Paroxysmal Sympathetic Hyperactivity: A New Era for Diagnosis and Treatment

In a recent issue of The Journal of Head Trauma Rehabilitation, Pozzi et al. present an interesting study that describes a retrospective series of 26 patients taken from a series of 407 children with acute brain injury admitted to the pediatric neurorehabilitation unit, of whom almost half had experienced a traumatic brain injury. This study is of great value as it further examines a complication that was until recently ignored and presented by a few patients with acute brain injury. The study has even greater relevance as it involves children, who have received much less attention in this respect, as well as having a follow-up for 3 years. This prolonged duration is able to determine the outcome of this serious complication, in terms of both greater short-term monitoring, for example, in intensive care units, and longer-stay areas, for example, in rehabilitation services.

To detect episodes of paroxysmal sympathetic hyperactivity (PSH), the study used as a diagnostic tool “acute events of agitation with at least 1 subjective alteration (sweating; muscular tone increase) and either 2 measured elevations (heart rate, systolic blood pressure, respiratory rate, or temperature) or 1 severe elevation, according to medical judgment.” We consider the diagnostic criteria rather confusing, as a diagnosis could be made with 2 of the 3 groups of symptoms proposed, in addition to which both the latter groups lack objective definitions. Previously, the criteria used to diagnose PSH were heterogeneous and this has undoubtedly constituted an obstacle when determining the course and prognosis of these patients. The recent Consensus Conference, with the participation of relevant specialists from various different treatment areas and geographical regions, all of whom had published reports about patients with this condition, unified the different nomenclature used to define PSH. Pozzi et al. use the term “PSH” to describe this complication, as proposed in the consensus conference that also suggested certain common diagnostic criteria based on which all the cases and series published constitute cases about which there is no doubt the patients had this syndrome. If this is not done, there is a risk (as has happened previously) that some cases considered to have PSH may in fact concern other types of disorders, such as deprivation of certain drugs or extracerebral complications, and/or may not actually include patients who do indeed have PSH. The fact that the series here involves children in no way invalidates these criteria, although the scale has not as yet been definitively validated in this population. Thus, despite the retrospective nature of the study, it would nevertheless have been interesting if the diagnostic scale proposed at the consensus conference had been used, as it should be in the future, to have more rigorous information about the incidence, course, prognostic repercussion, and treatment response of episodes of PSH.

As these authors found greater rates of morbidity and mortality in those patients who presented this syndrome than in those who did not, they sought forms of treatment that might improve the clinical manifestations. Although the authors state that their results “provide a systematic assessment of drug efficacy,” we consider that retrospective studies have inherent failings due to the nature of the study when it comes to evaluating treatment efficacy, especially for the symptoms of PSH, which, by definition, are self-limiting, even with no treatment. The period used to determine the efficacy of the drug used (30 minutes) is, for the same reason, arbitrary. Of note, too, is the use of certain drugs the action of which is largely unknown, such as hydroxyzine, or drugs not marketed in other areas of Europe, such as niaprazine, with an \( \alpha_1 \)-adrenergic antagonist effect, but which showed positive results in this study. Others have proposed numerous therapeutic alternatives, including baclofen, gabapentin, bromocriptine, or morphine, which have been assessed in small series but as yet with no verified result.

We consider that, at the present time, any advances in the search for an efficient treatment of PSH require better understanding of the underlying pathophysiological basis explaining the symptoms of PSH. Although
numerous theories have been put forward, there is still no certain comprehension of the brain areas involved, nor of the neurotransmitters or systemic hormones associated with the clinical manifestation of the syndrome, although several mechanisms have been proposed. We consider that study of these factors would be more feasible in the area of rehabilitation rather than in intensive care units, where the requirements for monitoring and the instability of the patients hinder their transfer, as well as the performance of magnetic resonance imaging with spectroscopy and diffuse tensor imaging. During the course of an episode of PSH, these new imaging techniques could help localize the specific brain areas involved, whether through discharge or inhibition. Computed tomographic studies have shown areas involved in the brain injury of PSH patients, although without discerning the specific areas involve in PSH.

In our opinion, future studies aimed at better understanding of the therapeutic options to prevent and treat PSH should be preceded by studies using imaging techniques or blood tests to determine the intimate pathophysiology of this complication. Likewise, we consider that new studies should only recruit those PSH patients who fulfill the parameters agreed by the expert committee.

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