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Paroxysmal Sympathetic Hyperactivity: A Structured Approach for Clinical Practice

Síndrome de Hiperatividade Simpática Paroxística: Uma Abordagem Sistematizada para a Prática Clínica

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ABSTRACT

Paroxysmal sympathetic hyperactivity is a neurological syndrome characterized by sudden episodes of sympathetic overactivity often triggered by non-noxious stimuli. First described by Wilder Penfield in 1929, it commonly follows severe brain injury and is associated with diffuse lesions involving diencephalic and brainstem structures. Its pathophysiology is not completely understood, but it is believed to result from an imbalance between excitatory and inhibitory pathways within the central nervous system. Diagnosis is clinical, based on exclusion of alternative causes and supported by the Paroxysmal Sympathetic Hyperactivity Assessment Measure. Management includes both pharmacological and non-pharmacological strategies. Despite growing recognition, clinical approaches remain heterogeneous. Further research is needed to clarify mechanisms and develop standardized, evidence-based diagnostic and treatment guidelines to improve outcomes.

Keywords: Autonomic Nervous System Diseases/diagnosis; Autonomic Nervous System Diseases/epidemiology; Autonomic Nervous System Diseases/etiology; Autonomic Nervous System Diseases/therapy; Brain Injuries, Traumatic/complications; Sympathetic Nervous System/physiopathology

RESUMO

A hiperatividade simpática paroxística é uma síndrome neurológica caracterizada por episódios súbitos de hiperatividade simpática geralmente desencadeados por estímulos não nocivos. Descrita pela primeira vez por Wilder Penfield em 1929, surge habitualmente após lesões cerebrais graves, associadas a atingimento difuso do diencéfalo e tronco cerebral. A fisiopatologia ainda não está completamente esclarecida, sendo atribuída a um desequilíbrio entre as vias excitatórias e inibitórias do sistema nervoso central. O diagnóstico é clínico, baseado na exclusão de outras causas, podendo ser utilizada a *Paroxysmal Sympathetic Hyperactivity Assessment Measure*. O tratamento combina terapêutica farmacológica com medidas não farmacológicas. Apesar do reconhecimento crescente, a prática clínica mantém-se heterogénea. São necessários estudos adicionais que permitam aprofundar os mecanismos envolvidos e estabelecer diretrizes padronizadas, baseadas na evidência, para melhorar os resultados clínicos.

Palavras-chave: Doenças do Sistema Nervoso Autónomo/diagnóstico; Doenças do Sistema Nervoso Autónomo/epidemiologia; Doenças do Sistema Nervoso Autónomo/etiologia; Doenças do Sistema Nervoso Autónomo/tratamento; Sistema Nervoso Simpático/fisiopatologia; Traumatismo Crânio-Encefálico

INTRODUCTION

Paroxysmal sympathetic hyperactivity (PSH) is a state of sympathetic hyperactivity that can persist for weeks or months, characterized by intermittent episodes of increased heart rate and blood pressure, sweating, hyperthermia and motor posturing, often triggered by external stimuli.[1-3] These episodes have a rapid onset and slow resolution unless interrupted by appropriate medication.[2] If not treated, PSH may progress over time and potentially cause serious secondary complications.[2]

It is a complication of various acute brain disorders, and while relatively common, it often goes unrecognized, resulting from disruptions in the central regulation of autonomic function.[2-5]

This work aims to consolidate and update current knowledge on PSH. Alongside the literature review, a practical clinical algorithm has been created to assist in assessing and managing patients suspected of having PSH. This effort not only raises awareness and understanding of PSH but also aids in developing future clinical guidelines for this frequently under-recognized condition.

METHODS

A literature search was performed in the PubMed database, limited to the period from January 2015 to March 2025, to include studies based on the standardized definition and diagnostic criteria for PSH established by the 2014 international consensus. This time restriction ensured that the review reflected recent and clinically relevant evidence, incorporating contemporary diagnostic and therapeutic approaches. The search terms used were “paroxysmal sympathetic hyperactivity”, “autonomic storm”, and “autonomic dysregulation”.

