

Understanding paroxysmal sympathetic hyperactivity after traumatic brain injury

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Abstract

Background: Paroxysmal sympathetic hyperactivity (PSH) is a condition occurring in a small percentage of patients with severe traumatic brain injury (TBI). It is characterized by a constellation of symptoms associated with excessive adrenergic output, including tachycardia, hypertension, tachypnea, and diaphoresis. Diagnosis is one of exclusion and, therefore, is often delayed. Treatment is aimed at minimizing triggers and pharmacologic management of symptoms.

Methods: A literature review using medline and cinahl was conducted to identify articles related to PSH. Search terms included paroxysmal sympathetic hyperactivity, autonomic storming, diencephalic seizures, and sympathetic storming. Reference lists of pertinent articles were also reviewed and these additional papers were included.

Results: The literature indicates that the understanding of PSH following TBI is in its infancy. The majority of information is based on small case series. The review revealed treatments that may be useful in treating PSH.

Conclusions: Nurses play a critical role in the identification of at-risk patients, symptom complexes, and in the education of family. Early detection and treatment is likely to decrease overall morbidity and facilitate recovery. Further research is needed to establish screening tools and treatment algorithms for PSH.

Key Words: Paroxysmal sympathetic hyperactivity, sympathetic storming, traumatic brain injury

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INTRODUCTION

Traumatic brain injury (TBI) affects 1.4 million Americans annually.^[1] Although only a small percentage of TBI is categorized as severe or comatose state, this subgroup of patients is at risk for prolonged hospital stay, persistent disability, and increased lifetime healthcare utilization.^[1] Consequently, survivors of severe TBI incur approximately 90% of all TBI-related costs.^[1] Paroxysmal

sympathetic hyperactivity (PSH) has been reported in up to 33% of patients with severe TBI.^[15]

PSH has been discussed in the literature under a variety of names. Early in the recognition of this phenomenon, the term diencephalic seizure was used.^[19] However, later investigations failed to demonstrate epileptiform discharges on electroencephalography, therefore, this term is no longer used.^[9,16] PSH later came to be known as dysautonomia, autonomic storming,

sympathetic storming, and autonomic dysreflexia or hyper-reflexia.^[1] The International Brain Injury Association has recently convened a consensus workgroup to clarify the nomenclature and diagnostic criteria for this entity. The proposed term from this consensus group is paroxysmal sympathetic hyperactivity.^[8] To date, the vast majority of reports pertaining to PSH is found in the medical literature, with attention paid to hypothesis of cause and pharmacologic management. With a few exceptions, studies are limited to retrospective reviews, case studies, or small case series, thereby limiting their generalizability.

PSH is a poorly understood phenomenon with varied symptomatology. As a result, identification of the condition is often delayed.^[14] Diagnosis is often one of exclusion, where other conditions with similar symptoms, such as infection or withdrawal, are ruled out. Brief, but frequent episodes of hemodynamic instability and physiologic distress are the hallmarks of this condition.^[8,16] Imaging studies in patients experiencing sympathetic storming often reveal evidence of diffuse axonal injury.^[6,9] Coma emergence and weaning from high-dose opiates and sedatives are the precursors that are frequently associated with sign or symptom exhibition. Baguley *et al.* suggest that PSH is present if the following symptoms occur without other identifiable causes: Hyperthermia, tachycardia, tachypnea, dystonia, hypertension, posturing, and diaphoresis.^[3, 16] Identification of these signs or symptoms often results in additional diagnostic work-up and increased medication requirements.

PATHOPHYSIOLOGY

PSH is thought to occur in stages. The first stage is often asymptomatic due to the heavy sedation and paralytics required for the acute management of intracranial hypertension and other trauma-related injuries. The second stage is characterized by the onset of symptom clusters and the third stage, by a decline in posturing and dystonia.^[3] Baguley *et al.* demonstrated that changes in heart rate variability associated with nociceptive stimuli accompany the onset of sympathetic storming.^[5,7] Retrospective reviews indicate that those with diffuse axonal injury on imaging studies are at highest risk for developing sympathetic storming.^[3,14] Symptoms are thought to persist for months to years, with one study revealing a mean duration of 5 years post-injury.^[5]

