Years: A Systematic Review of the Literature and a New Case Series. *J Arthroplasty.* 2024 Sep 1;39(9):S312–S317.e1.
43. Schuetz P, Mueller B, Christ-Crain M, Dick W, Haas H. Amnino-bisphosphonates in heterotopic ossification: first experience in five consecutive cases. *Spinal Cord.* 2005 Oct;43(10):604–10.
44. Jayamaraju D, Sarkar AS, Patra SK, Palanivelayutham SK, Rajasekaran S. A Surgical Protocol for Management of Post Traumatic Heterotopic Ossification of Elbow. *Indian J Orthop.* 2021 Feb 25;55(4):898–906.
45. Xu Y, Huang M, He W, He C, Chen K, Hou J, et al. Heterotopic Ossification: Clinical Features, Basic Researches, and Mechanical Stimulations. *Front Cell Dev Biol [Internet].* 2022 Jan 25 [cited 2025 Mar 30];10. Available from: <https://www.frontiersin.org/journals/cell-and-developmental-biology/articles/10.3389/fcell.2022.770931/full>
46. Salazar D, Golz A, Israel H, Marra G. Heterotopic ossification of the elbow treated with surgical resection: risk factors, bony ankylosis, and complications. *Clin Orthop.* 2014 Jul;472(7):2269–75.

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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 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 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 _{δ1} subunit binding to inhibit Ca2+ currents	Infants: 5 mg/kg/day, titrate by adding up to 3x day or increasing dose 3–11 years: 10–15 mg/kg/day in 3 divided doses >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 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 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 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

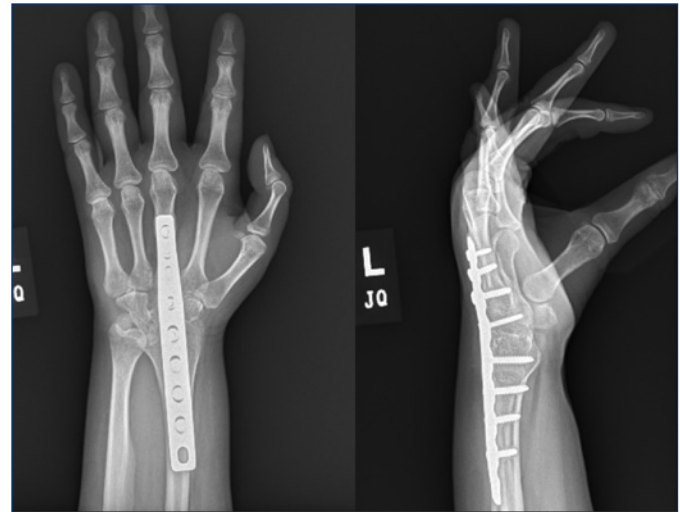
Upper extremity

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

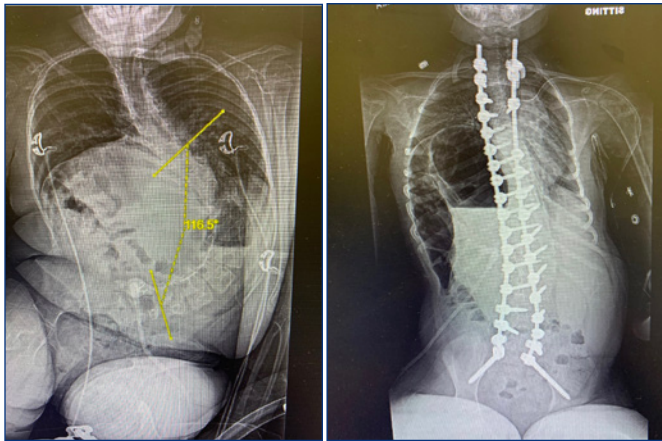
Figure 1. Twenty-year-old woman with left wrist contracture and a painful bunion deformity on left foot due to spasticity related to an intracranial bleed at age eight. [Radiographs are courtesy of Dr. Craig Eberson.]



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 (FCR) 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 is implicated in the deformity.²⁷

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

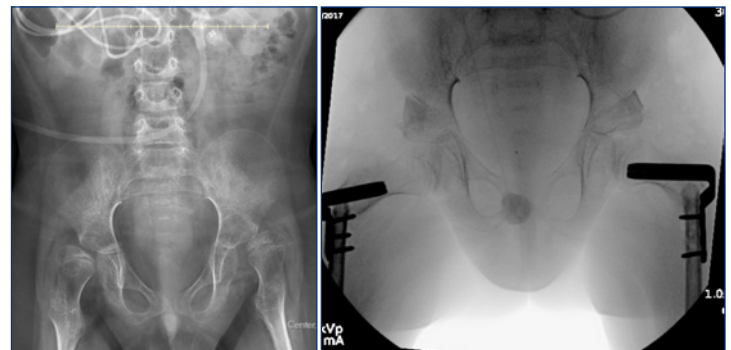
Thus, the final decision to pursue surgery requires detailed discussion of risks and benefits between providers and families in order to arrive at the optimal treatment plan.

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

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.



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)

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% 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: Femur: proximal femoral varus derotation osteotomy 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) Redirection: subtrochanteric valgus osteotomy 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.

References

1. Kheder A, Nair KPS. Spasticity: pathophysiology, evaluation and management. *Pract Neurol*. 2012;12(5):289-298. doi:10.1136/practneurol-2011-000155
2. Mu X, Deng B, Zeng J, et al. Orthopedic treatment of the lower limbs in spastic paralysis. *Brain Science Advances*. 2020;6(1):2-19. doi:10.26599/BSA.2020.9050001
3. Hashemi M, Sturbois-Nachef N, Keenan MA, Winston P. Surgical Approaches to Upper Limb Spasticity in Adult Patients: A Literature Review. *Front Rehabil Sci*. 2021;2:709969. doi: 10.3389/fresc.2021.709969
4. Dressler D, Bhidayasiri R, Bohlega S, et al. Defining spasticity: a new approach considering current movement disorders terminology and botulinum toxin therapy. *J Neurol*. 2018;265(4):856-862. doi:10.1007/s00415-018-8759-1
5. Pidgeon TS, Ramirez JM, Schiller JR. Orthopaedic Management of Spasticity. *R I Med J* (2013). 2015;98(12):26-31.
6. Fitoussi F, Lallemand-Dudek P. The upper limb in children with cerebral palsy. Evaluation and treatment. *Orthopaedics & Traumatology: Surgery & Research*. 2024;110(1, Supplement): 103763. doi:10.1016/j.otsr.2023.103763
7. Chad KE, Bailey DA, McKay HA, Zello GA, Snyder RE. The effect of a weight-bearing physical activity program on bone mineral content and estimated volumetric density in children with spastic cerebral palsy. *J Pediatr*. 1999;135(1):115-117. doi: 10.1016/s0022-3476(99)70340-9
8. Woo R. Spasticity: orthopedic perspective. *J Child Neurol*. 2001;16(1):47-53. doi:10.1177/088307380101600108
9. Howard IM, Patel AT. Spasticity evaluation and management tools. *Muscle & Nerve*. 2023;67(4):272-283. doi:10.1002/mus.27792
10. Tenforde AS, Borgstrom HE, DeLuca S, et al. Best practices for extracorporeal shockwave therapy in musculoskeletal medicine: Clinical application and training consideration. *PM R*. 2022;14(5):611-619. doi:10.1002/pmrj.12790
11. Yang E, Lew HL, Özçakar L, Wu CH. Recent Advances in the Treatment of Spasticity: Extracorporeal Shock Wave Therapy. *JCM*. 2021;10(20):4723. doi:10.3390/jcm10204723
12. Brandenburg JE, Rabatin AE, Driscoll SW. Spasticity Interventions: Decision-Making and Management. *Pediatric Clinics of North America*. 2023;70(3):483-500. doi:10.1016/j.pcl.2023.01.005

13. Rice J, Hugos C, Hildebrand A, Cameron M. Cannabis use in people with multiple sclerosis and spasticity: A cross-sectional analysis. *Multiple Sclerosis and Related Disorders*. 2020;41. doi:10.1016/j.msard.2020.102009
14. Chang E, Ghosh N, Yanni D, Lee S, Alexandru D, Mozaffar T. A Review of Spasticity Treatments: Pharmacological and Interventional Approaches. *Crit Rev Phys Rehabil Med*. 2013;25(1-2):11-22. doi:10.1615/CritRevPhysRehabilMed.2013007945
15. Cho SR. Intrathecal Baclofen Therapy: Pros and Cons. *Ann Rehabil Med*. 2023;47(1):1-3. doi:10.5535/arm.23003
16. Lee HP, Win T, Balakrishnan S. The impact of intrathecal baclofen on the ability to walk: A systematic review. *Clin Rehabil*. 2023;37(4):462-477. doi:10.1177/02692155221135827
17. Boster AL, Adair RL, Gooch JL, et al. Best Practices for Intrathecal Baclofen Therapy: Dosing and Long-Term Management. *Neuromodulation*. 2016;19(6):623-631. doi:10.1111/ner.12388
18. Chen BPJ, Wang KK, Novacheck TF. Selective Dorsal Rhizotomy for the Treatment of Gait Dysfunction in Cerebral Palsy: A Critical Analysis Review. *JBJS Rev*. 2019;7(11):e3-e3. doi:10.2106/JBJS.RVW.19.00020
19. Munger ME, Aldahondo N, Krach LE, Novacheck TF, Schwartz MH. Long-term outcomes after selective dorsal rhizotomy: a retrospective matched cohort study. *Developmental Medicine & Child Neurology*. 2017;59(11):1196-1203. doi:10.1111/dmcn.13500
20. Park T, Liu JL, Edwards C, Walter DM, Dobbs MB. Functional Outcomes of Childhood Selective Dorsal Rhizotomy 20 to 28 Years Later. *Cureus*. 9(5):e1256. doi:10.7759/cureus.1256
21. Lynn AK, Turner M, Chambers HG. Surgical Management of Spasticity in Persons with Cerebral Palsy. *PM&R*. 2009;1(9):834-838. doi:10.1016/j.pmrj.2009.07.016
22. Van Heest AE, Bagley A, Molitor F, James MA. Tendon Transfer Surgery in Upper-Extremity Cerebral Palsy Is More Effective Than Botulinum Toxin Injections or Regular, Ongoing Therapy. *The Journal of Bone and Joint Surgery*. 2015;97(7):529-536. doi:10.2106/JBJS.M.01577
23. Tranchida GV, Van Heest A. Preferred options and evidence for upper limb surgery for spasticity in cerebral palsy, stroke, and brain injury. *J Hand Surg Eur Vol*. 2020;45(1):34-42. doi:10.1177/1753193419878973
24. Ziran B, Nourbakhsh A. Proximal humerus derotational osteotomy for internal rotation instability after locked posterior shoulder dislocation: early experience in four patients. *Patient Saf Surg*. 2015;9(1):15. doi:10.1186/s13037-015-0062-9
25. Holbrook HS, Greenberg JA, Weller WJ. Application of Tendon With Z-Lengthening Technique. *J Hand Surg Am*. 2023;48(7):740.e1-740.e11. doi:10.1016/j.jhsa.2022.12.016
26. Gharbaoui I, Kania K, Cole P. Spastic Paralysis of the Elbow and Forearm. *Seminars in Plastic Surgery*. 2016;30(01):039-044. doi:10.1055/s-0035-1571255
27. Seruya M, Dickey RM, Fakhro A. Surgical Treatment of Pediatric Upper Limb Spasticity: The Wrist and Hand. *Semin Plast Surg*. 2016;30(1):29-38. doi:10.1055/s-0035-1571254
28. Seaver CD, Morgan SJ, Legister CS, et al. Long-term reoperation rates following spinal fusion for neuromuscular scoliosis in nonambulatory patients with cerebral palsy. *Spine Deform*. 2024;12(5):1393-1401. doi:10.1007/s43390-024-00878-z
29. Hasler CC. Operative treatment for spinal deformities in cerebral palsy. *J Child Orthop*. 2013;7(5):419-423. doi:10.1007/s11832-013-0517-4
30. Murphy RF, Mooney JF. Current concepts in neuromuscular scoliosis. *Curr Rev Musculoskelet Med*. 2019;12(2):220-227. doi:10.1007/s12178-019-09552-8
31. Miller DJ, Flynn J (Jack) M, Pasha S, et al. Improving Health-related Quality of Life for Patients With Nonambulatory Cerebral Palsy: Who Stands to Gain From Scoliosis Surgery? *Journal of Pediatric Orthopaedics*. 2020;40(3):e186-e192. doi:10.1097/BPO.0000000000001424
32. Schoenecker JG. Pathologic Hip Morphology in Cerebral Palsy and Down Syndrome. *Journal of Pediatric Orthopaedics*. 2013;33(Supplement 1):S29-S32. doi:10.1097/BPO.0b013e3182860034
33. Lins LAB, Watkins CJ, Shore BJ. Natural History of Spastic Hip Disease. *J Pediatr Orthop*. 2019;39(Issue 6, Supplement 1 Suppl 1):S33-S37. doi:10.1097/BPO.0000000000001347
34. Hosseinzadeh P, Baldwin K, Minaie A, Miller F. Management of Hip Disorders in Patients with Cerebral Palsy. *JBJS Reviews*. 2020;8(3):e0148-e0148. doi:10.2106/JBJS.RVW.19.00148
35. King BW, Ruta DJ, Irwin TA. Spastic Foot and Ankle Deformities. *Foot and Ankle Clinics*. 2014;19(1):97-111. doi:10.1016/j.fcl.2013.10.007

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Evaluation of the Mental Health Temperature of Rhode Island Emergency Medical Services (EMS)

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ABSTRACT

Mental health among first responders is often impacted by repeated exposure to stressors and traumatic events. As a result, Emergency Medical Services (EMS) professionals have a higher risk of suicide than the general public.¹ With each state having different EMS cultures and operations, the Rhode Island Department of Health (RIDOH) Center for EMS identified a need to assess the mental health of EMS professionals in Rhode Island. A survey was distributed to EMS professionals in Rhode Island to evaluate their mental health and identify demographic gaps, as well as assess the risk of suicide for EMS professionals. Survey results provided evidence of concerning mental health among first responders in Rhode Island, with 15.4 percent of responders reporting suicidal ideation in the past 12 months. The results suggest that increased programming to provide resources and awareness is necessary to improve mental health and ensure the longevity of EMS professionals' careers.

KEYWORDS: Emergency Medical Services; Mental Health; Occupational Stress

INTRODUCTION

EMS professionals in Rhode Island have an extensive scope of practice, allowing them to perform life-saving measures in the prehospital setting. In 2023, 233,445 patient care reports were completed in Rhode Island.² Nationally, EMS employment is projected to grow by five percent between 2022 and 2032, which is higher than the average across all occupations.³ As the profession grows, recruiting and retaining healthy employees is extremely important to provide life-saving care to communities across the state.

As of 2023, the State of Rhode Island had 4,576 licensed EMS professionals and 83 EMS agencies. The agencies include fire departments, stand-alone EMS agencies, private ambulance companies, collegiate, and corporate EMS.⁴ EMTs in Rhode Island can become licensed at the age of 18 and can retain their license as long as they meet the necessary continuing education requirements for re-licensure.⁵

Between January 2009 and October 2024, 16 EMS providers died by suicide in Rhode Island. This number was

derived from reports to the RIDOH Center for EMS from family and agency leaders. It is possible there are additional unreported cases. While this number may appear small, the culture of the tight-knit Rhode Island EMS community was profoundly affected by these losses.

Studies have shown that EMS professionals die by suicide at higher rates than the general public. Further research confirms this by demonstrating that EMS professionals have an increase in risk factors associated with suicide, including acute and chronic stress, fatigue, depression, and substance misuse. The presence of these risk factors significantly increases the risk of suicide. The presence of these risk factors is predicted to be the result of repeated instances of the physical and psychological stress associated with being an EMS professional.⁶ In addition, EMS professionals have a higher rate of completed suicides compared to the general population.⁷ Because of this, it is imperative that mental health is addressed before suicide attempts occur.

The goal of this survey was to assess the EMS mental health in Rhode Island, in order to identify the need for resources and policy updates. EMS professionals were surveyed on physical health, mental health, substance use, stigma, and availability of resources. The RIDOH Center for EMS wanted to evaluate the current mental health status of EMS professionals in addition to assessing how many professionals currently exhibit risk factors. Results from the survey will guide future mental health initiatives and resources for all first responders.

The survey was conducted anonymously due to the sensitive nature of the questions posed. It was felt that EMS professionals would be less likely to respond to the survey honestly if their names were recorded. The Rhode Island Department of Health Center for EMS is responsible for disciplinary actions, and while the authors had no intention of using this research for disciplinary investigations, fear of punishment or affected reputation was predicted to result in skewed data.

The survey primarily consisted of multiple-choice questions. The authors included one short answer question to survey advice on how to improve the Critical Incident Stress Management program that is put on by The Rhode Island Critical Incident Stress Management Team. The question asked "Critical Incident Stress Management could be

improved with the following.” These data were omitted from the paper as the authors were not looking to publicly critique the organization. These data were shared internally.

Peer-reviewed research that queries individual EMS professionals on their mental health with this scope does not exist. The only other state to conduct a similar survey was the Virginia Department of Health (VDH) in 2019.⁸ Due to significant differences in demographics, provider level, and employment status, the authors felt that it would not be relevant to directly compare the results of RIDOH’s survey to VDH’s survey.

METHODS

We identified a need to learn about the mental health of Rhode Island EMS professionals, as no other research on this topic existed. Fifty-one multiple-choice and short-answer questions were formulated. Some questions were objective-based, such as the subject’s demographics. Others were added from the Substance Abuse and Mental Health Services Administration (SAMHSA) mental health questions, in collaboration with the Comprehensive Suicide Prevention Program at RIDOH, and previously completed surveys from the Virginia Department of Health. The survey was created and distributed via Research Electronic Data Capture (REDCap, Nashville, TN). An email list was compiled from the RI state EMS data repository ImageTrend, the state licensing platform MyLicense Office (MLO), and the EMS Learning Management System Train.org. There were 9,072 email addresses collected from the three sources. Duplicate email addresses were removed; however, some individuals had different emails listed in each data source. About 1,000 to 1,500 emails were returned to the sender after the distribution of the survey. The survey was first distributed on May 21, 2024, and closed on July 15, 2024. Two reminder emails were sent out following the initial launch of the survey. Results were analyzed using Microsoft Excel (Microsoft, Redmond WA).

RESULTS

There were 953 survey responses recorded in the REDCap survey system. The study participants’ demographics were consistent with national data for EMS providers for both sex and race. About 91.0 percent of survey respondents reported their race and ethnicity as non-Hispanic White, compared to approximately 85.0 percent of nationally certified EMS personnel identifying as non-Hispanic white.⁹ With 78.9 percent of individuals assigned male at birth, this is also representative of the national EMS professional population, with 76.0 percent male.¹⁰ Since respondents were predominantly from similar demographics, no significant conclusions could be determined based on race, ethnicity, or sex. It is worth

noting that 48.2 percent of respondents are EMT-Cardiacs, a licensure type specific to Rhode Island but similar to the national Advanced EMT level.¹⁰ This should be considered for the generalizability of the study.

While mental health struggles are present across all demographics, young professionals seem to be the population most burdened with suicidal thoughts. The data show 22.2 percent of EMS professionals ages 18–24 have had suicidal ideation in the past 12 months, with the percentage of reported suicidal thoughts declining as age increases. Similarly, 21.3 percent of EMS professionals with three to five years of experience had thoughts of killing themselves. Of the 471 providers who reported burnout, 42.9 percent are aged 25–39. Of the providers ages 25–39 with burnout, 23.8 also reported suicidal thoughts. Also, 33.3 percent of all professionals who reported that they quit EMS experienced suicidal thoughts.

Mental health resource accessibility varied across respondents. Overall, 65.6 percent of responding EMS professionals reported that their agency offers mental health resources, with 20.8 percent of respondents unsure. When asked about specific resources, 68.6 percent of respondents reported an available employee assistance program with 21.3 percent of respondents unsure. In addition, 60.9 percent of respondents had received mental health training during the prior 12 months, and 81.5 percent of respondents knew where to find help for mental health issues in their agency. An overwhelming 92.8 percent of respondents knew about Critical Incident Stress Management (CISM). CISM is a program that provides debriefing after emotionally stressful or traumatic events.¹¹ Despite the program’s broad recognition, only 23.4 percent of providers had participated in a CISM debriefing.

