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RESPONSE TO LETTER TO THE EDITOR ON PUBLICATION: KUMAGAMI H, SAINOO Y, FUJIYAMA D, BABA A, OKU R, TAKASAKI K, SHIGENO K, TAKAHASHI H.

SUBJECTIVE VISUAL VERTICAL IN ACUTE ATTACKS OF MÉNIÈRE’S DISEASE. OTOL NEUROTOL 2009;30:206-9

To the Editor: Thank you very much for your interest in our research. In the present study (1), the subjective visual vertical (SVV) test was conducted to investigate whether and to what extent patients with Ménière’s disease have otolithic dysfunction in an acute attack. Because the SVV test is widely recognized as a test for evaluating the otolithic organs and the central graviceptive pathways in previous studies (2-4), and certain authors even define the SVV test as a utricular function test (5). However, we also recognize that the dysfunction of the posterior semicircular canal is a component included in abnormal tilts of SVV. In a previous publication (6), we presented patients with Ménière’s disease showing abnormal ocular torsion in an acute attack. We suggested the possibility of abnormal ocular torsion consisted of dysfunction of the posterior semicircular canal as well as of the otolithic organs. We have unpublished observations showing that patients with Ménière’s disease have abnormal tilts of SVV and ocular torsion occurring at the same time during an acute attack. Indeed, SVV tilts are observed during rotation (7), and therefore, SVV tilts are generated by the stimuli of the posterior semicircular canal. However, SVV tilts generated by the rotation are accompanied by the torsional nystagmus (7). In patients with Ménière’s disease showing abnormal tilts in acute attacks, the periods when nystagmus finally disappeared did not always correspond to normalization of SVV. The abnormal tilts of SVV observed during the attacks continued on longer than the nystagmus. We therefore assumed that the abnormal tilts and the static abnormal eye movement are mainly derived from dysfunction of the otolithic organs. We assume that these abnormalities could not have been observed after the nystagmus subsided if dysfunction in the posterior semicircular canal had mainly acted on SVV and ocular torsion. In addition, according to our data, the percentage of patients with posterior canal benign paroxysmal positioning vertigo showing abnormal tilts of SVV is relatively low (20-30%). Also from personal experience, in definite cases of Ménière’s disease which were initially diagnosed possible cases, some cases show abnormal tilts of SVV when a fluctuating hearing loss without a vertiginous episode or nystagmus is present. Although the influence of the posterior semicircular canal on SVV cannot be denied, we assumed that
abnormal tilts in acute attacks of Ménière’s disease are mainly derived from otolithic dysfunction for the above-mentioned facts. However, we agree that visual vertical perception remains unclear, and whether and to what extent dysfunction of the posterior semicircular canal is reflected on SVV is not well understood yet. Thus, the scientific basis with regard to whether and to what extent the posterior semicircular canal acts on SVV should be investigated further. However, from a clinical viewpoint, the SVV test seems to be a simple and good test for evaluating both the central graviceptive pathways and presumably otolithic organs. We think that the SVV test is a valid method to explain the condition of vertical perception to at least patients and that this explanation is beneficial for both patients and doctors.

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