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Lateropulsion in Wallenberg's syndrome and contrapulsion in the proximal type of the superior cerebellar artery syndrome

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ABSTRACT. Two patients with leftward bias of the eyes during vertical saccades were studied by electro-oculography. One patient with left Wallenberg's syndrome showed the characteristic eye-movement disorders with leftward overshooting and rightward 'staircase' undershooting during horizontal saccades. The other patient, with the right proximal type of superior cerebellar artery syndrome, showed overshooting dysmetria sequentially diminishing in amplitude to the contralateral side of the cerebellar lesion and ipsilateral hypometric dysmetria. Because these two syndromes are neurologically similar, a careful study of the eye movements may provide a useful clue for the clinical diagnosis. The authors' study showed that these two syndromes have opposite directions of lateropulsion during vertical saccades in relation with the neurological signs, *i.e.*, lateropulsion and contrapulsion respectively, and also a predominance of cerebellar oculomotor disturbances during horizontal saccades in the proximal occlusion of the superior cerebellar artery.

Key words: lateropulsion; contrapulsion; Wallenberg's syndrome; superior cerebellar artery syndrome; ocular dysmetria; magnetic resonance imaging

INTRODUCTION

The clinical manifestations of Wallenberg's syndrome (WS) resemble those of the superior cerebellar artery syndrome (SCAS). In both syndromes, the eye movement is characterized by lateropulsion. The lateropulsion in WS is characterized by the overshooting of saccades to the side of the lesion undershooting with a 'staircase' of saccades away from the side of the lesion, and oblique deflection in vertical saccades to the side of the lesion[1, 2]. Recently, a case of lateropulsion contralateral to the side of the superior cerebellar artery occlusion was reported. The patient had no Horner's syndrome and no sensorial disturbance, and the lesion was thought to have been induced by a distal occlusion of one superior cerebellar artery[3, 4]. On the other hand, there is a proximal type of SCAS. Its neurological features are: ipsilateral cerebellar signs and ipsilateral Horner's syndrome, and contralaterally, there is a loss of pain and temperature sensitivity of the face,

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trunk and extremities[5, 6]. The neurological manifestations are very similar to those of WS, and the clinically differential diagnosis of the two syndromes is difficult.

Two patients had lateropulsion of the eyes and clinically, they were diagnosed as left WS (case 1) and right SCAS, proximal type (case 2). In case 1 lateropulsion was to the ipsilateral side of the lesion and in case 2 lateropulsion was to the contralateral side. Eye movement could be accurately recorded with special attention paid to the horizontal saccadic movement. Emphasis will be placed on the direction of lateropulsion and on the different nature of horizontal saccadic movement for the diagnosis of the two syndromes.

CASE REPORTS

Case 1: A 70-year-old female was admitted to the Department of Neurology after the acute onset of headache and nausea. She had been treated for hypertension for 15 years. She was alert and well oriented. Tendon reflexes were normal but she was hypotonic. The left side coordination was poor. The sensory disturbances (of body and extremities) are not noticed, except for loss of pain and temperature sensitivity on the left side of her face. Two months later, during her hospitalization, sensations of touch and pain were diminished on the right side of her trunk and right limbs, and she was unable to stand unassisted. The eye movements were full, but the eyes were deviated conjugately leftward. She had a skew deviation with left hypotropia. Other abnormal ophthalmological findings were left side Horner's syndrome, rotatory-horizontal gaze nystagmus with directional preponderance to the left and diminished left corneal reflex. When the patient was asked to fixate on a target straight ahead, the eyes deviated conjugately to the left and they returned to the primary position with small corrective movements. Horizontal saccades were hypermetric to the left and hypometric to the right. Horizontal smooth pursuit was saccadic. On attempted vertical saccades, the eyes moved obliquely to the left and made refixation movements back to the target. The magnetic resonance imaging (MRI) showed a high intensity area in the left lateral dorsal medulla (Fig. 1), which corresponds with the neurological findings of Wallenberg's syndrome.