Rhode Island EMS professionals listed several reasons for not receiving mental health support, with, “I didn’t think it was needed,” and, “I already possess sufficient coping skills,” having the highest percentages with 58.1 and 23.6, respectively. Following this, the next highest reason was “I didn’t have time,” at 16.3 percent. [Tables 1–5]

Seven questions from the survey were directly linked to risk factors for suicide [Table 6]. While the presence of these risk factors may or may not be directly attributed to the EMS profession, research concludes that the presence of these risk factors is linked to suicide.⁶ They included physical injury on the job, stress level, emotional problems, mental health effects on relationships, frequency of alcohol consumption, and average number of drinks in one day.^{6,12,13} Results were filtered based on answers that reported very high levels of stress, mental health affecting relationships very often, daily drinking, and seven or more drinks per day, in addition to positive responses on the yes/no questions.

Table 1. Baseline characteristics of surveyed participants

	n (%)
Age	
18–24	87 (9.4)
25–39	296 (31.9)
40–49	195 (21.0)
50–64	299 (32.2)
65+	51 (5.5)
Sex assigned at birth	
Male	737 (78.9)
Female	183 (19.6)
Intersex	2 (0.2)
Prefer not to disclose	12 (1.3)
Gender Identity	
Agender	2 (0.2)
Genderqueer or genderfluid	2 (0.2)
Man	727 (78.0)
Non-binary	4 (0.4)
Questioning or unsure	1 (0.1)
Two-spirit	2 (0.2)
Woman	174 (18.7)
Prefer not to disclose	21 (2.3)
Additional gender category/identity not listed	5 (0.5)
Ethnicity	
Hispanic or Latino	40 (4.3)
Not Hispanic or Latino	895 (95.7)
Race	
American Indian or Alaska Native	5 (0.5)
Asian	20 (2.1)
Black or African American	22 (2.4)
Native Hawaiian or Other Pacific Islander	5 (0.5)
White	898 (96.2)

Table 2. EMS employment demographics

	n (%)
EMS License Level	
Emergency Medical Responder	36 (3.9)
EMT	239 (25.6)
Advanced EMT	23 (2.5)
EMT Cardiac	450 (48.2)
Paramedic	186 (19.9)
Serves with a fire department	
Yes	688 (73.5)
Average hours worked in a week	
<20	69 (7.4)
20–40	110 (11.8)
41–50	287 (30.7)
51–60	205 (21.9)
61–70	144 (15.4)
71–80	63 (6.7)
>80	57 (6.1)
Have you ever served in the military	
Yes	123 (13.2)
Employment and Volunteer Status	
Full-time	563 (60.2)
Part-time	45 (4.8)
Unpaid Volunteer	73 (7.8)
Full-time & Volunteer	47 (5.0)
Part-time & Volunteer	41 (4.4)
Full-time & Part-time	11 (1.2)
Stipend/Paid Volunteer	53 (5.7)
Taking a break, but plan to return in the future	21 (2.2)
Retired	54 (5.8)
Other	16 (1.7)
Quit	11 (1.2)
Years of Experience	
<1	34 (3.6)
1–2	61 (6.5)
3–5	85 (9.1)
6–10	131 (14.0)
11–15	128 (13.7)
16–20	134 (14.3)
21–25	117 (12.5)
26–30	86 (9.2)
>30	160 (17.1)

Table 3. Reported substance use and misuse of EMS providers

	n (%)	
Frequency of having a drink containing alcohol in the past year		
Never	141 (16.2)	
Monthly or less	176 (20.2)	
Two to four times a month	233 (26.8)	
Two to three times a week	185 (21.2)	
Four or more times a week	136 (15.6)	
Number of alcoholic drinks had on a day when drinking in the past year		
None, I do not drink	167 (19.2)	
1 to 2	388 (44.5)	
3 to 4	184 (21.2)	
5 to 6	90 (10.3)	
7 to 9	28 (3.2)	
10 or more	14 (1.6)	
Reports of frequent substance use		
Substance	Weekly use n (%)	Daily or almost daily use n (%)
Tobacco	26 (3.0)	117 (13.4)
6 or more alcoholic drinks	70 (8.1)	20 (2.3)
Prescription drugs for non-medical reasons	4 (0.5)	5 (0.6)
Cannabis	57 (6.5)	67 (7.7)
Illegal drugs	5 (0.6)	1 (0.1)

Table 4. EMS professional evaluation of health

Question	Yes
Suffered a physical injury due to job	183 (20.4)
If you suffered an injury, did you have to take time out of work	114 (62.3)
Problems with work or daily life due to emotional problems, such as feeling depressed, sad or anxious	429 (47.8)
Diagnosed with a mental health disorder	252 (28.1)
Thoughts about killing oneself	138 (15.4)
In the last 12 months, have you done anything, started to do anything, or prepared to do anything to end your life?	29 (3.2)
Called/texted/online chatted with 988 for oneself	9 (1.0)
Experienced burnout	471 (52.6)
Sought out help for mental health	281 (31.4)
Physical Health Rating	
Excellent	172 (19.2)
Average	574 (64.0)
Somewhat poor	122 (13.6)
Poor	28 (3.1)
Not sure	1 (0.1)
Mental Health Rating	
Excellent	151 (16.8)
Average	470 (52.4)
Somewhat poor	197 (22.0)
Poor	69 (7.7)
Not sure	10 (1.1)
Stress Levels	
Very low/none	25 (2.8)
Low	151 (16.8)
Moderate	459 (51.2)
High	217 (24.2)
Very high	45 (5.0)
Average Hours of Sleep in a 24 hour Period	
0–5	216 (23.1)
6	397 (42.4)
7	227 (24.3)
8	78 (8.3)
9	13 (1.4)
10+	5 (0.5)
Effect on Relationships	
Very often	102 (11.4)
Somewhat often	254 (28.3)
Not so often	357 (39.8)
Not at all	184 (20.5)

Table 5. Mental health stigma in EMS

	Strongly Agree or Agree	Neutral	Disagree or Strongly Disagree
My agency considers mental health important	479 (55.6)	226 (26.2)	157 (18.2)
I feel comfortable talking about my mental health with my colleagues	367 (44.0)	249 (29.9)	218 (26.1)
I am afraid to ask for mental health resources due to fear or retaliation or disciplinary action	126 (15.1)	182 (21.8)	526 (63.1)
I am afraid to ask for mental health resources due to a fear of being labeled or treated differently by my colleagues	199 (24.0)	208 (25.1)	422 (50.9)
I feel appreciated by my agency when I think about what they pay me	233 (29.9)	239 (30.7)	306 (39.3)

Table 6. Presence of risk factors for suicide in EMS professionals

Number of risk factors	Total	Male	Female	18–24	25–39	40–49	50–64	65+
1	318	245	67	34	105	62	104	13
2	151	115	34	11	65	41	33	1
3	82	64	15	12	26	25	18	1
4	18	13	4	1	10	4	2	1
5	10	6	3	0	6	1	2	1
6	0	0	0	0	0	0	0	0
7	0	0	0	0	0	0	0	0

DISCUSSION

RIDOH Center for EMS hypothesized that there would be a high percentage of EMS professionals suffering from mental health problems, and even higher percentages of professionals with suicide risk factors. However, the 15.4 percent of providers who have had thoughts of killing themselves was far larger than expected as the national average of adults ages 18 or older with serious thoughts of suicide in the past 12 months is approximately 5.0 percent.¹⁴ Rhode Island EMS professionals are struggling with their mental health at a rate higher than the national average, likely due to high presence of risk factors which have been linked to suicide in previous research. EMS professionals are burdened by their job and are not receiving and/or utilizing the resources they need to cope with job-related pressures. This burden enters their personal life, with high rates of relationship instability and substance misuse, putting these professionals at risk for suicide. Just one risk factor can lead to suicide, and our survey identified 110 professionals with three or more risk factors. EMS professionals between the ages of 25–39 reported the highest number of risk factors. This age group has also been overrepresented in EMS professional suicides in Rhode Island over the past 15 years.

Mental health stigma is an issue in the RI EMS community. Answers to stigma-related questions in **Table 5** suggest that RI EMS professionals

feel mental health is stigmatized, given answers to stigma questions did not reach an overwhelming positive majority. Some 25.5 percent of EMS professionals aged 25–39 either disagree or strongly disagree with the statement: “My agency considers mental health important.” This is the largest percentage of any age group to report this finding. Given that these professionals are more likely to be in junior leadership positions based on national recommendations for EMS leadership qualifications,¹⁵ they represent the bridge between the longest-serving professionals and the newest professionals. Their response to this question is particularly relevant because junior leadership interacts significantly with both staff and senior leadership, giving them the broadest view of organizational values. In addition, 44.4 percent of all respondents reported either a negative or neutral response to the same question. These data indicate that stigma is still a barrier to mental health care for many EMS professionals in Rhode Island. With a community as small as Rhode Island EMS, peer response plays a large part in a professional’s willingness to seek help.

Rhode Island EMS professionals’ most common reasons for not receiving mental health support, are, “I didn’t think it was needed,” and “I already possess sufficient coping skills.” These results would suggest that awareness of mental health’s importance and de-stigmatization of mental health care through the entire chain of command is essential to provide reform. EMS professionals need to recognize when it is imperative to seek help while departmental awareness and recognition of individuals in need is enhanced, and individuals can receive support and help without negative consequences.

Data addressing the accessibility of mental health resources show that there is a significant number of professionals who do not have access to mental health resources via their agency. With only 65.6 percent of EMS professionals with guaranteed resources, this leaves the remaining providers in need of locating and paying for services on their own. The one resource that is available to all providers is Critical Incident Stress Management (CISM). RI has a CISM program that, upon request, will send a team member to an agency to facilitate one group discussion after a particularly traumatic incident. In addition, CISM team members will refer EMS professionals to other mental health resources for additional support. It is also important to note that CISM debriefings are typically organized at the request of agency leadership and is most often optional for EMS professionals to attend.¹⁶ As a result, if an agency does not have a strong mental health culture, these debriefings will not occur. Although studies have not shown CISM single-session debriefing to be as effective as intended, as illustrated by a 2002 paper that found no significant benefit from single session psychological debriefing following trauma, the EMS professionals with suicidal ideation in our population reported that they valued CISM intervention.¹⁷ Of the professionals with suicidal

ideation who had completed a CISM debrief, 73.1 percent feel that CISM is helpful. In addition, of the professionals with suicidal ideation who had not completed a CISM debrief, 45.6 percent of professionals feel that CISM is helpful, with an additional 38.9 percent with a neutral opinion. These data would suggest that CISM could be beneficial to those who are most at risk for a suicide attempt and should be made broadly available as an optional resource.

CONCLUSION

EMS professionals in the State of Rhode Island experience job-related stressors that can degrade their mental health and put them at risk for suicide. The future of the EMS profession is projected to be significantly affected by high rates of mental health struggles, substance misuse, and burnout.¹⁸ Many EMS professionals do not feel as though they need help when stressed, which will delay care should a mental health challenge occur. Longitudinal studies would need to occur to determine how many professionals leave EMS due to mental health issues. According to the National Registry of EMTs, the number of EMS professionals being certified increases each year.¹⁹ This indicates that there is not necessarily a lack of interest, but rather a lack of sufficient incentive to work in such a mentally grueling field without adequate mental health support.²⁰ Increased funding for mental health programming targeted at the EMS system, in addition to de-stigmatization of mental health in the EMS community is essential to combat these challenges and ensure the safety of those who provide life-saving care to Rhode Islanders.

References

1. Martin CE, Tran JK, Buser SJ. Correlates of suicidality in firefighter/EMS personnel. *J Affect Disord*. 2017 Jan 15;208:177-183. doi: 10.1016/j.jad.2016.08.078. Epub 2016 Oct 15. PMID: 27788381.
2. Data Source: EMS Records by Event Date. Biospatial, Inc. RIDOH. Aug 2024
3. EMTs and Paramedics [Internet]. U.S. Bureau of Labor Statistics; 2024 [cited 2024 Aug 19]. Available from: <https://www.bls.gov/ooh/healthcare/emts-and-paramedics.htm>
4. Rhode Island Department of Health Center for EMS. 2023 Annual Report. [Internet]. [cited 2024 Aug 10]. Available from: <https://health.ri.gov/publications/annualreports/2023CenterForEmergencyMedicalServices.pdf>
5. Emergency Medical Services (Licensing) [Internet]. [cited 2024 Aug 19]. Available from: <https://health.ri.gov/licenses/detail.php?id=284>
6. Vigil NH, Grant AR, Perez O, Blust RN, Chikani V, Vadeboncoeur TF, Spaitte DW, Bobrow BJ. Death by Suicide-The EMS Profession Compared to the General Public. *Prehosp Emerg Care*. 2019 May-Jun;23(3):340-345. doi: 10.1080/10903127.2018.1514090. Epub 2018 Sep 14. PMID: 30136908.
7. Vigil NH, Beger S, Gochenour KS, Frazier WH, Vadeboncoeur TF, Bobrow BJ. Suicide Among the Emergency Medical Systems Occupation in the United States. *West J Emerg Med*. 2021 Jan 20;22(2):326-332. doi: 10.5811/westjem.2020.10.48742. PMID: 33856319; PMCID: PMC7972356.

8. Virginia Department of Health [Internet]. [cited 2025 Feb 10]. Available from: <https://www.vdh.virginia.gov/emergency-medical-services/emergency-operations-2/healthandsafety/mental-health-resiliency-resources/2019-ems-provider-mental-health-survey/>
9. Rivard MK, Cash RE, Mercer CB, Chrzan K, Panchal AR. Demography of the National Emergency Medical Services Workforce: A Description of Those Providing Patient Care in the Prehospital Setting. *Prehosp Emerg Care*. 2021 Mar-Apr;25(2):213-220. doi: 10.1080/10903127.2020.1737282. Epub 2020 Mar 24. PMID: 32119575.
10. Sullivan F, Williams KA, Rhodes J. An overview of prehospital emergency medical services. *RI Med J* (2013). 2013 Dec 3;96(12): 24-7. PMID: 24303513.
11. Defusings and Debriefings [Internet]. 2020 [cited 2025 Feb 10]. Available from: <https://rhodeislandcism.com/defusings-and-debriefings/>
12. Till B, Tran US, Niederkrotenthaler T. Relationship Satisfaction and Risk Factors for Suicide. *Crisis*. 2017 Jan;38(1):7-16. doi: 10.1027/0227-5910/a000407. Epub 2016 Jul 22. Erratum in: *Crisis*. 2017 Jan;38(1):63. doi: 10.1027/0227-5910/a000437. PMID: 27445016.
13. Applebaum KM, Asfaw A, O'Leary PK, Busey A, Tripodis Y, Boden LI. Suicide and drug-related mortality following occupational injury. *Am J Ind Med*. 2019 Sep;62(9):733-741. doi: 10.1002/ajim.23021. Epub 2019 Jul 12. PMID: 31298756; PMCID: PMC7485601.
14. Substance Abuse and Mental Health Services Administration. (2023). *Key substance use and mental health indicators in the United States: Results from the 2022 National Survey on Drug Use and Health* (HHS Publication No. PEP23-07-01-006, NSDUH Series H-58). Center for Behavioral Health Statistics and Quality, Substance Abuse and Mental Health Services Administration. <https://www.samhsa.gov/data/report/2022-nsduh-annual-national-report>
15. Three levels of EMS officers - National EMS Management Association [Internet]. [cited 2024 Aug 19]. Available from: <https://www.nemsma.org/page/ThreeLevelsofEMSOfficers>
16. Three levels of EMS officers - National EMS Management Association [Internet]. [cited 2024 Aug 19]. Available from: <https://www.nemsma.org/page/ThreeLevelsofEMSOfficers>
17. Emmerik, et al. Single session debriefing after psychological trauma: a meta-analysis - *The Lancet*. September 7, 2002;Vol 360. Rose SC, Bisson J, Churchill R, Wessely S. Psychological debriefing for preventing post-traumatic stress disorder (PTSD). *Cochrane Database of Systematic Reviews* 2002, Issue 2.
18. Crowe RP, Bower JK, Cash RE, Panchal AR, Rodriguez SA, Olivo-Marston SE. Association of Burnout with Workforce-Reducing Factors among EMS Professionals. *Prehosp Emerg Care*. 2018 Mar-Apr;22(2):229-236. doi: 10.1080/10903127.2017.1356411. Epub 2017 Aug 25. PMID: 28841102.
19. National Registry of Emergency Medical Technicians. National Registry of Emergency Medical Technicians Annual Report. [Internet]. [cited 2024 Aug 10]. Available from: <https://indd.adobe.com/view/4405972b-c580-46cd-9565-167a5cd9b62c>
20. Kurth JD, Powell JR, Gage CB, Fauvel AD, Crowe RP, Cash RE, Panchal AR. Evaluating changes in the emergency medical services workforce: A preliminary multistate study. *J Am Coll Emerg Physicians Open*. 2023 May 25;4(3):e12975. doi: 10.1002/emp2.12975. PMID: 37251350; PMCID: PMC10211462.

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Novel Patient Intake Survey for the Diagnosis and Management of Hip Osteoarthritis

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ABSTRACT

INTRODUCTION: Hip osteoarthritis (OA) is a common cause of hip pain in adults and a frequent presentation in primary care, emergency departments, and orthopedic clinics. To improve patient triage and optimize clinical efficiency, we developed a nine-item intake survey designed to assess patient symptoms and prior treatments. This study aimed to evaluate the survey's ability to differentiate hip OA from other hip pathologies and assess its correlation with treatment recommendations.

METHODS: New patients presenting with hip pain were administered a nine-item multiple choice survey. Each response was assigned a score, and the total cumulative score was recorded. Diagnoses and treatment recommendations, including total hip arthroplasty (THA), were documented. Logistic regression was used to assess associations between the survey scores and both diagnosis and treatment recommendations. Receiver operating characteristic (ROC) analysis and Youden's J statistics were applied to determine the optimal survey score threshold for diagnosing hip OA.

RESULTS: The survey effectively distinguished hip osteoarthritis from other hip pathologies based upon cumulative score. ROC analysis identified a total score of ≥ 9 as the optimal threshold, maximizing sensitivity (83.3%) and specificity (55.9%) for diagnosing hip OA. The positive predictive value for this threshold was 78.6%. Additionally, higher total survey scores were significantly associated with the recommendation for THA.

CONCLUSION: This study demonstrates that a simple nine-item, patient-reported survey can reliably differentiate hip OA from other hip conditions and may assist in guiding treatment decisions. Implementing such tools in primary care, emergency medicine, and orthopedic settings could enhance early diagnosis and streamline referrals.

LEVEL OF EVIDENCE: III

INTRODUCTION

Hip osteoarthritis (OA) is a progressive degenerative joint disease and a leading cause of pain, disability, and rising healthcare costs in the United States.¹ Its incidence is projected to increase significantly through 2050, largely due to the aging population and growing prevalence of obesity.² Age is one of the strongest predictors of OA, with prevalence rising sharply in individuals aged 65 years or older.³ Additionally, females are at a greater risk of developing OA compared to males, with studies suggesting differences in disease progression and severity.⁴ Other risk factors include genetic predisposition, occupations requiring prolonged standing and heavy lifting, prior trauma, obesity, and dietary factors.⁵⁻⁷

First line of treatment options for OA includes lifestyle modification, physical therapy (PT), analgesic medications, and intra-articular injections. While these options may provide symptomatic relief, none have been proven to halt disease progression.⁸ For patients with end-stage hip OA, elective primary total hip arthroplasty (THA) remains the gold standard of treatment, offering substantial pain relief and improved function.^{9,10} Patient selection for THA is multifactorial, requiring the orthopedic surgeon to consider age, comorbidities, body mass index (BMI), radiographic severity, pain levels, functional limitations, and physical deformity.¹¹

The demand for THA is expected to rise substantially in the coming decades, with utilization in the United States projected to increase by 284% by 2040.² Meeting this demand requires not only a need for more fellowship-trained adult reconstruction surgeons, but also the implementation of efficient systems to optimize clinical workflows and resource allocation. The emergence of artificial intelligence (AI) in healthcare presents opportunities to reduce administrative burdens, improve patient satisfaction, and enhance diagnostic and treatment planning.¹² As AI becomes increasingly integrated into clinical workflows, validated standardized surveys may serve as valuable tools for streamlining diagnosis and treatment recommendations.

