



Editorial

Vestibular evoked myogenic potentials (VEMPs) in systemic disease



See Article, pages 795–801

Recording of vestibular evoked myogenic potentials (VEMPs) is a non-invasive electrophysiological method to determine function and dysfunction of the vestibular nervous system (both peripheral and central), which pertains more specifically to otolith function. Clinical indications that have been examined up to now mainly involve specific vestibular causes such as vestibular neuritis, superior semicircular canal dehiscence, vestibular migraine and Meniere's disease. However, causes of vestibular dysfunction have rarely been systematically documented in the literature. According to a literature search on PubMed, there are no known reports of VEMP findings in cases such as mastocytosis, chronic fatigue syndrome, systemic vasculitis, sarcoidosis, hypothyroidism, adrenal insufficiency, coeliac disease, ulcerative colitis, Crohn's disease, hypertension, metabolic syndrome, Grave's disease, systemic lupus erythematosus, atherosclerosis, sickle cell disease and myasthenia gravis. However, a few reports exist with respect to type-2 diabetes (diabetes mellitus). D'Silva et al. (2017) reported a significant increase in abnormal ocular (oVEMP) and cervical (cVEMP) responses compared to normal controls. However, this was based on absolute latency and the presence or absence of a response. Amplitude was not analyzed, even though muscle contraction was monitored in the cVEMP group. Therefore what has been labelled as normal may in fact have been a response reduced in amplitude. Details were not provided as to what percentage of abnormal results was either a delayed or an absent response. This is unfortunate as a delayed response may typically suggest central rather than peripheral dysfunction (although there are exceptions). Ren et al. (2018) did not control for sternocleidomastoid muscle contraction, making their cVEMP amplitude measurements unreliable. Unfortunately, latency was not measured. It is remarkable that another well-performed study (Bektas et al., 2008) did not find any cVEMP abnormalities in diabetic patients. However, although vestibular symptoms are usually present (Perez et al., 2001), none of the patients in the above study had such symptoms.

Only one study was found with respect to fibromyalgia (Bayazit et al., 2010). Again amplitude measurements were unreliable here due to failure to control for muscle contraction. However, what was interesting in that study is a selective prolongation of n23 absolute latency, in contrast to the p13 absolute latency which was not significantly different from normal controls. In a study of patients with Human T cell lymphotropic virus type-associated myelopathy/tropical spastic paraparesis (Labanca et al., 2015),

amplitudes were not evaluated, with latency used as the only marker of abnormality. In addition, abnormalities were taken for granted to indicate involvement of the vestibulospinal tract, without taking into account that peripheral vestibular involvement may also have been a cause. Good and accepted methodology, with EMG monitored, was applied in a study related to rheumatoid arthritis, with the measurement of both latency and amplitude (Heydari et al., 2015). Significant prolongation in absolute latencies was found with no changes in amplitude in this patient group.

Clearly there is a need for further publications with respect to the effect of systemic disease on VEMPs, not only because such studies are rare but because studies with good and accepted methodology and interpretation are needed also. Therefore, the article by Jung et al. (2019) published in this issue of *Clinical Neurophysiology* that relates cVEMP measurements to glomerular filtration rate and free thyroxine level in the elderly is highly welcome in this respect. In this study, they report that decreased glomerular filtration rate and increased free thyroxine, as well as aging, appear to alter saccule pathway related activity as manifested in the cVEMP. Amplitude was altered and not waveform latency, and the possible reasons for this are analyzed in detail in the feature paper. Unfortunately oVEMPs were not studied.

It is difficult to generalize here with respect to systemic disorders and the effects on the recorded VEMPs. We would like to answer the question as to whether amplitude or latency is preferentially affected, but the literature is too incomplete. This would be an interesting fact to know, but further studies are needed. Most of the initial studies described above require validation. A thorough history should include noting comorbid conditions, as listed above, that may cause alterations in VEMPs. An abnormal VEMP may not necessarily indicate pure vestibular dysfunction but may be a result of systemic causes.

Conflict of interest

None of the authors have potential conflicts of interest to be disclosed.

References

- Bayazit YA, Celenk F, Gunduz AG, Gunduz B, Ondag N, Meray J. Vestibular evoked myogenic potentials in patients with fibromyalgia syndrome. *J Laryngol Otol* 2010;124:610–5.

- Bektas D, Gazioglu S, Arslan S, Cobanoglu B, Boz C, Caylan R. VEMP responses are not affected in non-insulin-dependent diabetes mellitus patients with or without polyneuropathy. *Acta Otolaryngol* 2008;128:768–71.
- D'Silva LJ, Staecker H, Lin J, Maddux C, Ferraro J, Dai H, Kluding PM. Otolith dysfunction in persons with both diabetes and benign positional vertigo. *Otol Neurotol* 2017;38:379–85.
- Heydari N, Hajiabohassani F, Fatahi J, Movaseghi S, Jalaie S. Vestibular evoked myogenic potentials in patients with rheumatoid arthritis. *Med J Islam Repub Iran* 2015;29:216.
- Jung I, Ahn S-H, Lee J, Lee S-U, Oh HJ, Kim H-J, et al. Age-related deterioration of saccule-related neural function is associated with decreased estimated glomerular filtration rate and increased free thyroxine. *Clin Neurophysiol* 2019;130:795–801.
- Labanca L, Starling ALB, de Sousa-Pereira SR, Romanelli LCF, de Freitas Carneiro-Proietti AB, Carvalho LN, et al. Electrophysiological analysis shows dizziness as the first symptom in human T cell lymphotropic virus type-associated myelopathy/tropical spastic paraparesis. *Aids Res Hum Retrovir* 2015;31:649–54.
- Perez R, Ziv E, Freeman S, Sichel JY, Sohmer H. Vestibular end-organ impairment in an animal model of type 2 diabetes mellitus. *Laryngoscope* 2001;111:110–3.
- Ren J, Ma F, Zhou Y, Xu A, Zhang J, Ma R, et al. Hearing impairment in type 2 diabetics and patients with early diabetic nephropathy. *J Diab Complic* 2018;32:575–9.

Eleftherios S. Papathanasiou PhD, FEAN *

Clinical Neurophysiology Laboratory, Clinic B, The Cyprus Institute of Neurology & Genetics, Nicosia, Cyprus

* Address: 6 International Airport Avenue, P.O. Box 23462, Nicosia 1683, Cyprus. Fax: +357 22 358238.

E-mail address: neurophy@cing.ac.cy

Accepted 25 February 2019

Available online 6 March 2019