Case 2: A 60-year-old male was admitted to the hospital suffering sudden vertigo, and difficulty in speaking and in standing. In the following days, he

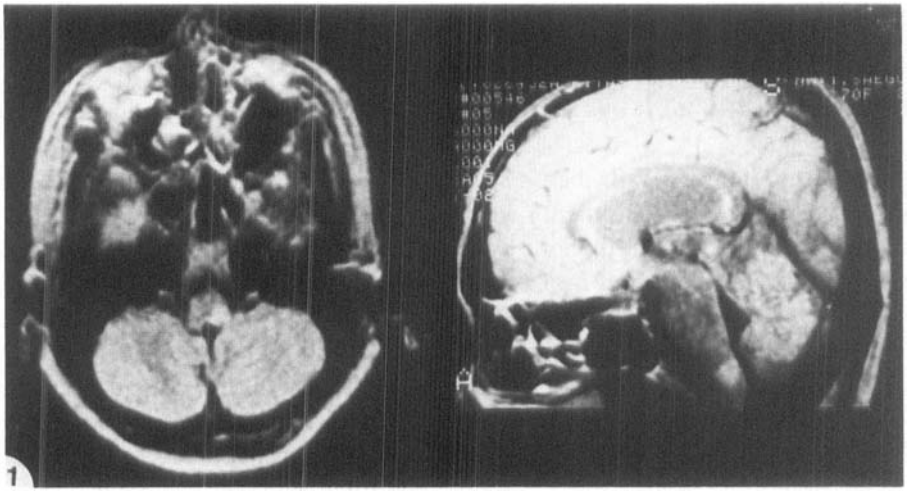

Fig. 1. The magnetic resonance imaging (MRI) of case 1 shows a high intensity area in the left lateral dorsal medulla.

started to vomit and noticed difficulty in taking a cigarette with his right hand. On neurological examination, he was found to have right ataxia with dysmetria and intention tremor. Walking was impossible. All tendon reflexes were normal. Touch and pain hypalgesia on the left side of the body were noticed. Ophthalmological findings were: right Horner's syndrome, large horizontal gaze evoked nystagmus in both directions, and alternating skew deviation. Left corneal reflex was decreased. Pupillary reflexes were normal. Slight left conjugate deviation was noticed. Leftward saccades consistently overshot the target and rightward saccades appeared to produce some hypometric dysmetria. On attempted vertical saccades, the eyes moved obliquely to the left followed by corrective movements to the right, mimicking the lateropulsion seen in the previous case. Smooth pursuit was saccadic in both directions. Magnetic resonance imaging (MRI) showed a high intensity area on the right cerebellar hemisphere, the dorsal pontine region, superior and middle cerebellar peduncles and the territory of the superior cerebellar and anteroinferior cerebellar arteries (Fig. 2). The right vertebral artery angiogram showed marked stenosis and tortuosity of the right vertebral artery, but none was detected in the right superior or anteroinferior cerebellar arteries. The left vertebral artery angiogram was normal. Therefore, the lateropulsion of the eyes was seen on the contralateral side of the lesion.

Electrooculographic analysis of eye movement

Case 1: By EOG recording of horizontal saccades on attempted rightward gaze, there was undershooting of saccades reaching the target by saccades of 5 degrees in amplitude. In contrast, saccades overshot the target to the left and returned to the target in small successive corrective saccades (Fig. 3A). True saccadic dysmetria was not seen. Smooth pursuit was saccadic in both directions, but the amplitude of corrective saccades was greater to the left (Fig. 4A). Vertical saccade recordings showed conjugate oblique deflection to the left. On attempted upward gaze, after an oblique movement to the left the eyes made a slow refixation movement to the right in order to reach the target. Although many artifacts interfered with the vertical eye movement recordings by electrooculogram, these findings were also confirmed in the clinical examination of lateropulsion, especially evident with upward saccades. Jerky movements interfered with fixation. The eyes deviated conjugately to the left, followed by small saccades back to fixation (Fig. 5A). The lateropulsion in vertical saccades was still present without change eight months after the first recording.