This study aims to evaluate the effectiveness of a short-form, nine-item survey as a diagnostic tool for hip OA and as a potential aid in surgical decision-making. As part of a quality improvement initiative, our adult reconstruction practice implemented this survey for all new patients presenting with hip pain at their initial visit. The survey was designed to supplement the diagnostic process, providing structured

clinical data to assist the surgeon in making accurate diagnoses and recommending appropriate treatment plans. We hypothesized that the survey would effectively differentiate patients with hip OA from those with other causes of hip pain and that total survey scores would correlate with the surgeon's recommendation for THA.

METHODS

This study received IRB approval by the Lifespan health system. Between October 2021 and April 2023, a total of 100 patient-completed intake surveys were collected and reviewed from an Adult Reconstruction Clinic within a large orthopedic practice. The survey consisted of nine multiple choice questions assessing patient demographics (age, gender, occupation), pain characteristics (location, duration, functional limitations) and previous treatments for hip pain [Table 1]. These nine questions were developed based on physician experience and another similar questionnaire used to diagnose knee OA.¹³ Answer choices were designed to differentiate patients at higher risk for severe hip OA from those with mild OA or alternative hip pathologies. The surveys were administered during the intake process of the patient visit in a blinded fashion. They were then seen immediately after by the surgeon, who did not look at the survey answers.

Following survey completion, each question was scored individually, and a cumulative score was calculated. Responses were assigned point values of 0, 1, or 2, where 0 indicating the lowest likelihood of severe hip OA and 2 indicating the highest likelihood. After survey collection, patient charts were retrospectively reviewed to document the attending surgeon's initial diagnosis and treatment recommendations. Diagnoses included hip OA, greater trochanteric bursitis (GTB), lower back pain, or other hip-related conditions. Treatment recommendations were recorded and included physical therapy (PT), non-steroidal anti-inflammatory drugs (NSAIDs), intra-articular hip injections, or total hip arthroplasty (THA).

Statistical Analysis

Descriptive statistics were used to summarize study variables. Continuous variables were reported as means with standard deviations, while categorical variables were presented as frequencies and percentages. Logistic regression was employed to assess the association between total survey score, individual survey responses, and treatment recommendations (i.e., clinical diagnosis and intervention recommendation). Odds ratios (ORs) with 95% confidence intervals (CIs), and model c statistics were reported. Classical sandwich estimation was utilized to protect against model misspecification, while a p-value of <0.05 was used to determine statistical significance. A separate model was run for each treatment recommendation and pain score combination. In the modeling, total survey score was treated as a

continuous variable while the individual survey responses were treated as categorical variables. The resulting ORs should be interpreted as the likelihood of having the diagnosis or intervention recommendation of interest compared to all others, with ORs >1 (ORs <1) indicating that increases in the pain score are associated with a greater (lower) likelihood of having outcome of interest. Receiver operating characteristic (ROC) analysis and Youden's J statistics were used to determine the optimal survey score threshold for diagnosing hip OA. Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and corresponding 95% confidence intervals (CI) were calculated to assess the diagnostic performance of the survey.

Table 1. Descriptive statistics

Variable	Mean (SD) or n (%)
Q1. What age range do you fall in?	
+0 Under 55 years of age	13 (13.0%)
+1 55 to 65 years of age	32 (32.0%)
+2 Over 65 years of age	55 (55.0%)
Q2. What gender do you identify with?	
+0 Female	61 (61.0%)
+1 Male	39 (39.0%)
+0 Self describe as _____	0 (0.0%)
Q3. How would you classify your current occupation?	
+0 Mainly desk work or retired	74 (74.0%)
+1 On my feet all day	21 (21.0%)
+2 Heavy lifting work	5 (5.0%)
Q4. Where is your pain concentrated?	
+0 Lower back/buttocks	7 (7.0%)
+1 Lateral side of the hip	54 (54.0%)
+2 Groin	39 (39.0%)
Q5. How long has your pain been going on?	
+0 Less than a month	5 (5.0%)
+1 1–6 months	35 (35.0%)
+2 6+ months	60 (60.0%)
Q6. When did the pain start?	
+0 During activity	15 (15.0%)
+1 No specific incident/woke up with pain	69 (69.0%)
+2 Fall or trauma	16 (16.0%)
Q7. What have you currently done about your pain?	
+0 Nothing	9 (9.0%)
+1 Took medications or completed exercises+PT	91 (91.0%)
+2 Exercises/Stretching/	0 (0.0%)
Q8. How limited are you due to your joint pain?	
+0 Not Limited	11 (11.0%)
+1 Limiting my prior baseline activities	71 (71.0%)
+2 Fully limited	18 (18.0%)
Q9. Have you been previously told you have OA?	
+0 No	29 (20.0%)
+1 Yes, my primary care provider told me	26 (26.0%)
+2 Yes, an orthopedic specialist told me	54 (54.0%)

SD: Standard deviation; BMI: Body mass index; PT: Physical therapy; NSAID: Non-steroidal anti-inflammatory drugs

RESULTS

Patient Characteristics

Of the 100 completed surveys, the mean BMI of participants was 28.76 (SD: 6.07). At the time of survey completion, 55% of patients were over 65 years of age, 32% were between 55 and 65 years of age, while 13% were below the age of 55. The majority of the respondents were female (61%), and 74% reported being retired or working a sedentary desk job. Only 5% of patients worked in occupations requiring regular heavy lifting.

Regarding self-reported pain characteristics, most patients described lateral hip pain (54%) while 39% reported groin pain. The majority (69%) could not attribute their pain to a specific injury, and 60% had been experiencing symptoms for more than six months at the time of survey completion. Nearly all patients (91%) had previously attempted medications or PT for hip pain, and 54% had been told from an orthopedic specialist that they had hip OA.

Following chart review, 66% patients were diagnosed with hip OA at their first initial visit, while 16% were diagnosed with greater trochanteric bursitis. Physical therapy was the most frequently recommended treatment (45%), while surgery was recommended for 38% of patients [Table 1].

Diagnosis

Analysis of the association between the survey and clinical diagnosis showed that the total score was significantly related to the diagnoses of hip osteoarthritis ($p=0.001$), greater trochanteric bursitis ($p=0.007$), and low back pain ($p=0.01$). Odds ratios analysis demonstrated that higher total scores were associated with an increased likelihood of a hip OA diagnosis (OR=1.72), and a decreased likelihood of both GTB (OR=0.61) and low back pain (OR=0.72). The discriminative ability of the survey was strong, with associated c-statistics exceeding 0.70 for these diagnoses [Table 2].

ROC analysis combined with Youden's J statistic identified a total score of ≥ 9 as the threshold that maximized both sensitivity (83.3%) and specificity (55.9%) for diagnosing hip OA. At this threshold, the PPV was 78.6% and the NPV was 63.3% [Table 3].

Management

Logistic regression analysis found that none of the individual survey questions were significantly associated with the likelihood of receiving specific treatment recommendations including PT, medications, or injections. However, the total survey score was significantly correlated with the recommendation for surgery (OR=1.62, $p=0.0005$). The model's c-statistic (0.66) indicated moderate discriminative ability in predicting which patients were recommended for total hip arthroplasty [Table 4].

Table 2. Results of logistic regression model examining the association between the intake form total score and initial diagnosis ($P<0.05$ are bolded)

Initial Diagnosis	Mean Score	95% CI	Odds-ratio (95% CI)	P-Value	c
Hip Osteoarthritis	9.76	7.96–8.98	1.72 (1.24–2.38)	0.001	0.72
Greater Trochanteric Bursitis	8.19	7.42–8.96	0.61 (0.43–0.87)	0.007	0.70
Lower Back Pain	8.50	7.58–9.42	0.72 (0.55–0.94)	0.01	0.71
Other	9.00	7.56–10.44	0.88 (0.41–1.89)	0.75	0.51

Table 3. ROC data for all the diagnoses.

Diagnosis	Sensitivity Value (95% CI)	Specificity Value (95% CI)	Positive predictive value Value (95% CI)	Negative predictive value Value (95% CI)	Accuracy
Hip OA	0.83 (0.74–0.92)	0.56 (0.39–0.73)	0.79 (0.69–0.88)	0.63 (0.46–0.81)	74%
Greater trochanter bursitis	0.75 (0.66–0.84)	0.56 (0.32–0.81)	0.90 (0.83–0.97)	0.30 (0.14–0.46)	72%
Back pain	0.75 (0.66–0.84)	0.67 (0.40–0.93)	0.94 (0.89–0.99)	0.27 (0.11–0.42)	73%
Other	0.71 (0.61–0.80)	0.40 (0–0.83)	0.96 (0.91–0.99)	0.07 (0–0.16)	69%

Table 4. Results of logistic regression model examining the association between the intake form total score and recommended intervention. ($P<0.05$ are bolded)

Intervention	Odds-ratio (95% CI)	P-Value	c
PT	0.78 (0.60–1.02)	0.07	0.60
NSAID	0.81 (0.62–1.06)	0.12	0.60
Intra-articular injection	0.91 (0.65–1.27)	0.57	0.53
Total hip arthroplasty	1.62 (1.24–2.11)	0.0005	0.66

DISCUSSION

Total joint replacement, including total hip arthroplasty (THA), is projected to remain one of the most prevalent elective surgical procedures in the coming decades.¹⁴ Currently, over seven million Americans have undergone total joint arthroplasty, experiencing improved function and pain relief despite advanced osteoarthritis (OA).¹⁵ The continued success of this procedure, coupled with an aging population and rising obesity rates, is expected to drive a substantial

increase in THA utilization. Meeting this growing demand will require not only healthcare policy adjustments to expand the workforce of fellowship-trained adult reconstruction surgeons but also strategies to improve clinical efficiency.² Standardized, reliable patient-reported surveys offer a promising tool to streamline patient assessment, enhance surgeon planning, and optimize resource allocation. To our knowledge, no prior studies have evaluated the accuracy of a patient-reported survey as a diagnostic tool for severe hip OA requiring THA.

In this study, we examined the relationship between patient responses on a nine-item survey and an adult reconstruction surgeon's initial diagnosis and treatment recommendation. Our findings demonstrate that the cumulative score effectively differentiated patients diagnosed with hip OA from those with alternative conditions such as greater trochanteric bursitis or lower back pain. A cumulative score of ≥ 9 was identified as the optimal threshold, maximizing both sensitivity and specificity for hip OA diagnosis. Notably, the survey score was also significantly associated with the surgeon's recommendation for THA, suggesting its potential role in surgical decision-making.

Beyond its application in orthopedic specialty clinics, this survey may have significant value for primary care and emergency department (ED) physicians, who are often the first to evaluate patients with hip pain. Hip OA is a common complaint in both settings, yet differentiating OA from other causes of hip pain, such as bursitis, lumbar radiculopathy, or referred pain, can be challenging, particularly in time-limited encounters. A simple, intake survey could assist primary care providers in stratifying patients based on their likelihood of having hip OA, guiding earlier referrals to orthopedic specialists when surgical intervention may be needed. Similarly, in the ED setting, where musculoskeletal pain is a frequent complaint but advanced imaging and specialist consultation may not always be immediately available, this tool could provide a structured approach to risk-stratifying patients, ensuring that those with high scores receive appropriate follow-up while those with lower scores are directed toward nonoperative management.

This study represents the first of its kind to assess a novel nine-item patient-reported intake survey as a diagnostic aid for hip OA and a predictor of surgical intervention. As artificial intelligence (AI) continues to be integrated into healthcare, standardized questionnaires may serve as valuable screening tools to assist orthopedic clinical workflows and enhance AI-driven diagnostic models. By pre-screening patients before their initial consultation, such tools could improve efficiency, reduce wait times, and allow surgeons to focus on higher-risk patients requiring advanced interventions. In primary care and ED settings, incorporating this survey into initial patient evaluations could help expedite appropriate referrals, reduce unnecessary imaging, and improve overall patient care efficiency.

While our findings are promising, this study has several limitations. The short-form nature of the questionnaire restricts the breadth of patient demographic and clinical data captured. Factors such as family history, race/ethnicity, and dietary habits, which may influence hip OA risk and severity, were not accounted for.^{16,17} Another inherent limitation is the potential for reporting bias, as patient-reported outcomes can be influenced by individual pain tolerance, recall accuracy, and emotional state at the time of survey completion. Additionally, this study was conducted within a single adult reconstruction clinic, which may limit the generalizability of findings to broader orthopedic and primary care populations. Further research is warranted to validate these findings in larger, more diverse cohorts and to assess the survey's performance in primary care and emergency medicine, where early identification of hip OA could facilitate timely referrals and interventions. Moreover, the generation and weighting of the survey questions was based on surgeon experience and prior studies which may be vulnerable to biases.¹³ Future studies are needed to optimize the question selection and weighting to further improve the efficacy of our screening survey. Lastly, a senior arthroplasty attending selected patients for the osteoarthritis cohort based on the criteria of having a clinical exam and history as well as radiographic evidence consistent with osteoarthritis. We did not perform a formal evaluation of the radiographic presence or severity of osteoarthritis in this study. Future studies may investigate the relationship between clinical findings and radiographic osteoarthritis, as well as the correlation between survey scores and radiographic findings.

CONCLUSION

Patient-reported surveys may serve as valuable adjuncts in orthopedic clinical workflows by improving diagnostic efficiency and guiding treatment decisions. This study assessed a nine-item intake survey as a useful tool for distinguishing hip osteoarthritis from other causes of hip pain and demonstrated its correlation with total hip arthroplasty recommendations. Implementing structured intake surveys in orthopedic, primary care, and emergency medicine settings could facilitate earlier identification of hip OA, streamline referrals, and optimize patient management. Further research is needed to assess the survey's effectiveness in diverse clinical settings and its potential role in standardizing hip OA evaluation and treatment planning.

References

1. Glyn-Jones S, Palmer AJR, Agricola R, et al. Osteoarthritis. *Lancet* (London, England). 2015;386(9991):376-387. doi:10.1016/S0140-6736(14)60802-3
2. Singh JA, Yu S, Chen L, Cleveland JD. Rates of Total Joint Replacement in the United States: Future Projections to 2020-2040 Using the National Inpatient Sample. *J Rheumatol*. 2019; 46(9):1134-1140. doi:10.3899/jrheum.170990

3. Felson DT, Lawrence RC, Dieppe PA, et al. Osteoarthritis: new insights. Part 1: the disease and its risk factors. *Ann Intern Med.* 2000;133(8):635-646. doi:10.7326/0003-4819-133-8-200010170-00016
4. Srikanth VK, Fryer JL, Zhai G, Winzenberg TM, Hosmer D, Jones G. A meta-analysis of sex differences prevalence, incidence and severity of osteoarthritis. *Osteoarthritis Cartil.* 2005;13(9):769-781. doi:10.1016/j.joca.2005.04.014
5. Croft P, Coggon D, Cruddas M, Cooper C. Osteoarthritis of the hip: an occupational disease in farmers. *BMJ.* 1992;304(6837):1269-1272. doi:10.1136/bmj.304.6837.1269
6. Spector TD, MacGregor AJ. Risk factors for osteoarthritis: genetics. *Osteoarthritis Cartil.* 2004;12 Suppl A:S39-44. doi:10.1016/j.joca.2003.09.005
7. Jiang L, Tian W, Wang Y, et al. Body mass index and susceptibility to knee osteoarthritis: a systematic review and meta-analysis. *Jt Bone Spine.* 2012;79(3):291-297. doi:10.1016/j.jbspin.2011.05.015
8. Abramoff B, Caldera FE. Osteoarthritis: Pathology, Diagnosis, and Treatment Options. *Med Clin North Am.* 2020;104(2):293-311. doi:10.1016/j.mcna.2019.10.007
9. Skou ST, Roos EM, Laursen MB, et al. A Randomized, Controlled Trial of Total Knee Replacement. *N Engl J Med.* 2015; 373(17):1597-1606. doi:10.1056/NEJMoa1505467
10. Ethgen O, Bruyère O, Richy F, Dardennes C, Reginster JY. Health-related quality of life in total hip and total knee arthroplasty. A qualitative and systematic review of the literature. *J Bone Joint Surg Am.* 2004;86(5):963-974. doi:10.2106/00004623-200405000-00012
11. Katz JN. Total joint replacement in osteoarthritis. *Best Pract Res Clin Rheumatol.* 2006;20(1):145-153. doi:10.1016/j.berh.2005.09.003
12. Liu PR, Lu L, Zhang JY, Huo TT, Liu SX, Ye ZW. Application of Artificial Intelligence in Medicine: An Overview. *Curr Med Sci.* 2021;41(6):1105-1115. doi:10.1007/s11596-021-2474-3
13. Liu J, Daher M, Callanan T, et al. Improving Diagnostic Efficiency for Knee Osteoarthritis with the Knee Intake Patient Survey (KIPS). *R I Med J* (2013). 2025;108(2):69-73. <http://www.ncbi.nlm.nih.gov/pubmed/39878667>
14. Kurtz SM, Lau E, Ong K, Zhao K, Kelly M, Bozic KJ. Future young patient demand for primary and revision joint replacement: national projections from 2010 to 2030. *Clin Orthop Relat Res.* 2009;467(10):2606-2612. doi:10.1007/s11999-009-0834-6
15. Maradit Kremers H, Larson DR, Crowson CS, et al. Prevalence of Total Hip and Knee Replacement in the United States. *J Bone Joint Surg Am.* 2015;97(17):1386-1397. doi:10.2106/JBJS.N.01141
16. Johnson VL, Hunter DJ. The epidemiology of osteoarthritis. *Best Pract Res Clin Rheumatol.* 2014;28(1):5-15. doi:10.1016/j.berh.2014.01.004
17. Sinusas K. Osteoarthritis: diagnosis and treatment. *Am Fam Physician.* 2012;85(1):49-56. <http://www.ncbi.nlm.nih.gov/pubmed/22230308>

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Glioblastoma: Epidemiology and Imaging-Based Review

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ABSTRACT

Glioblastoma (GBM) is an aggressive brain tumor, commonly occurring in the frontal and temporal lobes. GBM is characterized by low survival rates, high recurrence rates, and unclear risk factors, making management a significant challenge. Anatomic magnetic resonance imaging (MRI), including T1-weighted, T2-weighted, and fluid-attenuated inversion recovery (FLAIR), is the gold standard for diagnosis of GBM. These techniques have lower accuracy in evaluating treatment response, as pseudoprogression and radionecrosis can mimic true tumor progression (TrTP). Advanced imaging options that offer physiologic information, such as diffusion-weighted imaging, MR perfusion, MR spectroscopy, and Positron Emission Tomography (PET), have shown promise in aiding diagnosis and treatment response monitoring. The first-line treatment for GBM is maximal safe neurosurgical resection, followed by adjuvant radiotherapy and temozolomide, an oral DNA alkylating agent. Current research is focused on optimizing imaging to evaluate TrTP and developing novel treatments to increase survival rates.

KEYWORDS: Glioblastoma; imaging; MRI; CT; pseudoprogression

EPIDEMIOLOGY

Glioblastoma (GBM) is a rapidly progressing and fatal malignancy, with a five-year survival rate of only 7.1%.¹ It accounts for 51.5% of all primary central nervous system malignant tumors. While GBM tumors most typically present in the frontal and temporal lobes, they can also occur in other cortical and subcortical structures.² These tumors rarely metastasize, but they are inherently invasive, making surgical resection with clear margins infeasible. By definition, GBM is a grade IV glioma without a mutation in *isocitrate dehydrogenase (IDH)*, also known as *IDH-wildtype*.³ GBM is conventionally differentiated from other gliomas by classic histological features, such as microvascular proliferation or necrosis. However, the 2021 WHO classification introduced new molecular criteria that can be used to upgrade a tumor

to GBM in the absence of the typical histological features. One of three molecular markers must be present: *telomerase reverse transcriptase (TERT)* promoter mutation, *epidermal growth factor receptor (EGFR)* amplification, or combined gain of whole chromosome 7 and loss of whole chromosome 10 (+7/−10).³ Molecular sequencing is routinely employed in characterizing newly diagnosed brain tumors to detect actionable mutations and holds promise in changing GBM treatment. For example, tumors with *BRAF-V600E* or *NTRK* mutations can be treated with targeted therapy.⁴ Additionally, immunotherapy can potentially be used to treat hypermutated phenotypes of GBM with high tumor burden, which are caused by mutated DNA mismatch repair genes or DNA polymerase complex.⁵

In Rhode Island (RI), the age-adjusted incidence of GBM has remained relatively steady, slightly increasing from 3.8 per 100,000 from 1995–1999 to 3.9 per 100,000 from 2015–2019 [Table 1].⁶ These rates are higher than the national rate, which has hovered around 3.2 per 100,000 over that same time period.⁷ Nationally, GBM incidence is markedly higher in males than in females, with 2020 incidence rates of 4.1 and 2.5 per 100,000, respectively. In addition, GBM incidence greatly increases with age, with 13.3 cases per 100,000 in the 65+ age group.⁸ Both of these national trends were observed at the state level from 1995–2019, as the mean age of RI GBM patients was 64.8 years old, with males representing 55.1% of those patients. Lastly, 97.4% of RI GBM patients over this time period were White.

