Medullary lesion

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Medullary infarction may be divided into lateral and medial. Primary medullary hemorrhage is extremely rare. Lateral medullary infarction (LMI, Wallenberg syndrome) has been a prototype of central vestibular syndrome by mostly involving the vestibular nuclei. In contrast, the vestibular and ocular motor findings began to be recognized rather recently in medial medullary infarction (MMI). In LMI, spontaneous nystagmus is usually horizontal or mixed horizontal-torsional with a small vertical component. Typically, the horizontal nystagmus beats away from the lesion side while it may beat toward the lesion side during ipsilesional gaze. Head shaking nystagmus (HSN) is frequently observed, and the horizontal component of HSN is ipsilesional in most patients. Patients with LMI invariably show ocular ipsipulsion that comprises a steady-state ocular deviation to the lesion side, hypermetric ipsilesional saccades and hypometric contralesional saccades, and oblique ipsilesional misdirection of vertical saccades. LMI cause ipsiversive ocular tilt reaction (OTR) and SVV tilt, and prominent imbalance with falling to the lesion side as if being pulled by a strong external force. Body lateropulsion correlates with subjective visual vertical (SVV) tilt, i.e., the more pronounced the lateropulsion, the greater the SVV tilt. Horizontal nystagmus usually beats ipsilesionally, probably due to involvement of the NPH. Upbeat nystagmus is an occasional finding and may be ascribed to damage to the nucleus of Roller or nucleus intercalates. In caudal medulla, the nucleus of Roller and the caudal subgroup of the paramedian tract cells are involved in processing of vertical eye position through their projections to the cerebellar flocculus and may be the neural substrates for upbeat nystagmus. GEN is common and mostly more intense when looking to the lesion side. Damage to the climbing fibers before decussation may cause ocular contrapulsion. MMI may show contraversive OTR and SVV tilt, which indicates a damage to the graviceptive brainstem pathways from the vestibular nuclei after decussation. Recently, distinct clinical features of dorsal medullary infarction was described.