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Letters to the Editor

Hüfner and Sperner-Unterweger [1] raised a number of important points about the construct and classification of persistent postural-perceptual dizziness (PPPD) in the International Classification of Vestibular Disorders (ICVD), most of which were addressed in the manuscript that described PPPD in 2017 [2]. Nevertheless, their letter to the editor offers a welcome opportunity for additional contemplation and clarification.

The ICVD was created to advance clinical care and research for all patients suffering from vestibular symptoms. Vestibular and balance complaints are common throughout the world and are seen by clinicians from multiple medical specialties; thus, the ICVD diagnoses must be applicable across continents and clinical disciplines. Hüfner and Sperner-Unterweger wrote from the viewpoint of psychosomatic medicine, which is a distinct specialty separate from psychiatry and psychology in some parts of the world, especially in German-speaking regions of Europe where it is taught in its own training programs. Those who practice this specialty take an integrated mind-body approach to clinical problems. Throughout the rest of Western-influenced medicine, however, the Freudian roots of psychosomatic concepts are interpreted with greater emphasis on the psyche than the soma, pushing problems considered to be "psychosomatic" out of medical-surgical practices and research endeavors. The emphasis of psyche over soma also makes it difficult to translate psychosomatic insights into regions of the world that maintain close connections to Eastern-inspired medical traditions. This necessitated a more universally applicable formulation of PPPD grounded in emerging evidence about its underlying pathophysiological processes.

Nearly 200 years ago, the term functional was used to identify conditions thought to be caused by changes in the "mode of action" of organ systems not directly attributable to structural deficits [3]. This concept was adapted for the ICVD to identify conditions in which shifts of physiological functioning are considered to be the primary pathophysiological mechanisms, potentially influenced by, but not solely determined by, associated structural and psychological processes. When PPPD was defined in 2017, available evidence suggested that specific alterations in the functioning of postural control and spatial orientation systems in the brain may underlie the disorder. Subsequent research has strengthened this hypothesis and begun to clarify the relationships of these functional shifts to structural and psychological factors that may predispose and precipitate them. In clinical practice, this places the emphasis on identifying the key symptoms listed in the diagnostic criteria for PPPD, regardless of the presence or absence of additional symptoms that may indicate co-existing structural or psychiatric conditions. In research settings, this keeps the focus on putative alterations in physiological functions, while demanding that investigators account for the effects of structural and psychological processes as well. Hüfner and Sperner-Unterweger's statement that chronic stress may alter various physiological parameters is accurate, but generically applicable to any medical condition. A general concept of stress provides neither an explanation for the specific clinical symptoms of PPPD nor inspiration for detailed investigations of its pathophysiological processes, including those that may be distinct from, as well as shared by, other disorders.

The definition of PPPD was meant to identify a specific clinical condition, not to cover the broad range of patients who may present with enigmatic vestibular symptoms, with or without psychiatric morbidity. Criterion C enumerates its precipitants to bring all clinicians' attention to the broad range of potential triggering events, encouraging otologists, for example, to consider more than peripheral or central vestibular deficits or cardiologists more than dysrhythmias, emphasizing the cross-disciplinary nature of the diagnosis. Anxiety and depressive symptoms were not included in the definition of PPPD because a significant portion of patients with PPPD does not have these symptoms and, even when psychological symptoms co-exist, they add no sensitivity or specificity to the diagnosis [4]. Hüfner and Sperner-Unterweger misinterpreted criterion E, "Symptoms are not better accounted for by another disease or

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disorder." As stated explicitly in the defining document [Note (7) and Section 4.3], PPPD is not a diagnosis of exclusion and often co-exists with other conditions. This criterion, which is present in the definitions of all ICVD disorders, is meant to ensure that clinicians consider all symptoms reported by their patients as well as the full range of the differential diagnosis before coming to a final determination of the best diagnostic formulation of their patients' conditions. Hüfner and Sperner-Unterweger's alignment of PPPD with somatic symptom disorder/bodily distress disorder is inconsistent with key features of those conditions and emerging data on this topic. Those disorders include not just the presence of distressing somatic symptoms, but excessive illness-related worries and aberrant healthcare seeking behaviors [5, 6]. Those illness-anxious features were present in fewer than 15% of patients with chronic subjective dizziness, one of the forerunners of PPPD [7], a rate similar to that seen in patients with other medical conditions. In a recently completed investigation that specifically included patients with PPPD, the nature and severity of illness-anxious beliefs was found not to differ between patients with functional versus structural vestibular disorders [8]. Thus, PPPD does not appear to be any more closely related to somatic symptom disorder/bodily distress disorder than structural vestibular disorders or other medical illnesses. The defining manuscript of PPPD [Section 4.4.6] made provisions for diagnosing somatic symptom disorder in addition to or instead of PPPD in the minority of patients who truly meet criteria for that psychiatric disorder.

In the late 1800 s, the German neurologist Carl Westphal engaged in a wide-ranging debate with fellow physicians about the causes of vestibular, visual and psychological symptoms reported by patients struggling to navigate the busy marketplaces of European town squares [9]. He wrote that patients "want[ed] an explanation for their suffering but … [did] not want to be laughed at, or worse, be considered insane." [10, 11]. Many of the individuals observed by Westphal and his contemporaries would today receive a diagnosis of PPPD, possibly with additional diagnoses of comorbid conditions. Meeting Westphal's challenge to explain the suffering caused by a multi-faceted disorder like PPPD requires insights from clinicians and researchers of many disciplines, but it also demands that experts from various fields translate their unique contributions into a universally understandable construct. To that end, the members of the ICVD subcommittee that defined PPPD, including experts from otology, neurology, psychiatry, and psychosomatic medicine, conceptualized it as a functional disorder to emphasize the shifts in physiological functioning of postural control and spatial orientation systems that appear to underlie it. Various structural and psychological processes may predispose and precipitate PPPD as described in its defining manuscript and other research reports, but they are not as fundamental to its existence as these key functional shifts.

From the earliest inception of the ICVD, the Bárány Society expected that ongoing input from concerned clinicians and researchers would improve the definitions of diseases and disorders contained in it. Hüfner and Sperner-Unterweger's interest and thought-provoking comments about PPPD provided a chance to examine the disorder once again through the lens of psychosomatic medicine and to explain in more detail how its ICVD formulation offers patients, clinicians, and researchers of all disciplines a succinct description of its core symptoms, boundaries with co-existing conditions, and key pathophysiological processes based on currently available evidence.

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