The clearest modifier of GBM survival is age, with patients in the 65+ age group exhibiting the lowest five-year survival rate of all examined age groups at 3%, according to national data from 2016.⁸ Besides differences in age and sex, risk factors for developing GBM are not well-established. A recent

Table 1. Demographics and age-adjusted incidence rate of glioblastoma (ICD-O-3 9440/3) in RI (1995–2019)

Demographics	Mean Age (years)		Male (%)		White (%)	
	64.8 ± 14.0		55.1		97.4	
Incidence	1995–1999	2000–2004	2005–2009	2010–2014	2015–2019	
Age-adjusted incidence rate, per 100,000 individuals	3.8	3.8	3.2	3.6	3.9	

meta-analysis showed no increase in GBM risk with variables such as increased BMI, type 2 diabetes mellitus, alcohol consumption, NSAID use, or magnetic field exposure.⁹ Another study found a 17% increased incidence of GBM in the highest socioeconomic status counties, compared with the lowest socioeconomic counties, though researchers struggled to identify specific risk factors to support this association.¹⁰

Clinical presentations for GBM patients vary based on the brain regions impacted by the tumor itself or the tumor's mass effect, with symptoms ranging from focal deficits, such as motor weakness, visual disturbance and focal seizures, to global impairments, including headaches, syncope and generalized convulsions.¹¹ Following diagnosis, the typical goals of care focus on slowing the progression of GBM while preserving normal brain function. Specific tumor characteristics have been identified that aid in predicting how efficacious certain treatments will be for patients. For example, tumors with a methylated *O*-6-methylguanine-DNA methyltransferase (*MGMT*) gene are more likely to respond favorably to temozolomide (TMZ), the first-line chemotherapeutic in GBM treatment regimens, than those with an unmethylated *MGMT*.¹² With further establishment of trends relating tumor characteristics to responsiveness to treatment, care plans for GBM patients will continue to evolve to maximize treatment efficacy.

IMAGING

CT

Before the introduction of MRI, computed tomography (CT) was the neuroimaging gold standard for diagnosing GBM. Given its accessibility, CT is usually the first imaging modality in a patient's work-up for a suspected brain

lesion. On CT, the tumor can have a hypodense necrotic center with irregular, slightly hyperattenuating margins due to high cellularity, which can give a ring enhancement appearance on non-contrast imaging.¹³ Due to the infiltrative nature of the tumor, it may cross the midline, extending to the contralateral hemisphere via the genu, body, and the splenium of the corpus callosum. Typically, CT also shows calcification, hemorrhage, mass effect, and vasogenic edema surrounding the tumor (**Figure 1**). Additionally, GBM's high vascularity lends itself well to visualization via perfusion CT imaging, which highlights the brain microcirculation and usually demonstrates increased tumor blood flow, cerebral blood volume (CBV), and vascular permeability.¹⁴ These factors may help to distinguish GBM from grade 3 gliomas and other tumors, such as primary CNS lymphoma and metastatic brain tumors.^{14,15} However, once a brain tumor is suspected, MRI is the neuroimaging gold standard due to its high specificity and sensitivity for GBM evaluation.¹⁶

MRI

The gold standard for GBM imaging includes pre- and post-gadolinium (Gd) contrast-enhanced T1 weighted imaging (T1WI) (**Figure 2A,B**), T2 weighted imaging (T2WI), and fluid-attenuated inversion recovery (FLAIR) MRI (**Figure 2C**).¹⁷ Compared to CT, which uses density differences to distinguish tumor from normal tissue, MRI indirectly estimates tumor size by visualizing the gadolinium contrast that extravasates through the disrupted tumor vasculature. On T1WI, CSF appears hypointense (dark) and white matter tissue hyperintense (light), while the inverse is true on T2WI. FLAIR is similar to T2WI, except it attenuates normal CSF fluid, allowing easier detection of abnormal tissue. On pre-contrast T1WI, GBM appears as a hypointense or isointense mass with a central heterogeneous signal if

Figure 1. Non-contrast CT of the brain demonstrates an isodense lesion in the right frontotemporal brain associated with vasogenic edema and mass effect on the right lateral ventricle.

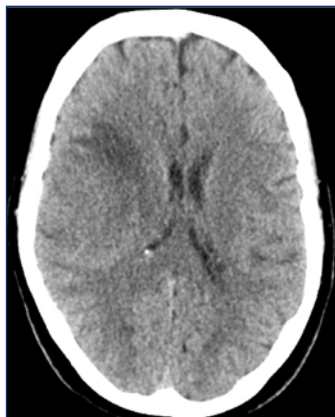
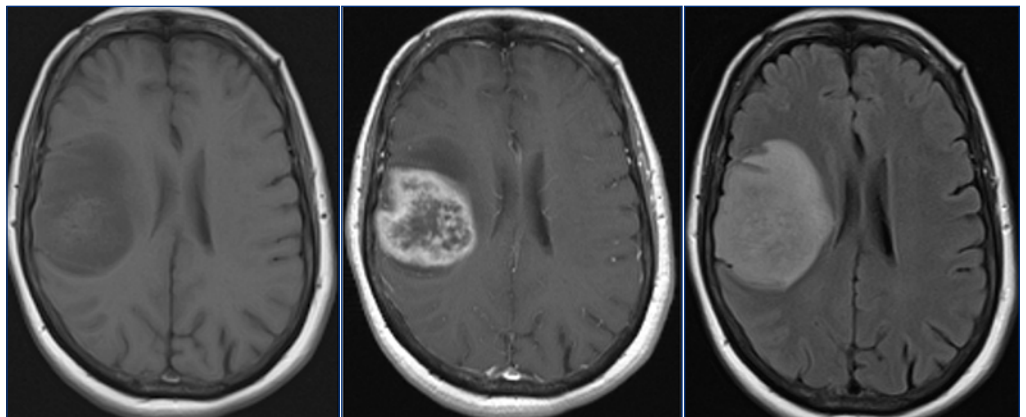


Figure 2. [A] Non-contrast enhanced T1-weighted imaging of the brain demonstrates a hypointense lesion in the right frontotemporal brain. [B] Post-contrast T1-weighted imaging demonstrates predominantly peripheral enhancement of the right frontotemporal lesion. [C] Axial T2-weighted fluid-attenuated inversion recovery imaging (FLAIR) demonstrates hyperintense signal associated with right frontotemporal lesion with mass effect resulting in right to left midline shift and effacement of the right lateral ventricle.

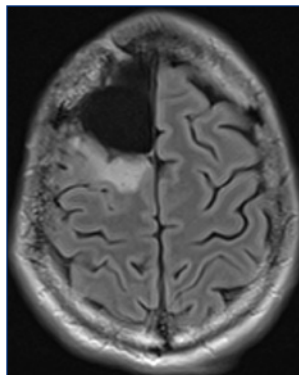


hemorrhage or necrosis is present.¹⁸ Post-contrast T1WI illustrates the vascularity of the brain and detects any breakdown in the blood-brain barrier (BBB) due to tumors, abscesses, and other pathologies. On post-contrast T1WI, the area of necrosis appears hypointense while regions of viable tumor demonstrate hyperintense enhancement. Even though contrast enhancement is a common feature of high-grade gliomas (HGGs), including GBM, around 9% of HGG tumors do not enhance.¹⁹ Additionally, it is difficult to determine the histologic grade of glioma on post-contrast T1WI.²⁰ GBM's highly infiltrative nature can help distinguish it from other gliomas, as it can extend into the contralateral hemisphere and deep nuclei of the cortex. Intratumoral hemorrhage and satellite lesions can also be highly suggestive of this malignancy. On T2WI/FLAIR, GBM appears as a hyperintense mass surrounded by vasogenic edema.

Although MR imaging is imperfect, it is necessary not only to guide the biopsy and confirmation of GBM but also to monitor treatment response. Post-contrast T1WI has conventionally been used for treatment response assessment but not without limitations. For example, a change in enhancement on imaging correlates with the breakdown of the blood-brain barrier (BBB), and, therefore, is not a direct measure of the tumor.²¹ Increased enhancement occurs with both true tumor progression (TrTP) and pseudoprogression (PsP), posing a challenge to clinicians. It is important to recognize PsP on imaging since it does not represent TrTP and should not alter treatment course (**Figure 3**). PsP typically occurs within the first six months post-radiotherapy (RT), especially in the first three months, and usually improves without any intervention. The exact mechanisms behind PsP are unknown, with one hypothesis that radiotherapy causes increased vascular permeability and edema due to endothelial cell death.²² A meta-analysis of HGGs showed that 36% of patients with MRI progression had PsP, 60% had TrTP, and 4% had an unknown outcome.²³

Given the clinical importance of distinguishing TrTP from PsP on imaging, standardized guidelines to differentiate the two are essential. The Response Assessment in Neuro-Oncology (RANO) criteria provide an objective measure of treatment response in gliomas, which helps guide management.²⁴

Figure 3. Axial T2-weighted fluid-attenuated inversion recovery imaging (FLAIR) demonstrates hyperintense signal posterior to right frontal resection cavity. This signal was not associated with post-contrast enhancement or elevated relative cerebral blood volume (rCBV).



The criteria take into account imaging factors like tumor size and presence of new lesions on MRI, as well as patient factors like clinical status and use of corticosteroids. The postradiotherapy MRI is used as the baseline for comparison with future scans. Depending on these factors, the treatment response is classified into complete response, partial response, stable disease, or progressive disease. Given the high incidence of PsP in the three months after radiotherapy, TrTP can only be confirmed by progression on repeat MRI during this period or with tissue sampling.

Radionecrosis is another treatment response that occurs due to radiation-induced damage of brain tissue and can mimic TrTP on imaging. In contrast to PsP, radionecrosis can occur six months to years after treatment and is usually irreversible.²⁵ Proposed mechanisms of radionecrosis include demyelination of white matter tracts, vascular endothelial damage, and changes in the fibrinolytic enzyme system. On MRI, findings of radionecrosis are most often seen at the tumor site, where the highest dose of radiation is delivered. On T2WI, this typically presents as an enhancing mass with a hyperintense necrotic center and surrounding edema. Given the similarity of the findings on MRI, it can be difficult to discern PsP and radionecrosis from TrTP. A meta-analysis looking at the diagnostic accuracy of MRI in evaluating treatment response in HGG patients found that anatomical MRI showed a pooled sensitivity of 68% and specificity of 77%.²⁶

Given the limitations of conventional MRI, more advanced imaging techniques have been implemented in the clinical management of HGG patients. Diffusion-weighted imaging (DWI) measures the random movement of water molecules in tissue, which is affected by tissue cellularity. To quantify the extent of water diffusion, an apparent diffusion coefficient (ADC) is calculated, where lower scores correspond to lower (more restricted) diffusion. In the context of tumors, ADC is impacted by the tumor size and the extracellular matrix complexity, making it an indirect measure of tumor cellularity.²⁷ Typically, non-enhancing cystic and necrotic areas have high ADC values, whereas the solid portion of GBM has lower ADC values (comparable to white matter regions). By showing the heterogeneity of diffusion in the brain, ADC mapping provides valuable insight into the type of lesion present, helping differentiate grade 3 gliomas from GBMs.²⁸ A meta-analysis found that pooled ADC maps were 71% sensitive and 87% specific in evaluating treatment response in HGG, showing higher accuracy than conventional anatomic MRI.²⁶

MR Perfusion

MR perfusion is another imaging modality that has shown promise in characterizing GBM physiology. This imaging can be done using three techniques: dynamic susceptibility contrast (DSC), dynamic contrast-enhanced (DCE), and arterial spin labeling (ASL) MR perfusion. DSC-MR perfusion,

or perfusion-weighted imaging (PWI), is the most widely used MR perfusion technique because it provides informative metrics such as CBV and cerebral blood flow.²⁷ This method relies on signal loss on T2 or T2* weighted images caused by the susceptibility effect from the Gd-based contrast agent passing through blood vessels. Studies have shown that GBM has an elevated CBV compared to lower-grade tumors and normal tissue, and that elevated CBV negatively correlates with prognosis.²⁹ DCE-MR perfusion, or “permeability” MRI, captures serial T1WI before, during, and after contrast to plot signal intensity over time in more detail than conventional T1WI. A useful metric derived from this technique is K-trans, which reflects the permeability of brain tissue and tumor angiogenesis.²⁷ A meta-analysis found that DSC- and DCE-perfusion have sensitivities of 87% and 92%, respectively, and specificities of 86% and 85%, respectively, making them more accurate than conventional MRI.²⁶ Lastly, ASL MR perfusion uses magnetically labeled water as a tracer and can be used to derive CBV. This technique is less widely used, potentially due to its lower signal-to-noise ratio and longer scanning time.²⁷ A recent meta-analysis found that DWI was slightly better than PWI (or DSC-MR perfusion) in terms of sensitivity (88% vs. 85%, respectively) and specificity (85% vs. 79%, respectively) in differentiating TrTP from PsP.³⁰ However, there was no significant difference in the area under the curve values between the two modalities (0.9156 for DWI and 0.9072 for PWI). These metrics highlight the strong performance of these modalities in treatment response evaluation of GBM, compared to conventional anatomic MRI. As seen, these methods not only help diagnose GBM but also show promise in evaluating treatment response and offering prognostic information.

MR Spectroscopy

Lastly, MR spectroscopy (MRS) is a useful technique for detecting metabolites present in brain tissue by using ¹H (proton) and phosphorus ³¹P resonances. In GBM, MRS typically reveals increased choline (indicating increased membrane turnover), lactate (indicating hypoxia and necrosis), and lipids (indicating necrosis).²⁷ Additionally, the tumor demonstrates decreased *N*-acetyl aspartate (indicating impaired neuron mitochondrial integrity) and myoinositol (indicating disruption of the BBB and osmotic equilibrium).^{27,31} A recent meta-analysis showed that elevated choline to *N*-acetyl aspartate ratio has high sensitivity and specificity for detecting TrTP.³² MRS has demonstrated a sensitivity of 91% and a specificity of 95% in evaluating treatment response in HGG.²⁶ Thus, this technique not only shows promise in distinguishing GBM from other tumors but also in differentiating TrTP from PsP and radionecrosis.

Positron Emission Tomography

Although not universally used for tumor monitoring, Positron Emission Tomography (PET) may provide additional benefits in diagnosing GBM and monitoring its progression. [¹⁸F]Fluoro-2-deoxy-D-glucose has traditionally been used as a surrogate for metabolic activity, which may be useful in differentiating metabolically active tumor from treatment-related changes. However, the brain's high glucose utilization at baseline decreases this radiotracer's specificity and limits its utility.³³ An amino acid tracer, ¹¹C-methyl-2-methionine, has been used for guiding tumor biopsies due to its elevated uptake in tumor tissue. While it may have additional benefits relative to the glucose tracer, it is less accurate than MR perfusion for monitoring tumors in the posttreatment setting.³³ Alternatively, ¹⁸F-fluoromisonidazole, a marker of hypoxia, shows higher uptake in GBM tumors than in other non-GBM gliomas, making this tool useful in the initial GBM workup.³⁴ An increase in this radiotracer's signal also correlates with early tumor recurrence, while a decreased signal is seen in those receiving bevacizumab therapy.³⁵ An additional radiotracer, ¹⁸F-fluorothymidine, is a marker of cell proliferation and helps differentiate low- and high-grade gliomas.³³ While different radiotracers each have specific applications in GBM workup, the use of PET for GBM is still largely investigational and remains an active area of research. There are logistical barriers to routinely using PET in a clinical setting, and the additional benefits of PET compared to the validated tools of MR perfusion/spectroscopy remain unproven.

TREATMENT

Given the complexity of GBM, the current standard is a multimodal treatment consisting of surgery followed by adjuvant radiotherapy (RT) and TMZ, an oral DNA alkylating agent.³⁶ For patients who qualify for surgery, the gold standard is maximal safe resection of the contrast-enhancing tumor, and those who underwent gross total resection (GTR) have shown improved survival.³⁷ However, despite extensive resection, many patients experience tumor recurrence near the prior surgical site.³⁸ Several studies have explored the supramaximal resection (SMR) of GBM as an alternative, which involves the removal of tissue beyond the contrast-enhancing region. A recent meta-analysis showed that, relative to GTR, SMR results in a significant reduction of disease progression and an increase in survival time.³⁹ Notably, SMR is mainly performed on non-eloquent brain tissue, which could explain the favorable complication rates in the literature. Currently, there are no established guidelines on the optimal extent of resection in SMR, as increasing resection margins without potentially impacting neurological function and prognosis proves challenging.⁴⁰

Surgical candidacy is determined after extensive imaging of the tumor and assessment of the patient's overall health.

Poor performance status and tumors that are multifocal, midline, or in deep brain areas are some factors that can preclude patients from resection.⁴¹ Although the number of patients with inoperable GBMs is not well cited, it is estimated to be between 35 and 40%.⁴¹ These patients usually undergo a stereotactic biopsy, which provides insight into tumor pathology and helps guide the treatment plan. Laser interstitial thermal therapy (LITT) has emerged as a new treatment modality for patients with unresectable GBMs.⁴² A laser is guided through a catheter using advanced intraoperative imaging to ablate the tumor area with high temperatures. A recent study showed that patients who underwent LITT had a median progression-free survival of four months and a median overall survival of 11 months.⁴³

After surgery, corticosteroids are added to treat the tumor-associated edema. Dexamethasone is the preferred medication due to its long half-life, high potency, and low mineralocorticoid activity.⁴⁴ Typically, the starting dose ranges from 2 to 16 mg depending on symptom severity, and it is administered for the shortest time possible, as prolonged dexamethasone use is detrimental to GBM patients.^{45,46} If the patient is unresponsive or intolerant to corticosteroids, bevacizumab, a VEGF-A monoclonal antibody, can be used instead to treat cerebral edema symptoms.⁴⁷

Typically, three to six weeks after surgery, patients receive radiation (30 fractions of 2 Gy over a six-week period for a total of 60 Gy) in addition to daily administration of oral TMZ.⁴⁶ Four weeks after the end of radiation, six 28-day cycles of adjuvant TMZ are done, where TMZ is administered for five consecutive days in each monthly cycle. This multimodal treatment was the result of the influential 2005 EORTC–NCIC phase III clinical trial, which demonstrated a median survival of 14.6 months for adjuvant TMZ and RT, significantly higher than the previous standard of care of adjuvant RT alone.⁴⁶ Although conventionally fractionated radiation is the gold standard if tolerated, hypofractionated radiation therapy is preferred in older patients or those with poor performance status.⁴⁷

In addition to the standard of care, two adjuvant treatments have been approved by the Food and Drug Administration (FDA): Gliadel® in 1996 and tumor-treating fields (TTF) in 2015.^{49,50} Although TMZ is the gold standard, only 20% of the drug in the plasma accumulates in the brain after oral intake, indicating inefficient delivery.⁵² Gliadel® is an implantable biodegradable wafer that delivers carmustine at the GBM resection cavity.⁵³ It has been shown to increase median survival to 18.2 months when combined with RT and TMZ, 3.6 months higher than RT and TMZ alone. However, its use remains limited due to its rigid structure, rapid release, and high cost. The TTF device has transducer arrays consisting of electrodes that are placed on the patient's scalp to deliver low-intensity alternating electric fields.⁵⁴ It has been shown to inhibit the proliferation of tumor cells and improve survival outcomes when combined

with maintenance TMZ relative to TMZ alone. Despite its demonstrated benefits in various trials, TTF adoption in clinical practice is still limited.

