Supranuclear Paralysis of Vertical Gaze
A healthy 36 year-old Lieutenant Commander on the day of admission slept late (a rare experience).

He awoke with vertical double vision and mild impairment of balance tending him to veer to the left and bump into objects walking.

His left hand was “clumsy typing” and he was slow constructing sentences.

He had no headache, vertigo, weakness, sensory symptoms or difficulties with his speech.
Ocular Motility

Global supranuclear vertical gaze palsy, upgaze > downgaze
Vertical oculocephalic movements intact
Normal convergence
Normal Bell’s reflex
Normal horizontal eye movements
Supranuclear Downgaze Palsy
Normal Convergence
Normal Horizontal Saccades Left
Normal Horizontal Saccades Right
Additional Signs

Left ocular tilt reaction
Left hypotropia
Ocular dysmetria
  Right gaze to center hypermetric
  Left gaze to center hypometric
Light-near dissociation of the pupils
Light-near Dissociated Pupils
Figure 1. MRI DWI hyperdensity in the right thalamus extending into the right parasagittal midbrain surrounding the red nucleus
On follow-up two months later he had:

Full vertical gaze with slow saccades up and down

On looking down the eyes lateropulsed to the left or converged

Beats of convergence nystagmus provoked by a fast upward saccade and Persistent somnolence. He has adopted the habit of setting 3 alarm clocks to wake him each morning
Normal Upgaze
Normal Downgaze
Figure 2A. Axial T2 MRI Chronic right thalamic infarct
Figure 2B. Coronal MRI, Chronic right thalamic infarct
Figure 2C. Sagittal MRI Chronic right thalamic infarct
Unilateral Midbrain Infarction Causing Upward and Downward Gaze Palsy

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Figure 3. Position of the patient’s eyes in (a) straight ahead gaze (b) left gaze (c) right gaze (d) attempted upgaze and (e) attempted downgaze.
Figure 4 T2 axial and diffusion MRI of the midbrain. The arrows indicate the ischemic lesion at caudal (left) and rostral (right) midbrain levels.
Figure 5. Histologic cross-section at caudal (left) and rostral (right) midbrain levels showing structures involved in the mediation of vertical gaze. PC, posterior commissure; PUL, pulvinar nucleus of the thalamus; SC, superior colliculus; PG, periaqueductal gray; RN, red nucleus; SN, substantia nigra; 3rd nuc, this cranial nerve nucleus; Int Caps, Internal capsule; riMLF, rostral interstitial nucleus of the medial longitudinal fasciculus; INC, interstitial nucleus of Cajal.
Palsy of upward and downward saccadic, pursuit, and vestibular movements with a unilateral midbrain lesion: Pathophysiologic correlations

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Figure 1. CT with contrast enhancement 15 days after the stroke. Axial sections at the level of the rostral midbrain (A), and diencephalon (B), show discrete right-sided enhancement in the tegmentum extending rostrally into medial thalamus as two bands adjacent to the third ventricle.
Figure 4. Transverse midbrain sections (H-E/LFB × 15 before 54% reduction), at the level of the posterior commissure (A) and the superior colliculus (B), show a wedge-shaped infarct in the tegmentum dorsomedial to the red nucleus. The infarct involved the rostral pole of the interstitial nucleus of Cajal (INC) (B). Section A is at the level of the tractus retroflexus of Meynert (TR); the infarct involved the riMLF, nucleus of Darkschewitsch, and paramedian tegmentum ventral to the posterior commissure (PC), but spared the commissure itself. (C) Extent of infarction, depicted in a right parasagittal plane, was reconstructed from serial transverse sections 100 µm apart. Within the area of infarction (light shading) lies the riMLF (dark shading). Planes of sections A and B are indicated. PC = posterior commissure; MT = mammillothalamic tract; mb = mammillary body; TR = tractus retroflexus of Meynert (habenulopeduncular tract); INC = interstitial nucleus of Cajal; III = oculomotor nerve; rn = red nucleus; sc = superior colliculus; ic = inferior colliculus; MLF = medial longitudinal fasciculus; riMLF = rostral interstitial nucleus of MLF; IIIo = third ventricle; pag = periaqueductal gray.
Figure 5. Schema of excitatory midbrain pathways that mediate vertical gaze. (A) Vertical saccades. The infarct destroyed burst neurons in the right rMLF mediating upward and downward saccades, and axons mediating upward saccades from the left rMLF as they traverse the right tegmentum, after decussation in the posterior commissure. Only burst neurons subserving downward saccades from the left rMLF were spared; these axons descend to innervate the oculomotor and trochlear nuclei. For clarity, descending axon pathways are shown innervating only ipsilateral oculomotor nuclei; there is evidence that innervation is bilateral, although predominantly ipsilateral.11

(B) Vertical pursuit. The infarct involved upward and downward pursuit pathways near the right interstitial nucleus of Cajal. The course of vertical pursuit signals to oculomotor neurons is unknown; they traverse, in part, the brachium conjunctivum (BC) and the medial longitudinal fasciculus (MLF). Only upward pursuit signals have been recorded in the BC. (C) Vertical VOR. The infarct involved both upward and downward "indirect" VOR pathways at the level of the right interstitial nucleus of Cajal, where integration of the eye velocity signal may occur. Direct VOR pathways were spared. Only unilateral vestibular pathways are shown. PC = posterior commissure; rMLF = rostral interstitial nucleus of MLF; INC = interstitial nucleus of Cajal; III = oculomotor nucleus; IIIv = third ventricle; o = inferior oblique subnucleus; s = superior rectus subnucleus; i = inferior rectus subnucleus; m = medial rectus subnucleus; IV = trochlear nucleus; MLF = medial longitudinal fasciculus; BC = brachium conjunctivum; SVN = superior vestibular nucleus; MVN = medial vestibular nucleus.
References