Eligible publications included original articles, reviews, and case reports that addressed clinical, diagnostic, or therapeutic aspects of PSH in adults. Articles published in English were preferred to ensure analytical consistency and

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comparability across studies. Pediatric studies and animal research were excluded. The initial search retrieved 59 records. After screening titles, 23 articles were considered potentially relevant. Following abstract review, 11 were excluded for lack of relevance. A total of 14 studies met the inclusion criteria and were included in the final analysis (Fig. 1). Alongside the literature review, a practical clinical algorithm was developed to help assess and manage patients suspected of having PSH.

Definition

Wilder Penfield was the first to describe the clinical features of PSH following traumatic brain injury (TBI) in 1929, suggesting it likely had an epileptic origin and naming the syndrome as 'mesencephalic seizure' or 'diencephalic autonomic seizure'.[6]

Over time, this condition has been referred to by various names, including 'autonomic storms', 'sympathetic storms', 'hypothalamic dysregulation syndrome', 'dysautonomia', 'paroxysmal autonomic instability with dystonia', and, in its initial description, 'diencephalic autonomic epilepsy'.[2] More than thirty clinical terms have been used to describe this condition.[3] The term 'paroxysmal sympathetic hyperactivity' was first proposed in 2010 as a unifying designation.[2,4]

In 2014, a steering committee comprising specialists in critical care medicine, neurology, neurosurgery, nursing, occupational therapy and rehabilitation medicine from Australia, Europe and the United States convened to establish a conceptual definition and develop consensus-based diagnostic criteria. As a result, the term 'paroxysmal sympathetic hyperactivity' was officially adopted and defined as a syndrome occurring in a subset of survivors of severe acquired brain injury, characterized by paroxysmal increases in sympathetic and motor activity.[1,3,7]

Epidemiology

Paroxysmal sympathetic hyperactivity is most commonly linked to TBI, representing about 80% of cases.[1] It has also been observed in conditions such as global brain anoxia, autoimmune encephalitis, intracranial hemorrhage, cerebral fat embolism, and central nervous system infections.[2,3,5]

The incidence of PSH among TBI patients varies significantly, ranging from 8% to 33%. This discrepancy indicates issues with diagnostic criteria, admission protocols, and limited awareness of PSH recognition.[1-4]

In the past, long-term clinical observations have revealed that PSH is more prevalent in men than in women.[4] However, this observation may reflect the higher prevalence of TBI in male patients rather than a true gender predisposition.[3]

Paroxysmal sympathetic hyperactivity has been linked to extended tracheostomy weaning in severe TBI cases, and some evidence suggests that the presence of a tracheostomy may be associated with an increased occurrence of PSH.[4] Other risk factors linked to the development of PSH include age, early fever onset, the extent of diffuse axonal injury and lower Glasgow Coma Scale (GCS) scores.[8]

Pathophysiology

The pathophysiology of PSH remains partially understood. There are several theories, but existing models suggest that it results from the disruption of cortical inhibitory centers, such as the insula and cingulate cortex, along with sympathetic control centers located in the hypothalamus, diencephalon, brainstem and spinal cord.[1,2,8,9] This theory, known as the excitatory/inhibitory ratio (EIR) model, unfolds in two phases: the initial disruption of inhibitory pathways causes spinal overexcitation, followed by a recovery phase as these inhibitory mechanisms resume function.[1-3,10]

Typically, various cortical and subcortical inputs regulate activity in brainstem centers. These brainstem nuclei exert an inhibitory effect on spinal reflex arcs, which helps to maintain a balance between the roles of inhibitory and excitatory interneurons on motor and sympathetic outputs, allowing normal stimuli to be perceived as non-noxious.[1]

When descending inhibitory modulation is disrupted, maladaptive changes occur within the spinal cord, resulting in increased excitatory interneuronal activity.[1,2] These changes may clarify how non-painful stimuli can trigger exaggerated spinal cord responses that the cortex interprets as painful (e.g., allodynic hyper-responsiveness).[2]