There have been several studies using immunotherapy in addition to RT and TMZ to treat GBM. However, most trials have failed to show survival benefits in patients, such as those using nivolumab and dendritic cell (DC) vaccines.^{55,56} A recent phase I trial pulsed an autologous DC vaccine with lysate from GBM stem cells, which was safe, well tolerated, and showed improved survival outcomes.⁵⁶ A phase III randomized controlled trial (the DCVax-L trial) showed that adding a DC vaccine to the standard of care showed increased overall survival for both newly diagnosed and recurrent GBM, but the study did not meet its target endpoints.⁵⁷ The BBB poses a significant challenge to chemotherapy administration, as it largely prevents the passage of drugs.⁵⁸ Intra-arterial chemotherapy can increase drug concentration in tumor areas despite the BBB limitation,⁵⁹ but late-phase trials are lacking. More recently, a phase I trial demonstrated that MR-guided focused ultrasound, which transiently disrupts the BBB, is safe when delivering systemic chemotherapy to glioma patients.⁶⁰ Lastly, injectable drug delivery systems that could bypass the BBB altogether, such as nanoparticles and hydrogels, have been a focus of recent research.⁶¹

CONCLUSION

GBM remains a significant clinical challenge due to its complexity and aggressiveness. Several advanced imaging modalities have shown promise when used in conjunction with conventional MRI for diagnosis and evaluation of treatment response. Tumor heterogeneity and the limitation of the BBB pose significant challenges to current and potential treatment options. Future research is focused on personalizing multimodal treatment based on tumor profile, disrupting the BBB to deliver chemotherapies, and developing novel drug delivery systems.

References 1-45

[Email corresponding author for complete reference list]

1. Price M, Ballard C, Benedetti J, et al. CBRUS statistical report: Primary brain and other central nervous system tumors diagnosed in the United States in 2017–2021. *Neuro Oncol.* 2024;26(Suppl 6):vi1–vi85. PMID: 39371035
2. Davis ME. Glioblastoma: Overview of disease and treatment. *Clin J Oncol Nurs.* 2016;20(5 Suppl):S2–S8. PMID: 27668386
3. Louis DN, Perry A, Wesseling P, et al. The 2021 WHO classification of tumors of the central nervous system: a summary. *Neuro Oncol.* 2021;23(8):1231–1251. PMID: 34185076
4. Padovan M, Maccari M, Bosio A, et al. Actionable molecular alterations in newly diagnosed and recurrent IDH1/2 wild-type glioblastoma patients and therapeutic implications: a large mono-institutional experience using extensive next-generation sequencing analysis. *Eur J Cancer.* 2023;191:112959. PMID: 37481865

5. Bouffet E, Larouche V, Campbell BB, et al. Immune checkpoint inhibition for hypermutant glioblastoma multiforme resulting from germline biallelic mismatch repair deficiency. *J Clin Oncol*. 2016;34(19):2206-2211. PMID: 27001570
6. Rhode Island Cancer Registry
7. Surveillance Research Program, National Cancer Institute. SEER*Explorer: An interactive website for SEER cancer statistics. Published April 19, 2023. Updated June 8, 2023. Accessed October 27, 2023. <https://seer.cancer.gov/statistics-network/explorer/>
8. Miller KD, Ostrom QT, Kruchko C, et al. Brain and other central nervous system tumor statistics, 2021. *CA Cancer J Clin*. 2021;71(5):381-406. PMID: 34427324.
9. Yoshikawa MH, Rabelo NN, Telles JPM, Figueiredo EG. Modifiable risk factors for glioblastoma: a systematic review and meta-analysis. *Neurosurg Rev*. 2023;46(1):143. PMID: 37340151.
10. Cote DJ, Ostrom QT, Gittleman H, et al. Glioma incidence and survival variations by county-level socioeconomic measures. *Cancer*. 2019;125(19):3390-3400. PMID: 31206646.
11. Wong ET, Wu JK. Overview of the clinical features and diagnosis of brain tumors in adults. UpToDate. Published October 19, 2023. Accessed May 27, 2025. <https://www.uptodate.com/contents/overview-of-the-clinical-features-and-diagnosis-of-brain-tumors-in-adults>
12. Della Monica R, Cuomo M, Buonaiuto M, et al. MGMT and whole-genome DNA methylation impacts on diagnosis, prognosis and therapy of glioblastoma multiforme. *Int J Mol Sci*. 2022;23(13):7148. PMID: 35806153.
13. Rees JH, Smirniotopoulos JG, Jones RV, Wong K. Glioblastoma multiforme: radiologic-pathologic correlation. *Radiographics*. 1996;16(6):1413-1438. PMID: 8946545.
14. Shankar JJ, Woulfe J, Silva VD, Nguyen TB. Evaluation of perfusion CT in grading and prognostication of high-grade gliomas at diagnosis: a pilot study. *AJR Am J Roentgenol*. 2013; 200(5):W504-W509. PMID: 23617517.
15. Onishi S, Kajiwaru Y, Takayasu T, et al. Perfusion computed tomography parameters are useful for differentiating glioblastoma, lymphoma, and metastasis. *World Neurosurg*. 2018;119: e890-e897. PMID: 30099179.
16. Lee EJ, Ahn KJ, Lee EK, Lee YS, Kim DB. Potential role of advanced MRI techniques for the peritumoural region in differentiating glioblastoma multiforme and solitary metastatic lesions. *Clin Radiol*. 2013;68(12):e689-e697. PMID: 23969153.
17. Ellingson BM, Wen PY, Cloughesy TF. Modified criteria for radiographic response assessment in glioblastoma clinical trials. *Neurotherapeutics*. 2017;14(2):307-320. PMID: 28108885.
18. Carrete LR, Young JS, Cha S. Advanced imaging techniques for newly diagnosed and recurrent gliomas. *Front Neurosci*. 2022; 16:787755. PMID: 35281485.
19. Scott JN, Brasher PM, Sevick RJ, Rewcastle NB, Forsyth PA. How often are nonenhancing supratentorial gliomas malignant? A population study. *Neurology*. 2002;59(6):947-949. PMID: 12297589.
20. Pope WB, Brandal G. Conventional and advanced magnetic resonance imaging in patients with high-grade glioma. *Q J Nucl Med Mol Imaging*. 2018;62(3):239-253. PMID: 29696946.
21. Brandsma D, Stalpers L, Taal W, Sminia P, van den Bent MJ. Clinical features, mechanisms, and management of pseudoprogression in malignant gliomas. *Lancet Oncol*. 2008;9(5):453-461. PMID: 18452856.
22. Ellingson BM, Chung C, Pope WB, Boxerman JL, Kaufmann TJ. Pseudoprogression, radionecrosis, inflammation or true tumor progression? Challenges associated with glioblastoma response assessment in an evolving therapeutic landscape. *J Neurooncol*. 2017;134(3):495-504. PMID: 28382534.
23. Abbasi AW, Westerlaan HE, Holtman GA, Aden KM, van Dullemen S, van der Hoorn A. Incidence of tumour progression and pseudoprogression in high-grade gliomas: a systematic review and meta-analysis. *Clin Neuroradiol*. 2018;28(3):401-411. PMID: 28466127.
24. Wen PY, van den Bent M, Youssef G, et al. RANO 2.0: Update to the Response Assessment in Neuro-Oncology criteria for high- and low-grade gliomas in adults. *J Clin Oncol*. 2023;41(33):5187-5199. PMID: 37774317.
25. Kumar AJ, Leeds NE, Fuller GN, et al. Malignant gliomas: MR imaging spectrum of radiation therapy- and chemotherapy-induced necrosis of the brain after treatment. *Radiology*. 2000;217(2):377-384. PMID: 11058631.
26. van Dijken BRJ, van Laar PJ, Holtman GA, van der Hoorn A. Diagnostic accuracy of magnetic resonance imaging techniques for treatment response evaluation in patients with high-grade glioma: a systematic review and meta-analysis. *Eur Radiol*. 2017;27(10):4129-4144. PMID: 28332014.
27. Wirsching HG, Galanis E, Weller M. Glioblastoma. In: *Handbook of Clinical Neurology*. Vol 134. Elsevier; 2016:381-397. PMID: 26948367.
28. Gühr G, Horvath-Rizea D, Hekeler E, et al. Diffusion weighted imaging in high-grade gliomas: a histogram-based analysis of apparent diffusion coefficient profile. *PLoS One*. 2021;16(4):e0249878. PMID: 33857203.
29. Yun J, Yun S, Park JE, et al. Deep learning of time-signal intensity curves from dynamic susceptibility contrast imaging enables tissue labeling and prediction of survival in glioblastoma. *Am J Neuroradiol*. 2023;44(5):543-552. PMID: 37105676.
30. Tsakiris C, Siempis T, Alexiou GA, et al. Differentiation between true tumor progression of glioblastoma and pseudoprogression using diffusion-weighted imaging and perfusion-weighted imaging: systematic review and meta-analysis. *World Neurosurg*. 2020;144:e100-e109. PMID: 32777397.
31. Steidl E, Pilatus U, Hattingen E, et al. Myoinositol as a biomarker in recurrent glioblastoma treated with bevacizumab: a ¹H-magnetic resonance spectroscopy study. *PLoS One*. 2016;11(12):e0168113. PMID: 28033329.
32. Aseel A, McCarthy P, Mohammed A. Brain magnetic resonance spectroscopy to differentiate recurrent neoplasm from radiation necrosis: a systematic review and meta-analysis. *J Neuroimaging*. 2023;33(2):189-201. PMID: 36631883.
33. Chiang GC, Kovanlikaya I, Choi C, et al. Magnetic resonance spectroscopy, positron emission tomography and radiogenomics—relevance to glioma. *Front Neurol*. 2018;9:33. PMID: 29459844.
34. Hirata K, Terasaka S, Shiga T, et al. ¹⁸F-Fluoromisonidazole positron emission tomography may differentiate glioblastoma multiforme from less malignant gliomas. *Eur J Nucl Med Mol Imaging*. 2012;39(5):760-770. PMID: 22307533.
35. Suzuki T, Takei J, Fukasawa N, et al. ¹⁸F-Fluoromisonidazole-positron emission tomography and immunohistochemistry verified tumor oxygenation, stemness, and immunosupportive microenvironment after preoperative neoadjuvant bevacizumab for newly diagnosed glioblastoma. *World Neurosurg*. 2023;175:e1364-e1374. PMID: 37187346.
36. Tolcher AW, Gerson SL, Denis L, et al. Marked inactivation of O6-alkylguanine-DNA alkyltransferase activity with protracted temozolomide schedules. *Br J Cancer*. 2003;88(7):1004-1011. PMID: 12671695.
37. Lacroix M, Abi-Said D, Fourney DR, et al. A multivariate analysis of 416 patients with glioblastoma multiforme: prognosis, extent of resection, and survival. *J Neurosurg*. 2001;95(2):190-198. PMID: 11780887.
38. Stupp R, Hegi ME, Mason WP, et al. European Organisation for Research and Treatment of Cancer Brain Tumour and Radiation Oncology Groups; National Cancer Institute of Canada Clinical Trials Group. Effects of radiotherapy with concomitant and adjuvant temozolomide versus radiotherapy alone on survival in glioblastoma in a randomised phase III study: 5-year analysis

- of the EORTC-NCIC trial. *Lancet Oncol.* 2009;10(5):459-466. PMID: 15758009.
39. Mier-García JF, Ospina-Santa S, Orozco-Mera J, Ma R, Plaha P. Supramaximal versus gross total resection in glioblastoma, IDH wild-type and astrocytoma, IDH-mutant, grade 4, effect on overall and progression-free survival: systematic review and meta-analysis. *J Neurooncol.* 2023;164(1):31-41. PMID: 37561356.
 40. McGirt MJ, Mukherjee D, Chaichana KL, Than KD, Weingart JD, Quinones-Hinojosa A. Association of surgically acquired motor and language deficits on overall survival after resection of glioblastoma multiforme. *Neurosurgery.* 2009;65(3):463-469; discussion 469-470. PMID: 19687690.
 41. Fazenzy-Dörner B, Wenzel C, Veitl M, et al. Survival and prognostic factors of patients with unresectable glioblastoma multiforme. *Anticancer Drugs.* 2003;14(4):305-312. PMID: 12679735.
 42. Kamath AA, Friedman DD, Akbari SHA, et al. Glioblastoma treated with magnetic resonance imaging-guided laser interstitial thermal therapy: safety, efficacy, and outcomes. *Neurosurgery.* 2019;84(4):836-843. PMID: 30137606.
 43. Viozzi I, Guberinic A, Overduin CG, Rovers MM, Ter Laan M. Laser interstitial thermal therapy in patients with newly diagnosed glioblastoma: a systematic review. *J Clin Med.* 2021;10(2):355. PMID: 33477796.
 44. Kostaras X, Cusano F, Kline GA, Roa W, Easaw J. Use of dexamethasone in patients with high-grade glioma: a clinical practice guideline. *Curr Oncol.* 2014;21(3):e493-e503. PMID: 24940109.
 45. Pitter KL, Tamagno I, Alikhanyan K, et al. Corticosteroids compromise survival in glioblastoma. *Brain.* 2016;139(Pt 5):1458-1471. PMID: 27020328.

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Accidental Drug Overdose Deaths: Rhode Island – January 1, 2022–December 31, 2024

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INTRODUCTION

In 2022, 436 individuals lost their lives to an accidental drug overdose in Rhode Island (RI), the highest annual number of fatal overdoses ever recorded in RI.¹ In response to the state- and nation-wide increases in overdose fatalities, RI created a goal to reduce overdose deaths by 30% by 2030 through a series of state and community level initiatives.² This work aims to describe the current state of the overdose epidemic in RI, how overdose trends have changed since the implementation of the RI 2030 goals, and how overdose prevention and intervention efforts may need to shift to address these changes.

METHODS

We analyzed data from the Office of State Medical Examiners (OSME) for deaths that occurred in RI from January 1, 2022 to December 31, 2024 that were determined to be accidental drug overdoses by the state medical examiners.

To comply with the RI Department of Health Small Numbers Policy, we combined sex categories, with transgender males captured as male and transgender females captured as female. We combined race and ethnicity to create mutually exclusive categories; Hispanic or Latino of any race, non-Hispanic White, non-Hispanic Black, and non-Hispanic other/unknown race. Any counts fewer than five are suppressed.

To obtain rates of overdose by decedent demographics, counts were restricted to RI residents. Population estimates were obtained from CDC Wonder, with 2023 estimates used to calculate 2024 rates.³

Substances contributing to the cause of death are those identified by the medical examiner as a contributing cause, and do not just reflect the presence of a particular substance. These categories are not mutually exclusive, as more than one substance may contribute to the cause of death in a single overdose.

We used chi-square to compare categories by year and Fisher's exact tests when cell counts were fewer than five. All analyses were conducted using SAS [Version 9.4].

RESULTS

From January 1, 2022 to December 31, 2024 there were 1,169 accidental overdose fatalities that occurred in RI. Overall, most overdose decedents were male (71%), aged 35–64 (70%),

Table 1. Demographic characteristics of individuals who died of an accidental overdose in Rhode Island: January 1, 2022–December 31, 2024.

Decedent Demographic	Overall N=1,169 n (%)	2022 N=436 n (%)	2023 N=404 n (%)	2024 N=329 n (%)	p-value ¹
Sex²					
Male	825 (71)	314 (72)	280 (69)	231 (70)	0.6801
Female	344 (29)	122 (28)	124 (31)	98 (30)	
Age Category					
Less than 25	46 (4)	23 (5)	16 (4)	7 (2)	0.0326
25–34	205 (18)	92 (21)	68 (17)	45 (14)	
35–44	298 (25)	106 (24)	113 (28)	79 (24)	
45–54	273 (23)	95 (22)	96 (24)	82 (25)	
55–64	259 (22)	91 (21)	78 (19)	90 (27)	
65+	88 (8)	29 (7)	33 (8)	26 (8)	
Race/Ethnicity					
Non-Hispanic, Black	110 (9)	41 (9)	36 (9)	33 (10)	0.6899
Hispanic or Latino	198 (17)	79 (18)	66 (16)	53 (16)	
Non-Hispanic, White	841 (72)	305 (70)	297 (74)	239 (73)	
Non-Hispanic, Other/Unknown Race	20 (2)	11 (3)	5 (1)	<5	

Source: Office of State Medical Examiners. ¹Chi-square test. ²Transgender males are categorized as male. Transgender females are categorized as female.

and non-Hispanic White (72%; **Table 1**). Overdoses typically occurred in a home or apartment (69%). Most overdoses involved only illicit substances (60%) or involved a combination of illicit substances and prescription medications (28%; **Table 2**). Opioids, including fentanyl, were involved in 80% of overdose fatalities, with fentanyl specifically involved in 71%. Outside of opioids, the most common substances contributing to cause of death were cocaine (56%), alcohol (24%), antidepressants (13%), and benzodiazepines (12%).

From 2022 to 2024, overdose fatalities decreased 24.5% from 436 to 329. The distribution of decedents sex, race and ethnicity, and overdose location were similar from 2022 to 2024. The number of overdose fatalities increased among individuals aged 55–64, from 78 deaths in 2023 to 90 in

Table 2. Substances that contributed to cause of death for individuals who died of an accidental overdose in Rhode Island: January 1, 2022–December 31, 2024.

Overdose Circumstances	Overall N=1,169 n (%)	2022 N=436 n (%)	2023 N=404 n (%)	2024 N=329 n (%)	p-value ¹
Drug Type					
Illicit Only	704 (60)	282 (65)	257 (64)	165 (51)	<0.0001 ²
Prescription Only	125 (11)	43 (10)	33 (8)	49 (15)	
Combination	333 (28)	109 (25)	112 (28)	112 (34)	
Unknown/ Missing	7 (1)	<5	<5	<5	
Substances Contributing to Death					
Any Opioid	931 (80)	358 (82)	345 (85)	228 (69)	<0.0001
Fentanyl	826 (71)	323 (74)	314 (78)	189 (57)	<0.0001
Alcohol	276 (24)	110 (25)	77 (19)	89 (27)	0.0243
Any Stimulant	711 (61)	242 (56)	252 (62)	217 (66)	0.0099
Cocaine	652 (56)	219 (50)	234 (58)	199 (61)	0.0103
Amphetamines	128 (11)	42 (10)	47 (12)	39 (12)	0.5366
Antidepressants	147 (13)	42 (10)	49 (12)	56 (17)	0.0090
Antipsychotics	58 (5)	18 (4)	15 (4)	25 (8)	0.0329
Over the Counter	48 (4)	18 (4)	13 (3)	17 (5)	0.4167
Buprenorphine	31 (3)	11 (3)	10 (2)	10 (3)	0.8744
Methadone	113 (10)	37 (8)	37 (9)	39 (12)	0.2701
Benzodiazepines	144 (12)	47 (11)	49 (12)	48 (15)	0.2808
Overdose Location²					
Private	812 (69)	302 (69)	284 (70)	226 (69)	0.8843
Semi-Private	61 (5)	23 (5)	23 (6)	15 (5)	
Public	101 (9)	37 (8)	30 (7)	34 (10)	
Unknown/ Missing	195 (17)	74 (17)	67 (17)	54 (16)	

Source: Office of State Medical Examiners. ¹Chi-square test. ²Private included apartment or residence, semi-public included hotel, motel, shelter, nursing home, hospital, prison, group home, assisted living, or treatment facility, while public included theater, concert, show, office, park, school, bar/restaurant, roadway, or cemetery. ³Fishers exact test.

2024, and now represents the age group with the highest proportion of overdose fatalities (27%) in RI and the only age group to show an increase in deaths over this time frame. The proportion of deaths involving opioids dramatically declined from 85% in 2023 to 69% in 2024, while the proportion of deaths involving cocaine continued to increase from 58% to 61%. In 2024, 49% of overdose deaths had at least one prescription medication contributing to the cause of death, with 15% solely attributed to prescription medication, and 34% involving a combination of prescribed and illicit substances.

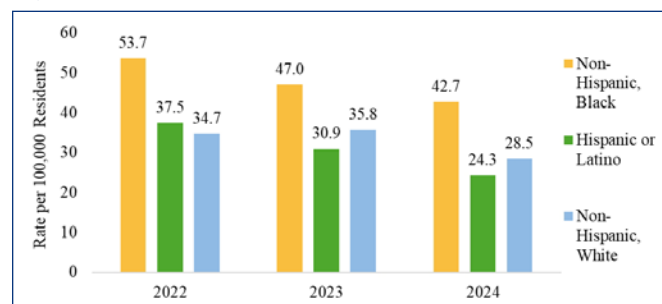
When looking at substances contributing to cause of death by age for 2024, opioid-involved deaths exhibited a near

linear relationship with 100% of deaths among individuals <25 involving opioids, dropping to 84% among individuals 25–34, 77% among 35–44, 68% among 45–54, 61% among 55–64, and 42% among 65+. In contrast, stimulant-involved deaths occurred in ~65% of deaths among all age groups. Of note, 74% of cocaine-involved deaths also involved fentanyl in 2022, which dropped to 59% in 2024.

