Paroxysmal Sympathetic Hyperactivity Syndrome Following Traumatic Brain Injury

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KEYWORDS

• Paroxysmal sympathetic hyperactivity (PSH) • Traumatic brain injury (TBI)
• Sympathetic storming • Autonomic dysfunction

KEY POINTS

- Paroxysmal sympathetic hyperactivity (PSH) is a syndrome defined by the presence of abnormal sympathetic nervous system or motor activity in response to non-nociceptive stimuli, most often observed in the context of severe traumatic brain injuries.
- Although the exact cause of PSH is unknown, a failure to identify and treat this syndrome can result in negative outcomes, such as prolonged hospitalization and/or worse Glasgow Coma Scale score.
- The consensus-based development of the Paroxysmal Sympathetic Hyperactivity Assessment Measure (PSH-AM) tool was a landmark advancement in the assessment and diagnosis of PSH.
- Current treatment approaches focus on pharmacologic management and prevention of specific sympathetic symptoms.
- Future research goals include implementation and validation of the PSH-AM tool, further development of treatment guidelines, and continued research in imaging and patient monitoring to advance theories of pathophysiology.

INTRODUCTION

Although the syndrome of paroxysmal sympathetic hyperactivity (PSH) has been observed for decades in patients with acquired brain injuries, only in recent years has the identification and definition of this condition been simplified in the literature.[1] PSH is a syndrome of episodic sympathetic and/or motor hyperactivity that has paroxysmal occurrence in patients who have an acquired brain injury due to stroke, head trauma, anoxic injury, or other condition, such as hydrocephalus.[2] Although PSH has been observed in the context of conditions like anoxic injury, stroke, hydrocephalus, and infection, it is most frequently observed in the clinical context of traumatic brain injury due to head trauma sustained through a variety of mechanisms including motor vehicle

Disclosures: None.
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Nurs Clin N Am 53 (2018) 459–467
https://doi.org/10.1016/j.cnur.2018.05.003
nursing.theclinics.com
0029-6465/18/

collisions or blunt trauma sustained in an assault or during a fall event. Some of the observed symptoms include tachycardia, fever, increased respiratory rate, diaphoresis, or dystonic posturing (Table 1).[1] One factor that has likely contributed to the confusion surrounding this condition involves the nomenclature attributed to this syndrome. In a 2014 search of the evidence, Baguley and colleagues[1] identified more than thirty different clinical terms used to describe the condition and subsequently aimed to standardize the nomenclature associated with this syndrome by narrowing the synonymous terms to *paroxysmal sympathetic hyperactivity*. With a singularly recognized identifier for this syndrome, both existing and future research may be more easily shared, and appropriate treatment more quickly implemented.

PSH after a sustained traumatic brain injury (TBI) has become a priority in clinical research, as case evidence has demonstrated the negative consequences resulting from undiagnosed or untreated cases of PSH.[1] Evidence on the long-term neurologic outcomes of patients with TBIs with PSH based on measures like the Glasgow Outcome Scale has been conflicting; however, this may be due to a failure of outcome measures to capture deficits with adequate sensitivity.[2,3] Future research pursuits may evaluate long-term neurologic outcomes for these patients in order to better define the extent of the impact of PSH. Although impressions gleaned from existing research may be distorted due to inherent study limitations like small sample size, the evidence has consistently indicated that the presence of PSH acts as a risk factor for worse neurologic outcomes in those who have sustained a TBI.[1–7] While the condition has been noted in pediatric patients with brain injuries, this work focuses on the presentation of PSH in the adult population.

INCIDENCE AND CLINICAL SIGNIFICANCE

PSH has also been identified in patients with a variety of neurologic abnormalities, including hydrocephalus and stroke; however, it is most commonly noted in patients who have TBIs.[2] The incidence of PSH in patients with TBIs has ranged from 8% to 33% in existing literature.[3,6,8] The true incidence may be difficult to gauge, however, because of the historical lack of a standardized set of diagnostic criteria for PSH and thus the absence of a standard diagnostic process in the routine management of these patients.

Table 1
Core symptoms of paroxysmal sympathetic hyperactivity by system

System	Abnormal Criteria
General	
Diaphoresis	Presence of mild to severe sweating
Hyperthermia	Body temperature ≥37.0°C
Cardiovascular	
Tachycardia	HR ≥100 beats per minute
Hypertension	Systolic blood pressure ≥140
Respiratory	
Tachypnea	Respiratory rate ≥18 breaths per minute
Musculoskeletal	
Posturing	Presence of mild to severe dystonic posturing of extremities

Abbreviation: HR, heart rate.

Data from Baguley IJ, Perkes IE, Fernandez-Ortega JF, et al. Paroxysmal sympathetic hyperactivity after acquired brain injury: consensus on conceptual definition, nomenclature, and diagnostic criteria. J Neurotrauma 2014;31(17):1515–20.