The EIR model elucidates why individuals with less brainstem involvement experience shorter paroxysm duration and improved recovery of upper-spinal inhibition.[4]

Due to the regulation of sympathetic activity by various cortical and subcortical regions, isolated brain lesions are typically insufficient to induce PSH. Instead, PSH is more frequently linked with widespread, diffuse, or multifocal brain injuries, particularly diffuse axonal injury and damage to the periventricular white matter, corpus callosum, diencephalon and upper brainstem, as indicated by neuroimaging studies.[2]

However, the precise contribution of lesion location and laterality to PSH onset remains unclear. Severe TBI often produces diffuse injury patterns that obscure the identification of discrete structures responsible for triggering PSH. In addition,

the limited availability of standardized neuroimaging data and the clinical heterogeneity of PSH make it difficult to isolate its specific effects from those of the primary brain injury.[4]

Clinical features

Paroxysmal sympathetic hyperactivity is characterized by sudden, recurrent episodes of increased sympathetic and motor activity, usually triggered by non-noxious stimuli like suctioning, passive movement, or changes in posture.[1,2] Up to 72% of cases are linked to these unavoidable factors, though some can occur spontaneously.[2]

A typical episode involves a paroxysmal rise in heart rate, blood pressure, respiratory rate, body temperature, and sweating, often with dystonic posturing, characterized by sustained abnormal body posture resulting from involuntary muscle contractions.[1,2] Less experienced observers might confuse these episodes with tonic seizures.[2] Tachycardia is the most common sign, while use of sedatives and analgesics may mask other symptoms. Additional signs of excessive sympathetic activity include pupillary dilation, tremors, piloerection, hyperreflexia, clonus, ileus, and urinary retention.[5]

Over time, these episodes usually become less severe. Although PSH often resolves within a few weeks, some symptoms — such as tachycardia, sweating, and posturing — may continue to appear even during the rehabilitation phase.[4]

These episodes can occur at any stage after brain injury, from the acute intensive care phase to rehabilitation, and typically last from a few minutes to two hours, with an average duration of about 30 minutes in ICU settings.[1-3]

Episodes generally evolve through three phases. The hyperacute phase (phase I) occurs within the first week after injury, when the brain remains unstable and diagnosis can be obscured by sedation or analgesia. The established phase (phase II) lasts up to approximately 2.5 months after injury, during which the syndrome fully manifests and concludes when sweating episodes cease. The resolving phase (phase III) happens during rehabilitation and may persist for years, though the frequency, intensity, and duration of episodes generally decrease over time.[11]

In severe or prolonged cases, PSH may cause complications such as tachyarrhythmias, stress-induced cardiomyopathy, pulmonary oedema, worsening intracranial hypertension, rhabdomyolysis, dehydration, malnutrition, and muscle contractures. Prompt recognition and treatment are essential to prevent these complications and improve outcomes.[5]

Diagnostic

Paroxysmal sympathetic hyperactivity is a clinical diagnosis typically confirmed after exclusion.[2,3,9] It depends on the presence of several signs of sympathetic hyperactivity occurring at the same time.[2] To improve the accuracy of diagnosing PSH, an international consensus in 2014 introduced the PSH Assessment Measure (PSH-AM), a clinical tool designed for this purpose.[2,8]

The PSH-AM consists of two parts: the Clinical Feature Scale (CFS), which assesses the severity of sympathetic and motor activity symptoms during episodes, and the Diagnostic Likelihood Tool (DLT), which evaluates the likelihood of PSH based on the frequency and duration of symptoms. These components yield a score that indicates whether PSH is unlikely (< 8), possible (8 - 16), or probable (≥ 17).[2-4] Evidence from previous and recent cases supports that the PSH-AM can provide reliable diagnostic criteria and help categorize the severity of PSH.[4]