When stratifying substances contributing to cause of death by race and ethnicity for 2024, cocaine-involved fatal overdoses were more common among non-Hispanic Black individuals (85%), when compared to Hispanic or Latino (53%) or Non-Hispanic White (58%) decedents. All other substances contributing to death showed relatively similar distributions by race and ethnicity.

When adjusting for the underlying population and looking at trends over time, overdose fatalities decreased among all racial and ethnic groups from 2022 to 2024, with the highest fatal overdose rate in 2024 among non-Hispanic Black (42.7 per 100,000) followed by non-Hispanic White (28.5 per 100,000) and Hispanic or Latino individuals (24.3 per 100,000; **Figure 1**).

Figure 1. Rate of accidental overdose deaths among Rhode Island residents, by decedent race and ethnicity: January 1, 2022–December 31, 2024.



Source: Office of State Medical Examiners. Note: Population denominator based on CDC WONDER single-race population estimates for each year accessed June 2, 2025; 2023 estimate applied for 2024 rates.

DISCUSSION

In 2024, RI experienced its second consecutive annual decrease in overdose deaths, with a 24.5% overall reduction in overdose mortality from 2022 to 2024, and a 36.3% reduction in opioid overdose deaths specifically. Encouragingly, the rate of overdose deaths decreased among all race and ethnicity groups; however, disparities remain, and non-Hispanic Black individuals still experience the highest burden of overdose deaths. Despite these promising trends, some changes in the overdose epidemic are concerning and may warrant changes in the prevention response. First, deaths among individuals aged 55 to 64 increased from 2023 to 2024, the only age group to do so. Second, the proportion of fatal overdoses that do not involve opioids has grown from 15% in 2023 to 31% in 2024, and for the first time since 2013 cocaine is responsible for more overdose deaths than

fentanyl. Finally, prescribed medications now play a role in 49% in overdose deaths, an increase from 36% in 2023.

These trends in RI mirror observed changes nationally with a 26.8% decrease in overdose fatalities from 2022 to 2024, and a 34.2% decrease in opioid overdose deaths specifically.⁴ Broader trends observed in RI, including a higher number of deaths among individuals 55–64 and an increased percentage of overdoses involving cocaine are seen in other neighboring states, including Connecticut.⁵

Considering the growing proportion of overdoses involving stimulants and prescription medications, and the increase among older individuals, prevention activities may need to be reassessed and shifted to address the changing epidemic. To prevent stimulant overdose fatalities, prevention activities might involve increasing: public education about the signs and symptoms of stimulant overamping (a stimulant overdose) [see **sidebar**], awareness of comorbid conditions that elevate an individual's risk of experiencing symptoms when using stimulants, knowledge on how to respond to someone overamping, and treatment opportunities available

Overamping: Signs & Symptoms

Overamping, or a stimulant overdose, occurs when someone is experiencing effects of a stimulant so severe that their health or safety may be at risk.

Because the effects stimulants have can vary based on the type of stimulant used, dose, how the stimulant was consumed, and any underlying physical or mental health issues, overamping can look different for different people.

Overamping can cause a variety of physical or psychological symptoms that can lead to overheating, stroke, seizure, heart attack, or a mental health crisis, including:

- Nausea or vomiting
- High temperature/sweating profusely
- Lots of sweating
- Headache
- Dizziness
- Confusion
- Fast heart rate
- Chest pain or discomfort
- Shortness of breath
- Feeling weak, light-headed, or faint
- Sudden numbness or weakness in arms or legs (especially on one side of the body)
- Sudden severe headache
- Sudden confusion, trouble speaking or difficulty understanding speech
- Shaking or convulsions
- Loss of consciousness
- Depression
- Agitation
- Paranoia
- Fear
- Anxiety
- Hallucinations

for individuals who use stimulants.⁶ To reach older adults, future work could explore new ways of reaching this population with messages more tailored to prevent, recognize, and respond to overdoses that often do not involve opioids. Future work should further investigate the growing role of prescribed medication in fatal overdoses both to ensure patients are not recipients of high-risk prescribing and to potentially better identify intervention and screening touch points that may be available when individuals at high risk of overdose are interacting with the healthcare system.

Although we are unable to show the specific factors attributable to the decrease in Rhode Island's overdose deaths for the last two years, it is likely due to several data-driven initiatives including: the large scale, statewide distribution of naloxone, increased focus on addressing social determinants of health and health disparities among RI communities, ongoing efforts to link individuals to care, and extensive media campaigns to increase Rhode Islanders' awareness of the ever-changing drug supply, harm reduction practices, and de-stigmatization of substance use. Additionally, these reductions would likely not have occurred without the tireless work of RI's community-based organizations and their dedicated staff with lived experience, healthcare professionals, municipalities, and government organizations aligned in a commitment to meet individuals where they are and provide free treatment/recovery options, harm reduction supplies, education, and basic needs services to individuals at risk of an overdose.

References

1. Weidele, HR, Hallowell BD. Accidental Drug Overdose Deaths in Rhode Island: January 1, 2019 – December 31, 2023. *Rhode Island Medical Journal*. 2024; 107(8): 61-63.
2. State of Rhode Island. (2025). *Charting A Course for the Future of the Ocean State*. <https://rhodeisland2030.ri.gov/sites/g/files/xkgbur1191/files/2025-01/RI2030-Version2025-final.pdf>
3. Single-race Population Estimates, United States, 2020-2023. July 1st resident population by state, age, sex, single-race, and Hispanic origin, on CDC WONDER Online Database. <http://wonder.cdc.gov/single-race-single-year-v2023.html>
4. Ahmad FB, Cisewski JA, Rossen LM, Sutton P. Provisional drug overdose death counts. National Center for Health Statistics. 2025. DOI: <https://dx.doi.org/10.15620/cdc/20250305008>
5. Connecticut Department of Public Health. (2025). *Drug Overdose Deaths in Connecticut Data Dashboard, 2015 to 2025*. [DataDashboard]. https://public.tableau.com/app/profile/heather.clinton/viz/SUDORS_Dashboard_final2/OverdoseDashboard
6. Centers for Disease Control and Prevention. A Stimulant Guide: Answers to Emerging Questions about Stimulants in the Context of the Overdose Epidemic in the United States. National Center for Injury Prevention and Control, Centers for Disease Control and Prevention, U.S. Department of Health and Human Services, 2022.

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**VITAL STATISTICS**

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PUBLIC HEALTH

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

VITAL EVENTS	REPORTING PERIOD		
	SEPTEMBER 2024	12 MONTHS ENDING WITH SEPTEMBER 2024	
	Number	Number	Rates
Live Births	863	10,897	10.3*
Deaths	954	10,663	10.1*
Infant Deaths	8	41	3.8#
Neonatal Deaths	6	30	2.8#
Marriages	493	6,716	6.3*
Divorces	224	2,546	2.4*

* Rates per 1,000 estimated population

Rates per 1,000 live births

Underlying Cause of Death Category	REPORTING PERIOD			
	MARCH 2024	12 MONTHS ENDING WITH MARCH 2024		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	211	2,388	217.6	3,007.5
Malignant Neoplasms	179	2,209	201.3	4,456.5
Cerebrovascular Disease	47	442	40.3	599.5
Injuries (Accident/Suicide/Homicide)	76	959	87.4	11,185.5
COPD	30	459	41.8	410.0

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 1,097,379 for 2020 (www.census.gov)

(c) Years of Potential Life Lost (YPLL).

NOTE: Totals represent vital events, which occurred in Rhode Island for the reporting periods listed above.

Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

SAVE THE DATE

RHODE ISLAND MEDICAL SOCIETY

CONVIVIUM

213TH ANNUAL MEETING & AWARDS DINNER

SEPTEMBER 25, 2025

THE SQUANTUM ASSOCIATION

6:00 PM RECEPTION

7:00 PM DINNER

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Turning the Tide: United States Volunteer Life Saving Corps in Rhode Island in the 1900s

MARY KORR
RIMJ MANAGING EDITOR

In 1929, **DR. MANCER W. TALCOTT**, Superintendent of the United States Volunteer Life Saving Corps (USVLSC), Rhode Island Department, comprised of over 200 members, presented its 24th annual report to the General Assembly.

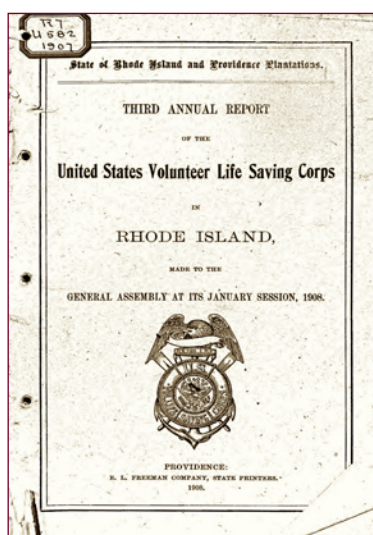


Figure 1. The third annual report of the United States Volunteer Life Saving Corps (USVLSC), Rhode Island, in 1908, features the group's insignia.

"dangerous bathing beaches and watering places." [See **Figure 4.**] Appropriated salaries were for two men known as quartermasters, who spent their entire time "inspecting, replacing, repairing, painting and splicing, and all the small detail work," to maintain the boats, stations, and equipment. The report gave the statistics for the prior year:

- 115 rescues, 37 of which required resuscitation
- 1,100 life buoys and lines at most of the dangerous places in Rhode Island
- 425 medicine chests; over 6,500 people assisted by these chests

He described the Corps, a supplement to the federal U.S. Life Saving Service, as "a humane organization whose 2,000 members voluntarily patrol beaches and dangerous places because they are interested in this work of saving human life. Every member is an expert swimmer and has to pass a rigid examination of rescuing a person from drowning, resuscitation, and First Aid." [See **Figures 1–3.**]

Funding by the State amounted to about \$4,000 per year, spent on the purchase of new surf boats and life buoys, and for medicine and First Aid supplies at the Corps' stations, located throughout the state's



"The doctor, on the other hand, who had probably had very little experience in resuscitation from drowning, thought that there was no possible hope of resuscitating this man, and so declared the case hopeless.

Figure 3. Rescue crew returning from a practice drill in 1927.



Figure 2. USVLSC boat, child sitting inside, and lifeguard at a Rhode Island beach in 1927.

Rescue/First Aid Demonstrations

Seasonal demonstrations were given in Rhode Island and other states with USVLSC divisions. The Corps also taught rescue and resuscitation methods to the Boy Scouts, at the YMCA, and at summer and church camps. The report stated that in 1928 over 50,000 people watched members "demonstrate the safest methods of rescue, including how to break away from a drowning man's death grip," and the best method of resuscitation, called the Schafer or prone-pressure method.

It was the standard method of artificial respiration, preceding mouth-to-mouth resuscitation in mid-century America. Introduced in 1903 by English physiologist **SIR EDWARD ALBERT SHARPEY-SCHAFER**, it involved placing the patient or drowning victim on his stomach and applying pressure to the lower part of the ribs [See **Figure 5.**]

Boy Directs Resuscitation after Doctor Declares Man Dead

One rescue story stands out in the 1929 annual report – that of a boy who challenged a physician on the beach. As Dr. Talcott recounted it: " 'He is dead, now run along and get out of the way.' That is what a doctor said on one of the Rhode Island beaches when a small boy asked if this doctor was not going to work more on a drowning victim. The boy, kidlike, was inquisitive and wanted to know why a man who had been brought ashore from the surf within fifteen minutes from the time he had gone underwater could not be revived.



Figure 4. Many accidents occurred at the canoe places, the report stated.
[SOURCE OF FIGURES 1-4: SECRETARY OF STATE, RI, DIGITAL STATE ARCHIVES]

"The boy, when he found out positively that the doctor was not going to do anything more, turned to two women and asked them if they would work on the drowned man. He told them what to do, because, as the kid said, he was not strong enough to do it alone. The boy told these two women how to lift the drowned man with their hands under his stomach and how to place him over their knee, and then he told them to hit hard between the two shoulders so as to get the water out of his air passages. And when they did this, the first thing that happened was he threw a large wad of chewing gum, which had got stuck in his throat, right out on the sand.

"They then laid him on the beach and the lad explained to them how to straddle the apparently drowned man and how to lift on the lower ribs and pressing in and then pressing downward and forward to work successfully the Schaefer resuscitation. And after a little over a half hour's work, this drowned man, who had been pronounced dead by a physician, showed definite signs of life and within a very short time after that was starting to breathe with regularity...the man was brought absolutely around safely and is alive today."

Dr. Talcott, in an aside, related that "in a demonstration [of the Schaefer method] which I was requested to give before over one thousand physicians and surgeons, a doctor came to me and said, 'If had only known what you were demonstrating ten years ago, I would have had a wife and three children alive today.'"

Rescue Medals Awarded

Dr. Talcott highlighted medals awarded by the Corps, "which are only given to the exceptional rescues." In 1928, at the West Elmwood Club House, medals were presented by Mayor **JAMES DUNNE** of Providence.

One was awarded to **ROBERT MCADAMS**, 13, who rescued a boy, James Rose. "Young McAdams was playing on the shore when somebody called to him that a boy was drowning. He ran up the shore and without stopping to take off any of his clothing, he dove in, but as he got to the boy, the boy grabbed him around the neck [the death grip] and, after giving him a very hard struggle, young McAdams broke away from him and then towed him to the shore."

Five men were also given medals for rescuing three boys who had fallen through the ice in mid-winter at Mashapaug Pond. Dr. Talcott described is as "one of the most dangerous rescues that had been made around the city of Providence in a good many years. There was no boat or any life-saving apparatus, and it was only after the most heartbreaking struggle and after all the men making the rescue had themselves gone under the ice several times, that the boys were finally brought to shore."

The Corps volunteers of all ages were truly heroes who turned the tide for drowning victims and their families. ❖

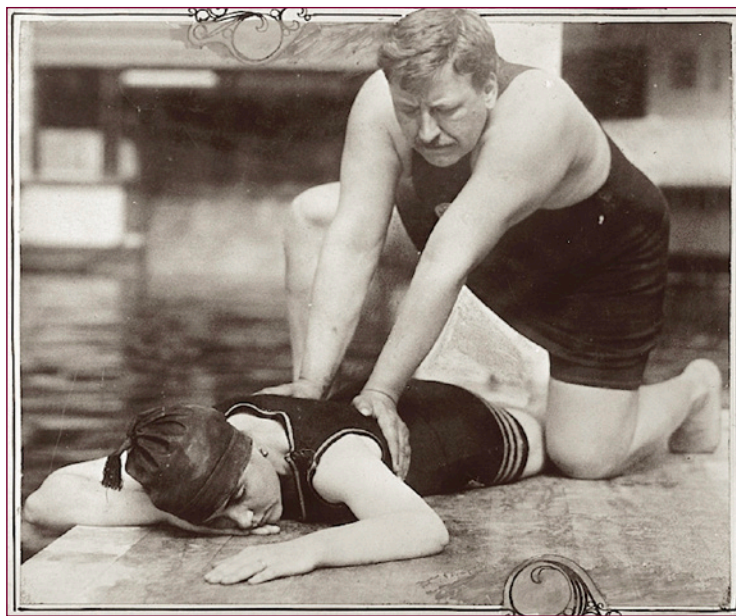


Figure 5. Wilbert E. Longfellow of Rhode Island, a leader in the USVLSC and later the Red Cross, demonstrating how to revive a near drowned person in 1919, using the Schaefer method of resuscitation. [LIBRARY OF CONGRESS]

For the above mentioned annual reports, visit:

https://catalog.sos.ri.gov/agents/corporate_entities/333

https://sosri.access.preservica.com/uncategorized/SO_b8e9a071-ac7e-46d2-ad17-3ae8bd922f1f/

Scientists develop high-performance MRI scanner in effort to define microscopic brain structures

BETHESDA, MD — A scientific team supported in part by the National Institutes of Health (NIH) has developed a new, ultra-high-resolution brain imaging system that can reconstruct microscopic brain structures that are disrupted in neurological and neuropsychiatric brain disorders. The new system is a significant advance over conventional magnetic resonance imaging (MRI) scanners that cannot visualize these tiny but clinically important structures.

The system, called the Connectome 2.0 human MRI scanner, overcomes a significant hurdle for neuroscientists: being able to bridge different brain regions and probe tiny structures necessary to define the “connectome,” the complex matrix of structural connections between nodes in the nervous system, and to do it noninvasively in living humans.

“This research is a transformative leap in brain imaging – pushing the boundaries of what we can see and understand about the living human brain at a cellular level,” said **JOHN NGAI, PhD**, Director of NIH’s Brain Research Through Advancing Innovative Neurotechnologies® Initiative, or The BRAIN Initiative®. “The new scanner lays essential groundwork for the BRAIN CONNECTS program’s ultimate goal of developing a wiring diagram for the human brain.”

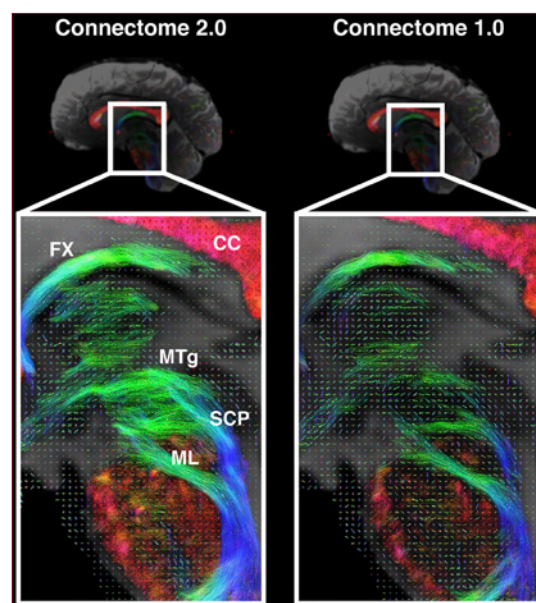
The scanner is innovative in two major ways: it fits snugly around the heads of living people, and it has many more channels than typical MRI systems. These

advances greatly increase the signal-to-noise ratio of the system, providing much sharper images of very small biological brain structures than previously possible. These technical upgrades will enable scientists to map human brain fibers and cellular architecture down to nearly single-micron precision to study how subtle changes in cells and connections relate to cognition, behavior, and disease.

In addition, the team showed that the scanner was safe in healthy research volunteers, revealing subtle microstructural differences (individual axon diameter or cell size) between individual brains. Before this new system, this was only feasible in postmortem or animal studies.

“Our goal was to build an imaging platform that could truly span scales – from cells to circuits,” said senior author **SUSIE HUANG, MD, PhD**, of the Department of Radiology at Mass General Hospital. “It provides researchers and clinicians with a powerful new tool to study brain architecture in health and disease, in real time.”

The research was funded in part by The BRAIN Initiative®. It supports the BRAIN Initiative Connectivity Across Scales (BRAIN CONNECTS) program, which aims to develop the research capacity and



Close-ups of the midline sagittal view for Connectome 2.0 (left) and Connectome 1.0 (right) protocols, showing diencephalic and brainstem pathways. Tractography results are shown superimposed onto the underlying fibre orientation distribution functions. [IMAGE BY CHIARA MAFFEI, PhD]

technical capabilities to generate wiring diagrams that can span entire brains across multiple scales. The findings were reported July 16 in *Nature Biomedical Engineering*.¹ ♦

Reference

1. Ramos-Llordén, G and Lee H-H, et al. Ultra-high gradient connectomics and microstructure MRI scanner for imaging of human brain circuits across scales. *Nature Biomedical Engineering*. 2025. <https://www.nature.com/articles/s41551-025-01457-x>

AMA, 79 medical societies back vaccination against respiratory viruses

CHICAGO — With respiratory viruses expected to surge this fall, the American Medical Association (AMA) and 79 leading medical societies, including the Rhode Island Medical Society, reaffirm their support for vaccination as the best way to protect against the flu, COVID-19, and RSV and their potentially serious complications. The organizations call on partners – insurers, hospitals, and public health agencies – to ensure these life-saving vaccines remain available to patients without cost sharing.