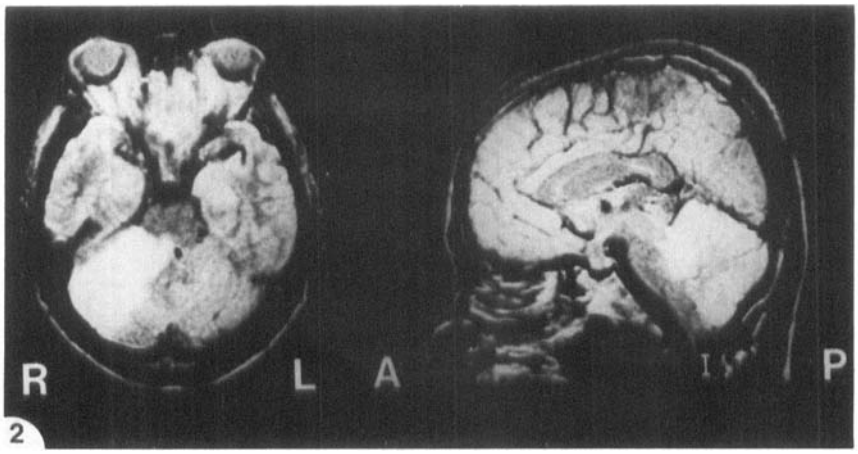

Fig. 2. The magnetic resonance imaging of case 2 shows a high intensity area in the right cerebellar hemisphere, the dorsal pontine region, and the superior and middle cerebellar peduncle.

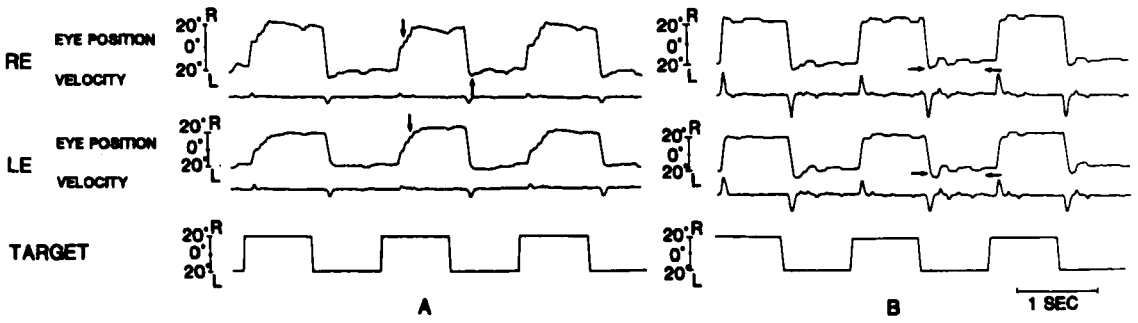

Fig. 3. Horizontal saccades. A. Case 1 shows undershooting, with a staircase pattern of rightward saccades (↓) and overshooting to the left (↑). B. Patient 2 shows saccadic dysmetria: rightward saccades are normometric or hypometric but the leftward saccades consistently presented hypermetric dysmetria (→). (RE: right eye, LE: left eye, R: right, L: left).

Case 2: The electrooculographic study was made during the patient's hospitalization. Horizontal saccades (Fig. 3B) were characterized by hypermetric saccades, followed by two or three corrective movements with diminishing amplitude, *i.e.*, hypermetric saccadic dysmetria contralateral to the lesion. Slight hypometric dysmetria was seen to the side of the lesion. In the beginning of one saccade the initial intersaccadic period averaged 160 msec and in each following corrective saccade, the intervals were greater. Smooth pursuit was not smooth, but mixed with larger amplitude of saccades towards the left, rather than towards the right (Fig. 4B). During vertical saccades, on attempted upward gaze, the eyes showed an oblique deflection to the left, followed by slow corrective movements to the right. During fixation of a target straight ahead (Fig. 5B), the eyes showed square wave jerks with a slow rightward movement followed by leftward saccades. During eccentric lateral gaze, large