The CFS component assesses symptom severity, including elevated body temperature, heart rate, respiratory rate, and symptoms like posturing and diaphoresis.[1] Each symptom is categorized based on how far it deviates from normal physiological conditions and is assigned a numerical grade; these values increase with worsening symptom severity. The total CFS score is calculated by summing the scores of all individual symptoms, resulting in a graded severity score for clinical features.[3] The DLT section of the PSH-AM criteria focuses on the frequency and duration of symptoms associated with sympathetic hyperactivity.[3]

The PSH-AM tool has been validated for feasibility and reliability, indicating that it could help decrease misdiagnoses, thereby reducing hospital stays and costs.[2]

Treatment

Successful management requires a comprehensive approach that combines pharmacological and non-pharmacological strategies to manage symptoms, prevent exacerbations, and reduce complications.[1-3]

The main goals of treatment include avoiding triggers that lead to paroxysms, reducing excessive sympathetic outflow, and providing supportive care for systemic effects.[1-4]

Paroxysmal sympathetic hyperactivity treatment encounters difficulties stemming from individual differences in pathophysiology, treatment responses, and overall outcomes. Significant challenges include a limited understanding of the brain regions involved, an ambiguous connection between neurotransmitters or hormones and clinical symptoms, an absence

of standardized methods to assess treatment efficacy and a scarcity of clinical trial evidence on the long-term benefits of interventions. These issues lead to variability in PSH treatment strategies.[4,10]

In addition to standard pharmacologic and supportive measures, adjunctive approaches that enhance cerebral oxygenation and metabolic recovery have also been explored in refractory cases, such as hyperbaric oxygen therapy (HBOT).[12]

Non-pharmacological treatment and supportive care

The non-pharmacological approach to PSH management emphasizes supportive care and environmental strategies to enhance patient outcomes and tackle complications.[2]

As PSH patients frequently exhibit heightened reactions to sensory input, a vital aspect of this is reducing external triggers. To prevent worsening of the condition, caregivers should consolidate nursing interventions, foresee potential symptom flare-ups during activities such as bathing, and minimize extraneous handling. Controlling temperature is also essential, as hyperthermia frequently arises in PSH cases. By controlling the room temperature, using cooling blankets, and ensuring proper hydration, caregivers can assist in regulating the patient's body temperature.[2,4]

Nutritional support plays a vital role in PSH management. With resting energy expenditure potentially reaching three times the baseline value, patients may face substantial weight loss. To counteract this, it is crucial to ensure adequate caloric intake and fluid replacement to meet metabolic needs. Moreover, effective hydration management is essential, as patients risk dehydration and electrolyte imbalances due to excessive sweating and heightened metabolic requirements. Intravenous fluids and enteral hydration are often necessary to sustain the appropriate fluid balance.[4]

Pharmacological treatment

Managing the complexities of PSH often requires a combination of medications, both abortive and preventive, to address various aspects of the condition. Abortive therapies aim to interrupt symptoms after episode onset, whereas preventive treatments are used to reduce the frequency or severity of episodes. Some drugs, including β-blockers and clonidine, serve dual purposes: they are both abortive and preventive options. In contrast, medications like morphine are primarily used for abortive treatment, while gabapentin is typically a preventive therapy.[2]

Opioids, particularly morphine, are among the most prescribed medications that not only relieve pain but also influence central pathways related to PSH.[1,6] Typically, the duration of opioid treatment is determined by the duration and intensity of the PSH symptoms, weighed against the need to prevent long-term opioid use. Nevertheless, opioid treatment frequently continues during the rehabilitation stage.[1,7]

Gabapentin is widely prescribed for neuropathic pain and has shown effectiveness in patients who do not respond to other treatments, especially in managing hypertonicity and sensory hypersensitivity.[1,7] Although evidence remains limited to case reports and small series, gabapentin can also be incorporated into abortive therapy when hypertonicity is a prominent feature or when paroxysmal discharge episodes persist despite maximum doses of morphine and propranolol.[13]