The open letter from the American Medical Association and medical society partners is available [here](#).

The full text is below:

An open letter to the American people:

With the severe influenza season the U.S. experienced during the 2024–25 respiratory virus season, and the recognition that we will likely see another surge in respiratory viruses this fall, we know strong physician leadership is essential to reducing preventable illness, hospitalizations, and death. Vaccines for influenza, RSV, and COVID-19 remain among the best tools to protect the public against these illnesses and their potentially serious complications – and physicians are among the most trusted voices to recommend them. We come together as physicians from every corner of medicine to reaffirm our commitment to these lifesaving vaccines.

Recent changes to federal immunization review processes raised concerns across the medical and public health community. In this moment of uncertainty, physicians must align around clear, evidence-based guidance for patients.

We commit to working together to promote public understanding and confidence in the use of vaccines to avoid another severe respiratory virus season and resurgence of vaccine-preventable illnesses and deaths. We call on our partners – from insurers to hospitals to public health agencies – to ensure vaccines remain available to patients without cost sharing.

The health and safety of the public remains our top priority, and we will continue to support evidence-based immunizations to help prevent severe disease and protect public health. ❖

Leading medical professional societies sue HHS for unlawful, unilateral vaccine changes

BOSTON — On July 7th, the American Academy of Pediatrics (AAP), American College of Physicians (ACP), American Public Health Association (APHA), Infectious Diseases Society of America (IDSA), Massachusetts Public Health Alliance (MPHA), Society for Maternal-Fetal Medicine (SMFM), and a pregnant physician, filed suit in American Academy of Pediatrics v. Robert F. Kennedy, Jr. in the U.S. District Court for the District of Massachusetts to defend vaccine policy.

The lawsuit charges that a coordinated set of actions by HHS and Secretary Kennedy were designed to mislead, confuse, and gradually desensitize the public to anti-vaccine and anti-science rhetoric, and that he has routinely flouted federal procedural rules. These actions include blocking CDC communications, unexplained cancellations of vaccine panel meetings at the FDA and CDC, announcing studies to investigate non-existent links between vaccines and autism, unilaterally overriding immunization recommendations, and replacing the diverse members of ACIP with a slate of individuals biased against sound vaccine facts.

The anonymous individual plaintiff in the lawsuit is a pregnant woman who is at immediate risk for being unable to get the Covid-19 vaccine booster because of the Secretarial Directive, despite her high risk for exposure to infectious diseases from working as a physician at a hospital.

“This administration is an existential threat to vaccination in America, and those in charge are only just getting started. If left unchecked, Secretary Kennedy will accomplish his goal of ridding the United States of vaccines, which would unleash a wave of preventable harm on our nation’s children,” said **RICHARD H. HUGHES IV**, partner at Epstein Becker Green and lead counsel for the plaintiffs. “The professional associations for pediatricians, internal medicine physicians, infectious disease physicians, high-risk pregnancy physicians, and public health professionals will not stand idly by as our system of prevention is dismantled. This ends now.”

The plaintiff organizations urge parents and patients to follow their qualified medical professionals’ vaccine guidance. AAP, ACP, APHA, IDSA, and SMFM websites provide evidence-based resources to help patients make decisions grounded in facts, not fear. ❖

W.M. Keck Foundation awards \$1.3M grant to Christopher Moore, PhD, to study how brain blood vessels relay real-time signals across the blood-brain barrier (BBB)

LOS ANGELES, CA — In June 2025, the W.M. Keck Foundation's Board of Directors awarded grants to 17 organizations, totaling \$19.6 million. Eleven grants were awarded in the Research Program, totaling \$12.8 million.

A \$1.3 million grant from the Foundation was awarded to Brown University for a research study led by **CHRISTOPHER MOORE, PhD**, associate director of the Carney Institute for Brain Science. It will fund research on how brain blood vessels relay real-time signals across the blood-brain barrier (BBB) directly to the brain. Dr. Moore's research team has found that blood vessels send signals through "plume events" that allow flashes of permeability across the BBB.

The abstract for the grant reads as follows:

Discovery of dynamic processes that enable rapid and focal brain-body communication

The brain evolved to meet challenging biological needs, with the mammalian forebrain integrating lifelong experience, ongoing sensations, and future predictions. This powerful computation relies on the quality of body information received at moments of choice and learning. Brain vasculature contains a rich supply of such body signals, delivered in their native chemical format, but researchers view this pathway as sluggish and diffuse, unsuited to real-time behavior due to the blood-brain barrier (BBB).

The recent discovery of Plume Events – rapid, local increases in BBB permeability timed to relevant behavioral events – suggest a dynamic solution for forebrain computation: brief vascular access when the risk is worth the information value. An investigator at Brown University and several of his collaborators will test key predictions, such as whether transient electro-calcium 'spikes' in vessels trigger these events, if Plume Events deliver impactful bio-active signals such as oxytocin, and if they are expressed across the forebrain.

The collaborative team, bringing diverse computational and biological expertise, will also create a broader community for insight through colloquia and retreats focused on understanding this new discovery.

Abstracts for all of this cycle's Keck research grants are available here: <https://www.wmkeck.org/our-focus-research/#focus-abstracts-research> ❖

AMA deeply concerned by reported USPSTF changes

Letter to HHS secretary urges retaining previously appointed Task Force members

CHICAGO – The American Medical Association (AMA) expressed "deep concern" directly to U.S. Health and Human Services Secretary **ROBERT F. KENNEDY** about news reports that he intends to remove all members of the U.S. Preventive Services Task Force (USPSTF).

"USPSTF plays a critical, non-partisan role in guiding physicians' efforts to prevent disease and improve the health of patients by helping to ensure access to evidence-based clinical preventive services," the AMA said in its letter. "As such, we urge you to retain the previously appointed members of the USPSTF and commit to the long-standing process of regular meetings to ensure their important work can continue without interruption."

The full text of the letter is below.

Dear Secretary Kennedy,

On behalf of the physician and medical student members of the American Medical Association (AMA), I am writing to express our deep concern with the recent reports of your intention to remove all of the members of the United States Preventive Services Task Force (USPSTF). As you know, USPSTF plays a critical, non-partisan role in guiding physicians' efforts to prevent disease and improve the health of patients by helping to ensure access to evidence-based clinical preventive services. As such, we urge you to retain the previously appointed members of the USPSTF and commit to the long-standing process of regular meetings to ensure their important work can continue without interruption.

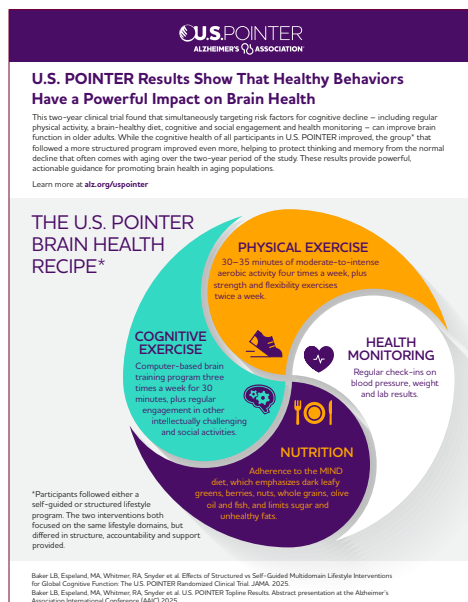
The USPSTF has long played an essential role in making evidence-based recommendations for clinical prevention of disease. USPSTF members have been selected through an open, public nomination process and are nationally recognized experts in primary care, prevention and evidence-based medicine. They serve on a volunteer basis, dedicating their time to help reduce disease and improve the health of all Americans—a mission well-aligned with the Make America Healthy Again initiative.

Importantly, the USPSTF puts forth recommendations that dictate coverage policy for health insurers nationwide. By law, insurers must cover USPSTF-recommended services without cost sharing. This means that patients have access to services such as screenings for colon, breast, and lung cancer; screenings for anxiety and depression in children; and screenings and preventive services for cardiovascular disease. Access to these services without cost sharing plays a critical role in keeping patients healthy and reducing the burdens of disease.

The most important role physicians play is improving the health of patients. Given the essential role USPSTF members play in weighing the benefits and harms of preventive services such as screenings, behavioral counseling, and preventive medications, and making evidence-based recommendations for implementation in primary care settings, we urge you to keep the previously appointed USPSTF members and continue the task force's regular meeting schedule to ensure recommendations are put forth, updated, and disseminated without delay. ❖

U.S. POINTER Alzheimer study results released

PROVIDENCE — Butler Hospital's Memory and Aging Program (MAP) is proud to share the results of the US Study to Protect Brain Health Through Lifestyle Intervention to Reduce Risk (U.S. POINTER). The New England Rhode Island site, based at Butler and Miriam Hospitals, is one of five sites that participated in the Alzheimer's Association study, which was a landmark, two-year clinical trial designed to evaluate whether healthy lifestyle changes can protect memory and other thinking abilities in older adults.



"We are opening a new era in promoting brain health, and it is encouraging that this study proves and gives further evidence that focused lifestyle intervention with exercise, diet, brain training, and heart health can improve memory," said **STEPHEN SALLOWAY, MD**, Principal Investigator, New England RI site of U.S. POINTER, and Founding Director, Memory and Aging Program at Butler Hospital.

Butler Hospital's MAP recruited 376 participants and now has over 250 participants in the U.S. POINTER Alumni extension, which is another four years of collecting more

data on participants and their lifestyles. These participants are all local – within New England – RI, MA, and CT.

The U.S. POINTER study has been the largest participant research trial in Memory and Aging history.

From the Alzheimer's Association International Conference 2025

The U.S. POINTER study shows structured lifestyle program targeting multiple risk factors improves cognition in older adults at risk of cognitive decline.

Key Takeaways

- Two lifestyle interventions in U.S. POINTER improved cognition in older adults at risk of cognitive decline. A structured intervention with more support and accountability showed greater improvement compared to a self-guided intervention.
- In a large, representative group of older adults at high risk for cognitive decline, multidomain lifestyle interventions were delivered with high adherence and safety.
- Cognitive benefits were consistent across age, sex, ethnicity, heart health status, and apolipoprotein E-e4 genotype.

Full Alzheimer's Association Press Release: <https://aaic.alz.org/downloads2025/USPOINTERALZNewsRelease.pdf> ❖

WalletHub finds Rhode Island second-best state for health care

MIAMI, FL — With the average American spending nearly \$14,600 per year on personal health care, the personal-finance website WalletHub recently released its report on 2025's Best & Worst States for Health Care, as well as expert commentary.

In order to determine where Americans receive the highest-quality services at the best prices, WalletHub compared the 50 states and the District of Columbia across 44 key measures of health care cost, accessibility and outcome. The data set ranges from the average monthly insurance premium to physicians per capita to the share of insured population.

Rhode Island

Rhode Island is the second-best state for health care, and its residents have the lowest out-of-pocket medical spending in the country, at just 5.6% of their income. Rhode Island also has a lot of medical professionals, with the sixth-most physicians per capita and sixth-most geriatricians per capita, so it's easier to get seen in a timely manner and get second opinions.

In addition, 94% of adults and 97% of children in Rhode Island have health insurance, the fourth-highest and fifth-highest percentages in the country, respectively.

To top things off, Rhode Island residents clearly have good access to preventative medical care. The Ocean State has the fourth-lowest percentage of people without a routine doctor visit in the past two years and the sixth-lowest share of people who haven't visited a dentist in the past year. The state also has a very high vaccination rate for children, which contributes to the 13th-lowest child death rate in the nation.

Health Care in Rhode Island (1=Best; 25=Avg.)

Overall Rank: **2nd**

11th Avg. Monthly Insurance Premium

19th Hospital Beds per Capita

6th Physicians per Capita

6th Dentists per Capita

4th % of Insured Adults

5th % of Insured Children

4th % of At-Risk Adults with No Routine Doctor Visit in Past Two Years

6th % of Adults with No Dental Visit in Past Year

For the full report, visit: <https://wallethub.com/edu/states-with-best-health-care/23457>

Appointments



Kent Hospital President Paari Gopalakrishnan, MD, MBA, to lead Baltimore Health System

PROVIDENCE — Care New England Health System (CNE) recently announced that **PAARI GOPALAKRISHNAN, MD, MBA**, President and Chief Operating Officer of Kent Hospital, has accepted

the position of President and Chief Executive Officer at GBMC HealthCare (Greater Baltimore Medical Center) in Maryland. He will remain with Kent Hospital through mid-September to assist with transition planning as CNE launches a national search for his successor.

“While we are saddened to see Dr. Gopalakrishnan depart, we are incredibly proud of this well-earned next step in his career,” said **MICHAEL WAGNER, MD, FACP**, President and CEO of Care New England. “Over the past three years, his leadership has propelled Kent Hospital to new heights in clinical excellence, operational performance, and patient-centered care.”

During Dr. Gopalakrishnan’s tenure, Kent Hospital has earned recognition for excellence in key service lines, including Emergency Medicine, Geriatrics, and Orthopedic Surgery. He also spearheaded a major financial turnaround and led the planning of an upcoming renovation of the Emergency Department and Ambulatory Services building, set to begin this fall.

Dr. Gopalakrishnan joined Kent Hospital in 2009 as the Director of the Inpatient Medical Group, was appointed as Chief Medical Officer in 2018, and was appointed President and COO in 2022. In addition to his leadership at Kent, he has played an integral role on the executive team at Care New England, contributing to strategic initiatives across the health system.

“Serving the patients, staff, and community at Kent Hospital has been one of the greatest privileges of my career,” said Dr. Gopalakrishnan. “I’m grateful for the talented and dedicated team I’ve had the honor to work alongside, and I leave confident that Kent’s best days lie ahead.”

Care New England will share updates regarding interim leadership and the national search process in the coming weeks. ❖



Tenny Thomas, MD, FACEP, MHL, named President and Chief Medical Officer of Newport Hospital

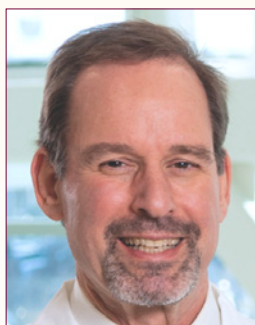
NEWPORT — **TENNY THOMAS, MD, FACEP, MHL**, has been named Newport Hospital’s president and chief medical officer (CMO), which became effective July 14, 2025.

Dr. Thomas joins Newport Hospital from Beth Israel Deaconess Hospital-Plymouth where he had served as chief medical officer since 2018.

“Dr. Thomas is an impressive leader and his passion for advancing healthcare quality and improving patient outcomes will be an asset to Newport Hospital and Brown University Health,” said **SARAH FROST**, Chief of Hospital Operations for Brown University Health. “I look forward to partnering with Dr. Thomas to amplify the award-winning care and services that Newport Hospital proudly provides to our community.”

In his previous roles, Dr. Thomas was instrumental in enhancing operational efficiencies, developing new service lines, and leading successful initiatives in physician wellness and telemedicine services. Dr. Thomas has served as CMO at Good Samaritan Medical Center and chief of the emergency department at Morton Hospital.

Dr. Thomas completed his medical degree at the College of Medicine, Sultan Qaboos University in Oman, and did his residency in Emergency Medicine at George Washington University Hospital. He received a master’s in health care leadership from Brown University. Dr. Thomas is an adjunct faculty member at the School of Public Health at Brown University, is board-certified in Emergency Medicine and is a Fellow of the American College of Emergency Physicians. ❖



Alexander Harmatz, MD, named Chief of the Division of Gastroenterology at Fatima Hospital

NORTH PROVIDENCE — **ALEXANDER HARMATZ, MD**, has been named Chief of the Division of Gastroenterology at Our Lady of Fatima Hospital, it was announced recently by **WILLIAM J. BELIVEAU, MD, FCCP**, Chairman of the Department of Medicine.

Board-certified in gastroenterology and internal medicine, Dr. Harmatz joined CharterCARE in 2018 as a member of the digestive disease center at Roger Williams Medical Center. He has been practicing for more than 35 years and specializes in the areas of hepatobiliary disease, gastroesophageal reflux, pancreatic disease, inflammatory bowel disease, colorectal cancer screening, and nutrition.

Dr. Harmatz is a graduate of the Medical College of Georgia and completed his residency at Sinai Hospital in Baltimore and fellowship at Stanford University in California. He also has a master’s degree in chemistry from Emory University. Prior to joining CharterCARE, he was an assistant professor of medicine at Drexel University College of Medicine. ❖

Appointments



Returning Home: University Orthopedics welcomes native Rhode Islander Nicholas Lemme, MD, to The Sports Medicine Center

EAST PROVIDENCE — University Orthopedics recently announced the addition of Rhode Island native **NICHOLAS LEMME, MD**, to its renowned Sports Medicine Center.

Dr. Lemme is fellowship-trained in both orthopedic sports medicine and orthopedic trauma, and treats

patients ages 10 and up, including recreational and competitive athletes, laborers, weekend warriors, and those seeking surgical revision or second opinions. His practice emphasizes both surgical and non-surgical treatment of musculoskeletal injuries, with a particular focus on the shoulder, knee, and elbow.

“Dr. Lemme’s dual expertise in sports medicine and orthopedic trauma makes him an outstanding addition to our team. His clinical skill, commitment to patient-centered care, and drive for excellent outcomes ensure our patients receive world-class, individualized treatment,” said Dr. Edward Akelman, President of University Orthopedics. “We are proud to welcome a surgeon of his caliber back to Rhode Island and into the UOI family.”

Education & Training

Before completing his orthopedic sports medicine fellowship at the internationally acclaimed Rush University Medical Center in Chicago – widely regarded as one of the top programs in the world – Dr. Lemme earned his medical degree, residency in orthopedic surgery, and a fellowship in orthopedic trauma at The Warren Alpert Medical School of Brown University.

While at Rush, Dr. Lemme served as assistant team physician for the Chicago White Sox (MLB) and the Chicago Bulls (NBA). A proud Rhode Islander, he completed his undergraduate

studies at the University of Rhode Island and is also a graduate of La Salle Academy.

“Returning to Rhode Island to join University Orthopedics is incredibly meaningful to me, both personally and professionally,” said Dr. Lemme. “This community shaped my education, my values, and my passion for orthopedic care. I’m honored to be part of a team so deeply committed to clinical excellence, innovation, and patient-centered treatment – and I’m excited to give back to the place I’ve always called home.”

Clinical Interests

Dr. Lemme’s clinical focus includes:

- Revision ligament reconstruction, particularly in patients with failed ACL or multiligament procedures
- Joint preservation strategies such as osteotomies, cartilage restoration, and meniscal allograft transplantation
- Shoulder arthroplasty for active patients with advanced shoulder pathology

Research & Academic Involvement

Beyond patient care, Dr. Lemme is deeply engaged in academic research and education. He has authored more than 100 peer-reviewed publications, podium and poster presentations, and book chapters. His work spans sports medicine outcomes, joint preservation, biomechanics, and surgical technique innovation.

He currently serves on the Research Committee for the American Orthopaedic Society for Sports Medicine (AOSSM) and is a peer reviewer for both *The American Journal of Sports Medicine* and *Sports Health*. ❖

Appointments

NettieRose Cooley, MBA joins Block Island Health Center as its new CEO

BLOCK ISLAND — **NETTIEROSE COOLEY, MBA**, from Warwick, recently joined the Block Island Health Center (BIHC) as its Chief Operating Officer (CEO). A news release from BIHC stated her 16 years of leadership experience at UnitedHealthcare, most recently acting as the National Senior Director of Value Based Solutions. She also carries over four years of experience in pharmaceutical sales and over 12 years working in the medical practice and hospital sectors.

According to the release, Block Island is special to NettieRose, as she spent summers on the



island as a child. In 2004, she married her husband, Bruce Cooley, at the Spring House and they have been summer residents on their boat, in Old Harbor, for over 20 years. Starting a couple of years ago, with the company of their rescue dog, Rhody, they have enjoyed spending the quiet winter months on the island as well.

"BI has always been considered home in my heart," she said. "It is with great pleasure that I am able to work here and give back to the community that makes the island the special place that it is." ❖

Neighborhood Health Plan welcomes new chair, two board members



SMITHFIELD — Neighborhood Health Plan of Rhode Island (Neighborhood) recently announced **ALISON CROKE, MHA**, CEO of Wood River Health Services, as the new Chair of its Board of Directors and welcomed two community leaders to its Board of Directors.