Various dissociation theories provide the theoretical framework for PSH. These include those of structural disconnection, where lesions in the mesencephalon cause disruptions in relay from the medulla/hypothalamus, and the more widely accepted excitatory: Inhibitory ratio (EIR) model, where dysfunction of the diencephalic–brainstem inhibitory center that normally

controls afferent stimulus processing in the spinal cord occurs.^[2,13,16] Autonomic dysfunction is thought to result from functional disconnections related to traumatic damage involving the deep structures of the brain.^[2] This is complicated by a relative reduction in functional dopaminergic activity.^[2] As a result, there is unopposed adrenergic outflow with increased levels of circulating catecholamines.^[2] Within this framework, there is an increase in the excitatory influences without a compensatory increase in inhibitory function, thus creating a tendency to develop an exaggerated response to normally benign stimuli.^[5,7] Multisystem organ dysfunction can follow if the condition is untreated.

TREATMENT

There is no accepted treatment algorithm for the management of PSH. Treatment is aimed at mitigating signs and symptoms to decrease associated adverse events such as cardiac hypertrophy, dehydration, muscle wasting, contractures, and delayed recovery which contribute to increased morbidity [Table 1]. Multiple medications are often required to successfully control the multiple symptoms. Dopaminergic agents, specifically bromocriptine, have been shown to decrease body temperature and sweating.^[10,20] Alpha-agonists such as clonidine act to decrease heart rate and blood pressure.^[18,21] Gamma-aminobutyric acid (GABA) agonists are also useful in some cases. Baclofen can be given enterally or intrathecally. Intrathecal baclofen significantly reduces symptoms at substantially lower doses.^[12] Gabapentin is thought to act on the dorsal horn of the spinal cord and in one small study, reduced the need for concomitant medications.^[4] Opiates and non-selective beta-blockers have also been used.^[6,10]

Table 1: Multisystem dysfunction associated with excessive circulating catecholamines

Organ system	Sign/symptom
Cardiovascular	Tachycardia Increased contractility Increased cardiac output Hypertension
Pulmonary	Tachypnea Bronchial dilation Pulmonary edema
Eyes	Pupillary dilation
Gastrointestinal	Decreased GI motility Increased tube feed residual Ileus
Musculoskeletal	Dystonia Posturing Contractures Spasticity
Adrenal	Increased release of epinephrine Increased release of norepinephrine

In cases of medication failure, a case series of six demonstrated that hyperbaric oxygen (HBOT) controlled autonomic discharges and posturing in the subacute TBI phase.^[17] However, this treatment modality is of limited utility as there are an inadequate number of facilities with HBOT capabilities. It is challenging to conduct large experimental studies to validate the findings of these smaller studies due to difficulties in early identification of PSH and the relatively small number of qualifying patients.

IMPLICATIONS

Early identification and treatment of PSH is critical to facilitate recovery from TBI and avoid permanent organ dysfunction. Expensive diagnostic testing is rarely warranted. Instead, the observational and analytical skills employed by most ICU nurses are more useful in recognizing PSH. In addition to communication of findings in order to facilitate diagnosis and treatment, nursing surveillance also affords the opportunity to identify and, therefore, potentially mitigate triggers. A final contribution of nursing involves family and caregiver teaching. In this role, the nurse explains the concept of PSH, along with its causes and treatments, thereby aiding the family member to better cope with the paroxysms that can be very frightening or chaotic to watch.

Early initiation of symptom-specific therapy, although beyond the scope of this paper, is thought to decrease complication rates and ICU length of stay and to facilitate recovery.^[15] Understanding both the cause and complexity of these storming events will lead to improved therapeutic interventions.

CONCLUSION

PSH should be considered when patients emerging from coma exhibit multiple, concurrent symptoms of sympathetic overactivity: Hyperthermia, posturing, dystonia, tachycardia, tachypnea, diaphoresis, or hypertension. Nurses play a pivotal role in identifying the condition of PSH, observing and mitigating the triggers which precipitate the event, communicating patient needs to the interdisciplinary team, and finally, educating the family/caregivers. Ongoing research should include efforts to develop scales to document the frequency and severity of these paroxysmal episodes and treatment algorithms to guide the clinician in the management of PSH.

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