Clonidine and dexmedetomidine are α2-adrenergic agonists that suppress adrenergic outflow at both the central and peripheral levels, leading to decreased heart rate, blood pressure and catecholamine levels. However, their effectiveness in regulating temperature is limited.[1,7]

Non-selective β-blockers, especially propranolol, are commonly used for their ability to cross the blood-brain barrier and alleviate cardiovascular and thermoregulatory issues. Conversely, cardioselective β-blockers, like metoprolol, appear to be less effective.[1,7]

Additional medications include dopaminergic agonists (e.g., bromocriptine), which demonstrate inconsistent efficacy in regulating temperature and autonomic instability, as well as baclofen, a GABA B receptor agonist that assists in controlling spasticity.[1,7]

Dantrolene, a potent muscle relaxant commonly prescribed for malignant hyperthermia, may be beneficial in challenging cases, particularly with concern to posture.[1,2,7]

Antipsychotic agents (e.g., haloperidol, quetiapine) are not recommended as targeted therapy for PSH. Their use should be limited to cases of concurrent agitation or delirium, and even then, applied with caution, as they do not address the underlying sympathetic overactivity.[2,7]

Pharmacological management is usually tailored based on the patient's individual response, where a combination of medications tends to be more effective than using a single drug alone. Ideally, pharmacological treatment should start in the ICU and continue at least through the initial rehabilitation phase.[2] Recently, a retrospective study indicated that initial symptom severity does not play a significant role in guiding drug selection, challenging the prior assumption that medication influences the progression of PSH.[4] Despite the variety of drugs available, no singular treatment is effective for

everyone, and clinical evidence remains somewhat limited.

Prevention and challenges

Preventative measures seek to reduce excessive sympathetic activity, with some studies indicating that the early administration of dexmedetomidine in patients with TBI might lower the occurrence of PSH.[11] However, there is a lack of standardized treatment protocols due to an incomplete understanding of the pathophysiology of PSH, a lack of definitive biomarkers, and differences in patient reactions. Although treatment mainly aims to manage symptoms, approaches in precision medicine have been inconsistent across studies.[7]

The efficacy of preventive drug treatment for patients at risk of PSH but without a PSH diagnosis remains uncertain and cannot be recommended.[9]

Prognosis

Paroxysmal sympathetic hyperactivity is closely linked to poorer short- and long-term outcomes in patients suffering from TBI.[10] The severity and extent of brain injuries, particularly those affecting midbrain regions like the periaqueductal gray matter, play a crucial role in the onset and recovery from PSH.[1] Nevertheless, it remains uncertain whether PSH itself worsens prognosis or reflects more severe brain injuries.[2,8]

Research presents mixed results regarding the independent effect of PSH on clinical outcomes; however, most of the evidence suggests that it results in longer hospital stays, lower GCS scores, and increased complications (e.g., infections, muscle contractures, and heterotopic ossification, defined as the abnormal formation of bone within soft tissues such as muscles and tnedons).[3,4,10]

Delayed recognition and inadequate management of PSH can lead to worse outcomes, causing unnecessary diagnostic tests, inappropriate medication use, and prolonged hospital stays. Patients exhibiting severe PSH symptoms face a greater risk of secondary brain injury from hypertension, hyperthermia, and cardiac issues, which can be life-threatening.[4,14]

The number of PSH symptoms appears to be a more significant predictor of poor outcomes than the duration of the syndrome itself, as a higher symptom burden is associated with worse neurological recovery.[11] While some studies report no significant outcomes, clinicians generally agree that PSH impairs rehabilitation potential and overall recovery, especially in patients with prolonged dysautonomic symptoms and reduced levels of consciousness.[5,7,11]

