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# Abnormal Vertical Eye Movements in the Locked-in Syndrome

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As a rule, vertical ocular movements are thought to depend on mesencephalic structures, horizontal gaze on pontine structures. The ocular motility examination of a patient with pontine hemorrhage and no lesion of the mesencephalon showed bilateral palsy of horizontal gaze and a dissociated loss of vertical movements: saccades were slowed, while pursuit managements were unchanged. The findings suggest that the pontine reticular formation is necessary in generation of normal vertical ocular saccades.

> Larmande P, Hénin D, Jan M, Elie A, Gouazé A: Abnormal vertical eye movements in the locked-in syndrome. Ann Neurol 11:100-102, 1982

The locked-in syndrome, identified by Plum and Posner [12], is a rare clinical entity consisting of tetraplegia with facial paralysis and bilateral paresis of horizontal gaze. The only remaining voluntary movements are blinks of the eyelids, which allow contact with the patient, and vertical eye movements, which are usually maintained.

No qualitative study of the vertical eye movements in locked-in syndrome has been published, but these movements are often described as normal [7, 11]. In one of our patients suffering from locked-in syndrome, we could confirm the presence of vertical ocular movements. The clinical and graphic recorded abnormalities of these movements are described.

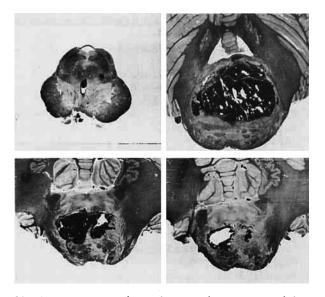
### **Case Report**

A 50-year-old woman with longstanding hypertension was admitted after she suffered a left hemiplegia without loss of consciousness. Immediately following admission her condition deteriorated severely: bilateral hemiplegia developed associated with respiratory paralysis that necessitated tracheal intubation and assisted ventilation. Nevertheless, the patient remained conscious and communicated

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Massive recent pontine hemorrhage. In the upper part of the pons, lesions are mainly located in the tegmentum next to the fourth ventricle. In the lower part of the pons the hemorrhage is less marked. (Celloidin sections, Loyez's myelin impregnation.)

by blink. Both pupils measured 3 mm and reacted correctly. The eyes were parallel; no abnormal spontaneous movements were noticed. In the horizontal plane, the patient was unable to move her eyes: voluntary pursuit and caloric movements were absent. In the vertical plane, eye movements appeared unrestricted. She directed her gaze voluntarily and followed a target. The fast vertical eye movements, however, appeared abnormally slow.

The speed of vertical ocular movements was measured by oculographic recording: silver-silver chloride electrodes were placed in the horizontal and vertical planes and coupled with a DC amplifier. Signals were recorded on a polygraph. The speed was identical upward and downward for a given amplitude: 85 degrees per second for a 20degree amplitude, 135 degrees per second for a 45-degree amplitude, and 140 degrees per second for a 70-degree amplitude. Normal speed according to our laboratory standards is 175, 200, and 250 degrees per second, respectively, for these amplitudes.

Visual pursuit movements were normal: the eyes precisely followed the moving object. Vertical optokinetic nystagmus, both upward and downward, produced a slow movement of the eyes in the direction of rotation of the Barany drum without the opposite quick phase, indicating that the quick phase of vertical optokinetic nystagmus is suppressed. Vertical oculovestibular reflexes were absent both with caloric stimulation and with the "doll's-head" maneuver.

## Neuropathological Observations

Brainstem sections embedded in celloidin and stained with hematoxylin and eosin and Loyez's myelin impregnation displayed a recent and extensive pontine hemorrhage (Figure). The midbrain, in particular the oculomotor nuclei (III and IV), was normal, as were the medial longitudinal fasciculus and the mesencephalic reticular formation (MRF).

In the upper part of the pons, the lesions were mainly located in the tegmentum next to the fourth ventricle, and involved the superior central nucleus, the medial longitudinal fasciculus, the medial lemniscus, and the central tegmental tract. The hemorrhage had spared the ventral part. In the middle pons the hemorrhage had a similar topography, destroying the middle tegmentum next to the fourth ventricle. The ventral pons and middle cerebellar peduncles were free of lesions. In the lower pons the hemorrhage affected only the middle part of the tegmentum, involving the fibers of the root of the abducens nerve (VI). The nucleus was intact. The medulla, cerebellum, and hemispheres showed no lesions. There was moderate arteriosclerosis of the basilar artery without stenosis or thrombosis.

# Discussion

Paralysis of horizontal gaze is a classic feature of pontine lesions, linked here to destruction of the two pontine reticular formations (PRF). The absence of vertical oculocephalic reflexes can be explained by interruption of ascending connections between the vestibular system and oculomotor nuclei within either the medial longitudinal fasciculus or the brachium conjunctivum [6]. Slowing of vertical saccades, however, has not been described with human pontine lesions.

Christoff [5] observed disappearance of upward movements of the eyes with persistence of downward movements in bilateral pontine lesions. However, he made no distinction between voluntary and tracking movements. Bender and Shanzer [1] described the absence in monkeys of upward saccades with paramedial lesions of the pons and of downward saccades with lesions of the mesencephalon. After bilateral lesions of the medial longitudinal fasciculus in monkeys, Evinger et al [6] observed dissociation of vertical movements: the saccades appeared normal but tracking movements were jerky, and the animal had great difficulty maintaining fixation in upward and downward positions. These investigations prove that the PRF influences control of vertical movements. Our study demonstrates that vertical saccades specifically are under its control.

The point is the relationship between the MRF and PRF in control of vertical saccades. Presently, anatomical [13] and physiological [2, 4, 10] studies' indicate that the rostral part of the MRF generates vertical saccades. Numerous cells in the PRF also discharge during vertical saccades. The latter discharge precedes that of the MRF, suggesting that the MRF is controlled by the PRF [8, 9]. In support of this hypothesis, Buttner-Ennever [3] has demonstrated the existence of a direct ascending anatomical pathway between the two structures.

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