With 30 years of experience in healthcare, Croke has focused on publicly financed health care programs, including roles in program management, health policy, and strategic planning. Prior to joining Wood River Health in 2018, she served as Vice President of Medicare-Medicaid Integration at Neighborhood.

Croke succeeds Brenda Dowlatsahi, Chief Operating Officer at Tri-County Community Action Agency. "We are grateful to Brenda for her stewardship and unwavering dedication during her tenure as Board Chair," said Peter Marino, President and CEO of Neighborhood. "Her guidance and commitment to our mission have been instrumental in advancing our work to improve the health and well-being of Rhode Islanders. We look to Alison to continue to help and grow Neighborhood during these challenging times," he said.

Additionally, Neighborhood added two new members to its Board of Directors: Allison Brindle, MD, a board-certified pediatrician and Assistant Professor of Pediatrics at The Warren Alpert Medical School at Brown, and Liz Catucci, President and CEO of the Northern Rhode Island Chamber of Commerce. Both will help shape Neighborhood's long-term initiatives that strive to build a holistic culture of care.



ALLISON BRINDLE, MD

With over 20 years of experience practicing pediatrics, Dr. Brindle specializes in general pediatric preventative care and care for the infant through adolescence. She has a special interest in providing a medical home for children with medical complexity. Dr. Brindle is also affiliated with several area hospitals, including Rhode Island Hospital and Hasbro Children's Hospital. She is an Associate Professor of Pediatrics at the Warren Alpert Medical School of Brown University. A member of several professional organizations, including the American Academy of Pediatrics, where she previously served as President of the Rhode Island Chapter, Dr. Brindle received her medical degree from the University of Maryland School of Medicine in Baltimore.



LIZ CATUCCI

Liz Catucci is the President and CEO of the Northern Rhode Island Chamber of Commerce. Catucci is responsible for all the internal operations and advocacy efforts and is charged with carrying out the Chamber's mission. Prior to this role, she served as Director of Marketing and Business Development for PKF O'Connor Davies, a full-service, certified accounting and advisory firm. Ms. Catucci earned her Bachelor of Arts degree from Providence College and earned the honor of Providence Business News' "40 under 40" in 2019. ❖

Appointments



Samuel M. Mencoff named Chair of the Board for Brown University Health

PROVIDENCE — Brown University Health recently announced the appointment of **SAMUEL M. MENCOFF** as Chair of the Board of Directors, effective December

9, 2025. He currently serves as a member of the Brown University Health board.

A Brown University alumnus and seasoned leader in business and philanthropy, Mencoff brings decades of experience in organizational leadership, governance, and health system oversight.

Mencoff, a 1978 graduate of Brown University, former Chancellor of the University (2016–2024) and a longtime member of the Brown Corporation whose service ended on June 30th, is widely recognized for his transformative contributions to higher education, biomedical research, and community health initiatives. His new role at Brown University Health marks a continuation of his longstanding commitment to advancing health equity and excellence in care across Rhode Island and beyond.

“Sam’s leadership and strategic vision will be instrumental as Brown University Health enters a new era of growth, innovation, and service,” said **JOHN FERNANDEZ**, President and CEO of Brown University Health. “His deep ties to Brown and his experience at the intersection of business, healthcare, and education make him uniquely qualified to guide our system well into the future.”

Mencoff is co-founder and former Co-CEO of Madison Dearborn Partners, a leading private equity firm based in Chicago, and he currently serves as a Senior Advisor to the firm. He also holds leadership positions on several national boards, including as a Commissioner of the Smithsonian Institution’s National Portrait Gallery, Vice Chair of the Art Institute of Chicago, Director of the John Carter Brown Library, and is Lead Independent Director of Packaging Corporation of America.

Mencoff succeeds **LAWRENCE A. AUBIN, SR.**, who will be stepping down in December, from the role he has held since 2015. ♦

Recognition

BCBSRI announces nonprofit partners for 2025 day of service – Blue across Rhode Island

PROVIDENCE — Blue Cross & Blue Shield of Rhode Island (BCBSRI) recently announced the nonprofits that will benefit from Blue across Rhode Island 2025, the insurer’s signature day of service during which hundreds of employees spend the workday volunteering at community and social service organizations across the state.

Marking the 14th anniversary of Blue across Rhode Island, this year’s theme is “1 Blue 4 RI.” The event will take place on Friday, Sept. 12 and support 14 nonprofits that applied for help with their efforts to improve Rhode Islanders’ quality of life. Blue across Rhode Island is one of the largest annual volunteer events of its kind in the state.

Participating nonprofits this year were revealed at a spirited kickoff for Blue across Rhode Island. Team leaders presented details of the 2025 volunteer opportunities to their colleagues, who are then encouraged to sign up for the project of their choice.

“Our associates look forward to the kickoff every year to learn how they can roll up their sleeves to help improve the health and well-being of their neighbors,” said BCBSRI Vice President of Corporate Social Responsibility Carolyn Belisle. “This year’s theme ‘1 Blue 4 RI,’ represents our 14th year of showing up for our community-based partners, who work tirelessly year-round to better the lives of Rhode Islanders.”

BCBSRI volunteers will support a variety of community-based organizations and their projects, including ones that address food, housing, dental care, mental wellness, and community recreation.

The following are the 14 participating organizations that BCBSRI employees will support:

- Amenity Aid
- Crossroads Rhode Island
- Dorcas International
- Feed RI
- Gotta Have Sole Foundation, Inc.
- Higher Ground International
- Hope Alzheimer’s Center
- NeighborWorks Blackstone River Valley
- Playworks New England
- Rhode Island Oral Health Foundation
- Sleep in Heavenly Peace
- The ELISHA Project
- The Village Common of RI
- YMCA of Greater Providence

Since the launch of Blue across Rhode Island in 2012, BCBSRI has supported 80 organizations across the state on more than 140 projects and associates have logged more than 40,000 volunteer hours. These projects have had an impact on the lives of more than 205,000 Rhode Islanders. ♦

Recognition

2025 Worksite Health Awards honor 48 Rhode Island-based companies

WARWICK — Blue Cross & Blue Shield of Rhode Island (BCBSRI) and the Greater Providence Chamber of Commerce presented the 31st annual Worksite Health Awards to 48 companies at a celebration at the Crowne Plaza July 17th.

The Worksite Health Awards honor Rhode Island-based employers that demonstrate sincere commitment to maintaining a healthy workplace through policy, culture, and educational opportunities. With some companies having locations outside the state and with workforces that are hybrid or virtual, the awards help showcase employer commitment in Rhode Island and beyond, as well as best practices for improving the health and well-being of employees.

This year's awards included a special category – Inspiring Voices – that required submission of essays recounting notable workplace wellness initiatives. Winning essays included:

- **BankNewport:** On the transformative power of its Vitality Wellness Program and featuring two employees who improved chronic health issues through exercise and nutrition.
- **Highbar Physical Therapy:** On its Special Interest Groups, which foster learning, innovation and connection, and a company challenge, September of Move, that inspired employees and their families to get more active.
- **Raytheon Company–Naval Power:** On how company employees rallied to improve their readiness to respond to heart attacks, strokes and seizures following a medical event on the company's production floor in which employees rushed to provide aid to a colleague.

"At Blue Cross, we're proud to recognize – and celebrate – local businesses that are dedicated to the health and well-being



Blue Cross & Blue Shield of Rhode Island (BCBSRI) and the Greater Providence Chamber of Commerce at celebration held at the Crowne Plaza July 17th. [PROVIDENCE CHAMBER OF COMMERCE]

of their employees," said **JOHN DONOHUE**, BCBSRI Vice President of Commercial Markets. "With so many companies headquartered in Rhode Island, their best practices are a model for businesses within our state and across the nation. It was inspiring to see the creative, innovative ways that employers are helping their employees improve their physical, mental, and social well-being."

LAURIE WHITE, President of the Greater Providence Chamber of Commerce, said, "Now in the 31st year of the Worksite Health Awards, we remain inspired by the creativity and commitment of those Rhode Island companies that go above and beyond to make employee wellness a priority. By supporting healthy lifestyles and boosting morale, worksite wellness programs are a win-win for everyone. We are once again proud to partner with Blue Cross & Blue Shield of Rhode Island to recognize local businesses finding innovative and engaging ways to help their employees live better, healthier and happier lives."

Businesses were named 2025 Worksite Health Award honorees in these three categories:

Exemplary honorees

American Mathematical Society, BankNewport, Blue Cross & Blue Shield of Rhode Island, Brown University, Brown University Health, Bryant University, Care New England Health Services, Coastal Credit Union, Comprehensive Community Action Program, Cooley Group, Dominion Diagnostics, LLC., Gilbane,

Inc., Greenwood Credit Union, Highbar Physical Therapy, Hinckley Allen, Meeting Street, Navigant Credit Union, Ocean State Job Lot, Ortho Rhode Island, Providence College, Raytheon–Naval Power, Rhode Island Zoological Society, RI Housing, RIPIN (Rhode Island Parent Information Network), Roger Williams University Simply Wellness, South County Health, State of Rhode Island, Swarovski Optik North America, LTD, The Beacon Mutual Insurance Company, The Washington Trust Company, Toray Plastics (America), Inc., Westbay Community Action Inc.

Superior honorees

AIPSO, Brookwood Finishing, CME Corp., Falvey Insurance Group, Living in Fulfilling Environments (LIFE), Inc, Paul Masse Automotive Group, Rhode Island Commerce, Rhode Interlocal Risk Management Trust, Saint Elizabeth Community, Taco Comfort Solutions, Wood River Health.

Outstanding honorees

Blackstone Valley Community Health Center, Crossroads RI, Plumbers & Pipefitters Local 51, The RISE Group, University Orthopedics.

All Rhode Island-headquartered businesses are eligible to apply for the Worksite Health Awards. BCBSRI, in partnership with the Chamber, is a proud presenting sponsor of the awards, which have recognized local businesses for creating healthy workplaces since 1995. ❖

Recognition

Rhode Island, Miriam, Newport hospitals recognized for exceptional stroke treatment

PROVIDENCE/NEWPORT — Three Brown University Health hospitals have been nationally recognized again for providing exceptional patient care and outcomes by The American Heart Association (AHA).

Rhode Island Hospital, The Miriam Hospital, and Newport Hospital received the prestigious Get With The Guidelines®-Stroke GOLD PLUS Quality Achievement Award. The award recognizes programs that demonstrate commitment to ensuring stroke patients receive the most appropriate treatment according to nationally recognized, research-based guidelines, ultimately leading to more lives saved and reduced disability.

All three hospitals also received AHA's Target: Type 2 Diabetes Honor Roll designation in recognition for demonstrating they met quality measures with over 90% compliance for the prior year.

Additionally, the hospitals were honored for:

- **Rhode Island Hospital** received the AHA's Target: Stroke Honor Roll Advanced Therapy and Target: Stroke Honor Roll Elite Plus designations, in addition to Advanced Therapy and AHA's Target: Type 2 Diabetes Honor Roll.
- **The Miriam Hospital** received AHA's Target: Stroke Honor Roll Elite Plus as well as the AHA's Target: Type 2 Diabetes Honor Roll designations.
- **Newport Hospital** received AHA's Target: Stroke Honor Roll designation and AHA's Target: Type 2 Diabetes Honor Roll. ❖

South County Health earns CMS infection prevention recognition

WAKEFIELD — South County Health has been recognized as one of only 514 hospitals across the United States to achieve zero catheter-associated urinary tract infections (CAUTIs), based on the most recent Centers for Medicare & Medicaid Services (CMS) data.

The data, compiled in the Healthcare Associated Infections-Hospital database and collected through the CDC's National Healthcare Safety Network, reflects a full year of reporting – from July 2023 through June 2024. These results underscore South County Health's dedication to infection prevention and the delivery of exceptional care across all settings.

CMS and the CDC calculate standardized infection ratios using a range of factors including hospital size, patient health status, and care location. The measures apply to all patients in acute care hospitals, whether adult, pediatric, neonatal, Medicare or non-Medicare. ❖

Hasbro Children's announces 2024 'Brite Lites' hospital employees recognized for exemplary care

PROVIDENCE — Hasbro Children's announced its 2024 Brite Lite award winners at a recent ceremony held in the hospital's Balise Healing Garden. Recipients are Brown University Health employees nominated by Hasbro Children's patients and their families for their dedication and compassion shown to pediatric patients under their care.



The Brite Lite employee recognition program started 20-plus years ago. It celebrates five employees who have excelled in their everyday duties to make patients and families feel safe, important, and well cared for. Brite Lite winners are chosen for exemplifying “the four Cs” at Hasbro Children's: caring, communication, cooperation and competence.

The 2024 Brite Lites winners, selected from among more than 40 nominations, are:

- **LAURA CIOE**, family advocate, patient & family centered care, Hasbro Children's
- **KRISTEN GALLAGHER, RN**, 5th floor, Hasbro Children's
- **MARGARET SCHEFFLER, MD**, pediatric critical care, Hasbro Children's
- **MARY SETTE, NP**, Emergency Department, Hasbro Children's
- **MADDISON ZUBA, BSN, RN**, pediatric float pool, Hasbro Children's ❖

Recognition

Roger Williams celebrates 30 years of BMT services

PROVIDENCE — Close to 100 cancer survivors, family members and friends, providers and hospital staff gathered recently at Roger Williams Medical Center (RWMC) to celebrate National Cancer Survivor Month, their cancer care journey, and mark the 30th anniversary of the Blood and Marrow Transplant and Cellular Therapy Program (BMT).



Sen. **Jack Reed** joined with cancer survivors, family, and staff on Saturday, June 21 in Kay Auditorium. [RWMC]

The program was highlighted by remarks from Sen. **JACK REED**, where it was a bit of a homecoming given he was on hand in 1994 to help open the BMT unit at Roger Williams.

"You know 'Hope' is the motto of our state, and for over 30 years, BMT has been bringing hope to cancer patients throughout our state," he said. "Over the last decade, we have seen incredible advancements in cancer research. Survivorship rates are improving and quality of life improving. That's why we can't let our foot off the pedal. We need to keep investing in biomedical research and clinical trials. We need to redouble our efforts, and I can think of no better place to look for inspiration than the many individuals and families here who have fought for their lives. You are all battle tested...and we need to match that strength."

Remarks were also delivered by **DR. RITESH RATHORE**, Medical Director of the Cancer Center, and **DR. TODD ROBERTS**, Director of the BMT program. "Most people know Senator Reed as a leader on the Senate Appropriations and Armed Services Committee," commented Dr. Rathore, adding, "but not everyone is familiar with his leadership in authoring major bills on healthcare and his work in supporting health care access, especially in RI. We would like to recognize him as a true champion for healthcare in RI."

He went on to say, "Today, we want to thank all our patients for trusting us to take care of them in the most difficult and trying phase of their lives. We will strive continuously to provide the best care and support and partner with you during your therapy and your recovery."

The BMT Program at Roger Williams is the only full-service program of its kind in Southern New England. ❖

Roger Williams Sleep Center reaccredited

PROVIDENCE — The Roger Williams Medical Center Sleep Disorders Center has been officially awarded reaccreditation for a period of five years by the American Academy of Sleep Medicine (AASM).

The Sleep Center provides sleep testing as well as out-patient consultations for patients with sleep related complaints such as snoring, daytime fatigue, fragmented sleep, insomnia and sleep apnea. Sleep tests include overnight attended in-center polysomnography, home sleep apnea testing and multiple sleep latency testing. A team of highly experienced registered polysomnography technologists perform the testing and analyze the data. By successfully completing the re-accreditation process and upholding the standards of accreditation, the RWMC Sleep Disorders Center has shown its dedication to the advancement of the field.



Members of the Sleep Center: **Eymy Soto** (Medial Assistant), **Tracy Banaczuk** (Sleep Scorer), **Patrice Lawrence** (Medical Assistant), **Debra Forsythe** (Medical Secretary), **Erica Brousseau** (Nurse Practitioner, APRN), and **Zulfikar Alli** (Manager). [RWMC]

Since 1977, the American Academy of Sleep Medicine (AASM) Standards for Accreditation have been the gold standard by which the medical community and the public evaluate sleep medicine facilities. Achieving AASM accreditation demonstrates a sleep medicine provider's commitment to high quality, patient-centered care through adherence to these standards. The RWMC Sleep Center has been continuously accredited since 2009.

"This is another example of our commitment to provide specialty services that follow industry best practices," said **JEFFREY LIEBMAN**, CharterCARE CEO, parent company of Roger Williams Medical Center. "This is validation once again of the exceptional clinical leadership and patient care this sleep program has provided for more than 10 years." **DR. F. DENNIS MCCOOL**, ABIM, board-certified in Sleep Medicine, is Medical Director of the Sleep Disorder Center. ❖

Recognition

Two URI nursing professors to be inducted as Fellows in American Academy of Nursing

KINGSTON (URI) — Two University of Rhode Island College of Nursing professors, Associate Professor **AMY D'AGATA, PhD, RN**, and Assistant Professor **ERICA LIEBERMANN, PhD, MSN**, have been selected to receive the highest recognition of accomplishment in the nursing profession, induction as Fellows in the American Academy of Nursing.

Invitation to fellowship requires nurses to demonstrate sustained exceptional contributions throughout their careers, and recognizes a nursing leader's accomplishments in the profession.

Dr. Liebermann is an adult/women's health nurse practitioner and clinician scientist with extensive international experience. Her clinical work and health services research are driven by a desire to advance the health of women and girls throughout the



College of Nursing professors Amy D'Agata (left) and Erica Liebermann (right) are the latest URI faculty members to be inducted as Fellows in the American Academy of Nursing.

world, through promoting equitable access to high quality health care. Her current research focuses on advancing progress toward cervical cancer elimination in the U.S. through innovative strategies to increase HPV vaccination rates and improve cervical cancer screening and follow-up.

Dr. D'Agata has extensive clinical experience in neonatal intensive care. Her research has evolved to examining the lifetime effects of preterm birth. She serves as the principal investigator for the RHODE Study, the longest continuously running preterm birth study in the United States.

Funded by the National Institutes of Health through an R01 grant, the study explores the impact of preterm birth on adult cardiometabolic, immune, and endocrine health, as well as epigenetic aging at the age of 35. ♦

Obituaries



DAVID LOUIS KITZES, MD, age 87, of Providence, passed away peacefully on June 29, 2025 at his residence, surrounded by loved ones. He now joins his beloved sweetheart, the late Mary Ellen (Richey) Kitzes, in another great and mysterious adventure.

David's family wishes to memorialize David's commitment to love, compassion, healing and service. Donations may be made in honor of Dr. David Kitzes to Jewish Collaborative Services' Community Services and Food Security Fund (visit: [JCSRI.org](https://www.jcsri.org)). This fund supports programs that assist caregivers, families, older adults, and underserved populations with emergency financial assistance, food access, and social services.

Full obituary to follow soon at www.sugarmansinai.com ❖



DENNIS S. KRAUSS, MD, age 77, of Walnut Creek, CA, passed away on July 1, 2025, surrounded by loved ones. He was a former Barrington resident and physician in Providence.

Born in New York, New York, on May 7, 1948, Dennis grew up in Brighton, MA. He knew from an early age that he wanted to be

a doctor, like his father. After graduating from Harvard College, he attended medical school at The University of Vermont.

In 1979, he moved to Barrington with his wife, Paula, and their children. He joined a private endocrinology practice in Providence, focusing on the treatment of diabetes. He became known in the community as a caring and thorough physician.

Dennis owned a vacation home on Lake Winnepesaukee in Moultonborough, NH, where he loved spending quality time with his family and friends. At his lake home, and especially on his motorboat, he experienced immense joy and gratefulness.

In 2017, following his retirement, he and Paula moved to California to be closer to children and grandchildren. There, he fought valiantly against Parkinson's disease while living an active lifestyle in a retirement community called Rossmoor.

Dennis leaves behind his wife, Paula; his children, Jeff Krauss, Greg Krauss, and Emily (Krauss) Grossberg; his grandchildren, Lila Krauss, Eva Krauss, Ella Grossberg, and Jake Grossberg; his son-in law, Eric Grossberg; his daughter-in-law, Carrie Sullivan Krauss; and his siblings, Sandra Webber, Beverly Hugo, and Robert Krauss. He was preceded in death by his brother, Malcolm Krauss.

In lieu of flowers or gifts, please send donations to the Parkinson Network of Mt. Diablo by going to: <https://www.pnmd.net/donating> ❖