Algorithm for management of PSH

Given the complexity and heterogeneity of PSH presentation, we developed a practical clinical algorithm (Fig. 2) based on the literature review and expert consensus. The algorithm outlines the key stages of diagnosis and management, incorporating both pharmacological and non-pharmacological strategies. Its main goal is to standardize clinical methods, promote early recognition, reduce complications, and enhance patient outcomes. The first step involves confirming PSH in a suspected patient using the PSH-AM [Appendix 1 (Appendix 1: https://www.actamedicaportuguesa.com/revista/index.php/amp/article/view/23395/15867)]. Because PSH is a clinical diagnosis, differential diagnoses with overlapping symptoms must be excluded—this process is detailed in Appendix 2 (Appendix 2: https://www.actamedicaportuguesa.com/revista/index.php/amp/article/view/23395/15868). Once alternative causes are ruled out and PSH is confirmed, treatment should follow the sequential approach outlined in the algorithm, combining non-pharmacological measures [Appendix 3 (Appendix 3: https://www.actamedicaportuguesa.com/revista/index.php/amp/article/view/23395/15869)] and pharmacological interventions, including abortive and preventive therapies [Appendix 4 (Appendix 4: https://www.actamedicaportuguesa.com/revista/index.php/amp/article/view/23395/15870)].

CONCLUSION

Paroxysmal sympathetic hyperactivity is a complex and often neglected syndrome that primarily occurs after severe TBI but can also result from other acute brain conditions. It is marked by episodic sympathetic and motor hyperactivity. Early detection is vital to avoid misdiagnosis and complications. Tools like the PSH-AM help identify and assess severity, while imaging can reveal relevant brain injuries.

Effective management requires a multimodal approach that combines pharmacological and non-pharmacological strategies. Although awareness of PSH is growing, significant gaps in knowledge and ongoing debates remain. Current evidence is limited by small retrospective studies and the lack of randomized controlled trials. Clinical practice varies widely, with ongoing discussions about the best medication protocols, the predictive value of initial symptoms, and the role of preventive therapy in high-risk but undiagnosed patients. These issues hinder the development of standardized management

guidelines.

Future research should focus on creating reliable diagnostic tools, multicenter prospective studies, and consensus-driven treatment protocols to standardize clinical practice. Although the exact mechanisms of PSH are not yet fully understood, ongoing advances in understanding and managing the condition offer hope for better diagnosis, consistent therapy, and improved patient outcomes.

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AUTHOR CONTRIBUTIONS

RSA: Study conception and design, writing of the manuscript.
JF: Study conception and design, critical review of the manuscript.
BQ, PC, IB, CFS, AA: Critical review of the manuscript.
All authors approved the final version to be published.

CONFLICTS OF INTEREST

The authors have no conflicts of interest to declare.

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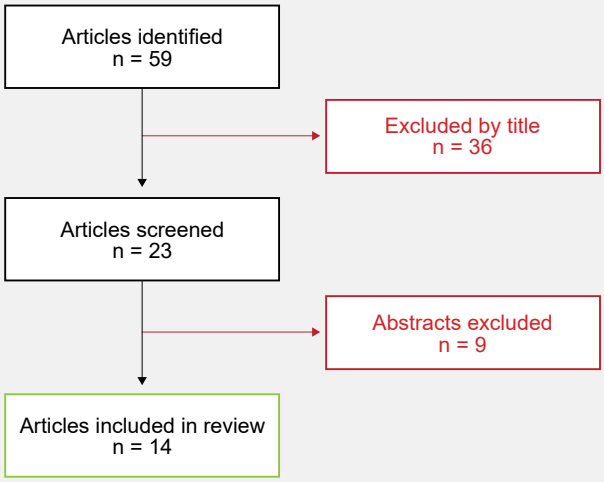

Figure 1 – Flow diagram showing the selection process of studies included in the narrative review on paroxysmal sympathetic hyperactivity

