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Introduction

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The last 30 years has seen a growing awareness of migraine as a major cause of dizziness, imbalance, and vertigo. Historically, the migraine literature has been dominated by migraine's role as a cause of headache. Landmark papers by Kayan and Hood (Brain, 1984), by Cutrer and Baloh (Headache, 1992), by Neuhauser et al. (Neurology, 2001), and others, have succeeded in slowly turning the spotlight on migrainous balance symptoms. Of course, migraine is associated with many symptoms – scintillating scotomata, cutaneous dysesthesias and allodynia, abdominal pain, photophobia, phonophobia, osmophobia, tinnitus, vertigo, disequilibrium, and more. The traditional notion of migraine as a headache illness with adjunctive ocular, vestibular, and other symptoms may be giving way to the more modern conception of migraine as a central disturbance of sensory signal processing, of which headache is the cardinal symptom. Certainly, in the clinical domain of "dizzy doctors," otologists, otoneurologists, physiatrists, and others, there has been an explosion of awareness of and interest in migraine as a diagnosable, treatable cause of dizziness. These patients may now account for as much as one third of patients found in dizzy clinics worldwide. The 2010 Midwinter Meeting of the Association of Research in Otolaryngology (ARO) hosted a Presidential Symposium on this topic of migrainous dizziness entitled, "Migraine Dizziness: From Bedside to Bench and Back Again." As the title indicates, the objective of the symposium was to help define and articulate the clinical problem, look to basic science for insights into the underlying pathophysiologic underpinnings of migraine, present some early translational science that demonstrates how the basic science can aid us in the clinic, and finally to consider what the future might hold. In this issue of JVR we present manuscripts based upon these ARO presentations.

The first paper in this suite of manuscripts is by von Brevern and Neuhauser, entitled, "Epidemiologic evidence for a link between vertigo and migraine." This paper presents a summary and update of the fine epidemiologic studies of the German team. They note that the worldwide lifetime prevalence of migraine is 14% and that 20–30% of the general population is affected by dizziness and vertigo, so there is a statistical certainty that many people will experience both conditions. However, they have confirmed a link between migraine and vertigo at the epidemiologic level. The proportion of the population exhibiting both symptoms exceeds the expected level if the two groups were independent and the symptoms co-occurred by chance. They found migrainous vertigo to be common, with a lifetime prevalence of about 1% in the general population. This prevalence is much higher in groups with other vestibulopathies, such as benign paroxysmal positional vertigo (BPPV) and Meniere's disease. They also found migraine to be associated with motion sickness, ataxia disorders, and psychiatric syndromes. Vestibular migraine is the most common cause of spontaneous recurrent vertigo

The second paper, by Burstein et al., is a review of some of the elegant animal research his lab has done to elucidate the central nociceptive pathways and processes that underlie migraine headache and allodynia. They have discovered that the first set of activated neurons in migraine headache, in the trigeminal ganglion, undergo molecular changes within 10–20 minutes after onset of the attack that make them sensitive to changes in intracranial pressure, such as bending, straining, or coughing. If the pain is not stopped by treatment within 60–120 minutes after onset of the attack, a second set

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of neurons, in the spinal trigeminal nucleus, undergo molecular changes that convert them from dependence on the dural sensory signals to an independent state as pain generators of the headache and as a cause of cutaneous allodynia from hair brushing, showering, shaving, touching the skin, etc. The transition from activity-dependent to activity-independent central sensitization correlates with a loss of responsiveness to triptan migraine abortive medications. Implications of these findings for migraine pharmacotherapy are obvious.

In the third paper, Balaban pulls together diverse threads of research and logic to paint a speculative but convincing picture of the various ways that central activation may produce both pain and peripheral and central vestibular symptoms in migraine. He proposes that additive effects of afferent vestibular and pain information in pre-parabrachial and pre-thalamic pathways influence cortical mechanisms of perception, interoception, and affect. He enumerates parallel neurochemical phenotypes in the inner ear and trigeminal ganglion cells and describes how they converge for vestibular and nociceptive information processing. He presents new evidence on the distribution of serotonin receptors in the primate crista ampullaris that could play a role in inner ear ionic homeostatic mechanisms that accompany migrainous audiovestibular symptoms. His putative schema hints at therapeutic approaches that may hold promise for arresting or reversing these symptoms.

Lewis et al. have capitalized on the model of migraine dizziness as a manifestation of failed sensory integration due to central activation. They have developed a novel and exciting psychophysical measure of motion perception and shown that the detection thresholds for certain motions are altered in patients with migraine-associated dizziness but not in controls nor in migraineurs without dizziness. They compared head motion detection thresholds for roll rotation while supine, for quasi-static roll tilt, and for dynamic roll tilt using a staircase paradigm whereby the peak acceleration of the motion was increased or decreased based on incorrect or correct reports of movement direction. There was a dramatic reduction in motion thresholds in the vestibular migraine cohort compared to normal and migraineurs without vestibular symptoms in the dynamic roll tilt paradigm. The authors conclude that patients with vestibular migraine may have enhanced perceptual sensitivity for head motions that dynamically modulate canal and otolith inputs together.

Finally, Staab, a psychiatrist and biomedical engineer with special expertise and interest in psychosomatic disorders and balance, explores the relationships of anxiety, migraine, and dizziness. While these relationships are popular and widely held notions, a review of the literature reveals surprisingly little robust research to validate them. Staab hypothesizes some possible central pathways to explain these relationships and proposes creative clinical trials using "pharmacologic dissection" to differentiate between pathways on the basis of symptom response to specific drugs. He charts a path to better definitions of clinical phenotypes, better understanding of underlying pathophysiology, and evidence-based therapeutic interventions.

Migraine dizziness represents fascinating neurobiology and fascinating clinical medicine. It is an evolving field. It is common in the clinic as a pleomorphic but treatable cause of balance symptoms. Whether you are a basic scientists, translational scientist, or clinician, we hope that you will find that the overview of this topic in these excellent papers will give you new and exciting information and will pique your interest to learn more.