Background and aim:
Acute unilateral vestibular loss leads to vertigo sensation, nystagmus and postural imbalance. As a rule, a behavioral recovery of the signs and symptoms follows over days to months due to central vestibular compensation (VC). The present study aimed to investigate the determining factors for impaired VC by following a large cohort of patients after acute unilateral vestibulopathy (AUV).

Methods:
745 patients (mean age: 54.2±15.2, 53.4% men) with the clinical picture or history of AUV, who were seen at the Department of Neurology and the German Center for Vertigo and Balance Disorders over the last 10 years, were included in the study. All patients underwent a detailed neuro-ophthalmological and neurological examination, including measures of the subjective visual vertical (SVV), ocular torsion (OT), head impulse test (HIT), provocation nystagmus (PN), visual acuity and postural control. 375 patients were followed along their course of VC (mean number of visits 3.5±1.2, mean time of follow-up 217±625 days). In 235 patients a cranial MRI was available at onset of vestibular failure. The MRI was assessed by two senior neuro-radiologists. Extent of white matter lesions was classified by the ARWMC scale. Patients were indicated as poorly compensated, if SVV deviation (>2.5°), PN and postural asymmetry persisted for longer than 3 months after AUV.

Results:
13.3% of patients were classified as poorly compensated at 3 months (PN: 55%, SVV deviation: 36%, postural asymmetry: 22%), 12.8% at 6 months and 10.6% at 12 months after vestibular failure (Figure 1). These patients had a significantly higher age (mean: 59.8 vs. 52.3 years, p<0.003), poorer visual acuity (p<0.002), higher rate of polyneuropathy (p<0.001) and higher ARWMC scales as compared to patients with regular VC. In the entire group of patients mean time of SVV normalization almost doubled with every 20 years of aging (Figure 2). There was a non-significant tendency of poorer compensation in women. Supratentorial white matter lesions were an independent predictor of VC, whereas lesions in the central vestibular network were less important (Figure 3).

Conclusion:
About 10% of patients have an impeded course of VC after vestibular failure. The most important risk factors are age, visual or somatosensory deficits and supratentorial white matter lesions. On the basis of these data a risk score may be developed to predict the individual course of VC and define the necessity of intensified physical treatment and aftercare.

Literature:

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Vascular encephalopathy impairs behavioral compensation following acute unilateral vestibulopathy
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