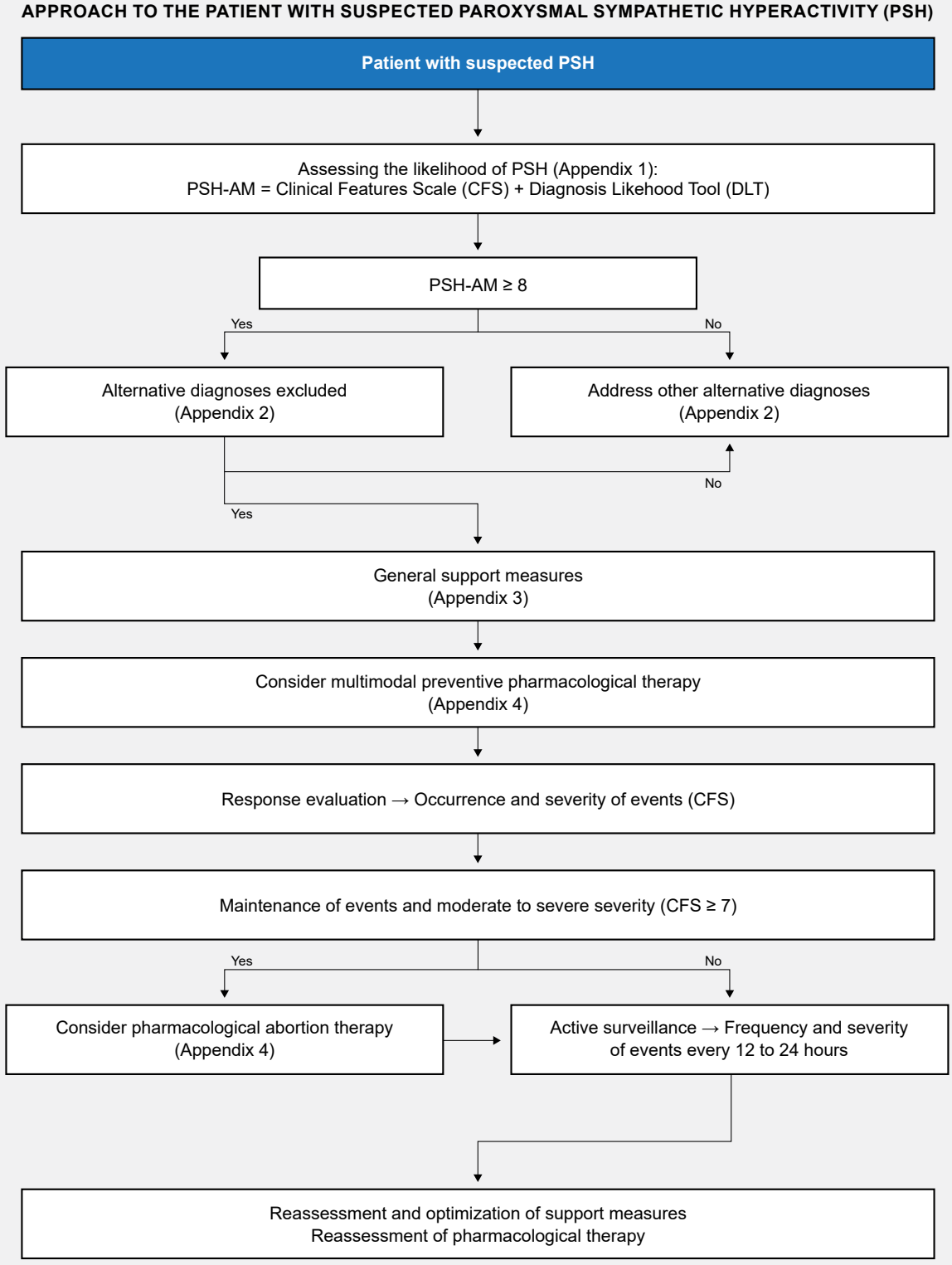

Figure 2 – Proposed clinical algorithm for the identification and management of paroxysmal sympathetic hyperactivity. The algorithm summarizes a stepwise approach based on literature review and expert consensus, integrating diagnostic assessment, exclusion of differential diagnoses, and multimodal treatment strategies. It is intended as a practical tool to guide clinicians in intensive care, neurology, and rehabilitation settings.