Impaired bilateral conjugate horizontal saccades can occur from bilateral abducens nuclear lesions,¹ as seen in our patient with multiple sclerosis (Video 1 and Figure). Each abducens nucleus is a horizontal gaze center and innervates the ipsilateral lateral rectus through the abducens fascicle and the contralateral medial rectus via the medial longitudinal fasciculus (Figure). Thus, bilateral lesions of the abducens nuclei cause complete horizontal gaze paresis.

The patient also exhibits ocular dysmetria. Cogan² described ocular dysmetria from lesions of the cerebellum or its immediate connections. This presents with overshoot (less commonly undershoot) movements of the eyes, with fast, small-amplitude corrective saccades of diminishing size until reaching the visual target. The involvement of the middle cerebellar peduncle is the likely cause for this sign in our patient.

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The authors report no disclosures relevant to the manuscript. Go to Neurology.org/N for full disclosures.
Figure Localization of the Lesion Responsible for Complete Horizontal Gaze Palsy

(A) Axial and sagittal MRI brain shows T2-weighted hyperintensity in dorsal pons extending to right middle cerebellar peduncle. (B) Anatomic relationships between the abducens nuclei (VI), middle cerebellar peduncles, oculomotor nuclei (III), and vergence center. Pathways for right conjugate gaze are in green; for left conjugate gaze, in blue. Bilateral VI lesions (shaded area) cause loss of horizontal saccadic, pursuit, and reflex vestibular movements. By contrast, a more ventral lesion of the paramedian pontine reticular formation (PPRF) would impair horizontal saccadic generation without affecting pursuit or vestibular ocular reflex. A more rostral lesion causes internuclear ophthalmoplegia by interrupting the medial longitudinal fasciculus (MLF). The figure was created with Biorender.com. LR = lateral rectus; MR = medial rectus; MVN = medial vestibular nucleus.

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References

Teaching Video NeuroImage: Bilateral Horizontal Gaze Palsies With Vertical Ocular Dysfunction From a Demyelinating Lesion of the Pontine Tegmentum

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