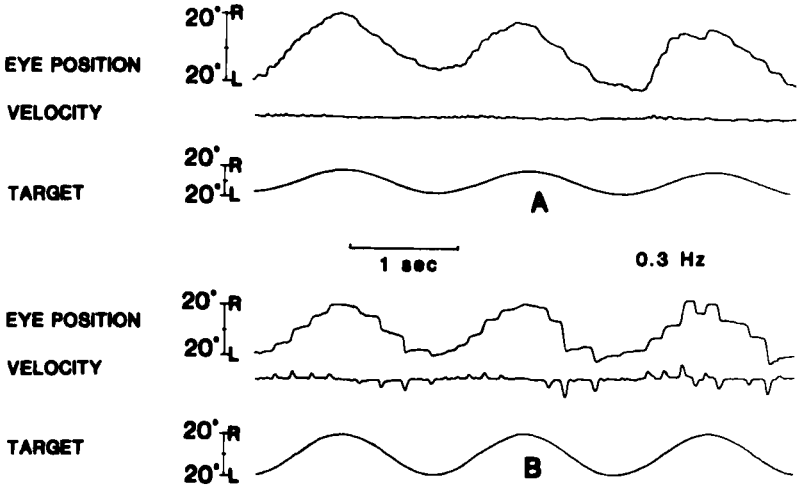

Fig. 4. Horizontal smooth pursuit movement. In both cases (A and B), the eyes made jerky pursuit movements with the largest amplitude of saccades towards the left. (R: right, L: left).

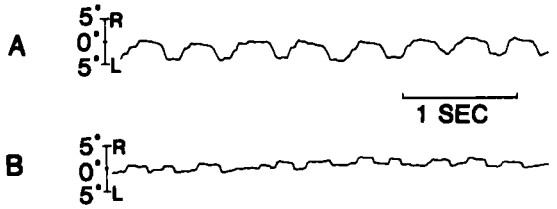

Fig. 5. Fixation on a target straight ahead. A: fixation in case 1 showed jerky movements to the left, followed by corrective small saccades to the center. B: fixation in case 2 was interfered with by square wave jerks. (R: right, L: left).

horizontal dissociated nystagmus was seen in both directions. One year later the lateropulsion in vertical saccades persisted.

DISCUSSION

In this paper we compared the lateropulsion by electrooculography in WS and in proximal type of SCAS[5]. In vertical saccadic movements, the lateropulsion in WS was ipsilateral and in SCAS it was contralateral to the side of the lesion. During horizontal saccades in WS the lateropulsion was present with overshooting to the side of the lesion and staircase undershooting to the opposite side. However, in SCAS the eyes showed hypermetric dysmetria to the contralateral side of the lesion and hypometric dysmetria to the side of the lesion. The neurological manifestations in WS and in SCAS are very similar. Thus, the direction of lateropulsion especially in vertical saccades was an important feature to differentiate both syndromes, as well as the different nature in horizontal saccades. The contralateral lateropulsion seen in horizontal saccades of case 2 is also different from previous reports of distal occlusions of the superior cerebellar artery[3, 4], which showed a mirror image of the lateropulsion of WS. However, during vertical saccades the same oblique deflection was seen in both distal and proximal type of SCAS. The dissociation of lateropulsion during horizontal and vertical saccades in proximal and distal occlusion of the SCAS suggested a different physiopathology of horizontal and vertical lateropulsion.

Lateropulsion of the eyes may be associated with many disorders[2, 7] but vascular lesions in the lateral medulla are the most common cause. The medullary lesion probably involved the vestibular nuclei and its connections with the cerebellum especially the juxtarestiform body[1, 8, 9]. The isolated vestibular nuclei lesion did not show lateropulsion[10]. Damage of the cerebellar-adaptive control of the saccadic system[11] in the lateral medullary infarction may account for the difficulty of recovering from lateropulsion in this syndrome, which took over eight months.

Recently, contralateral lateropulsion has also been described by Ranalli and Sharpe[3] and Benjamin *et al.*[4] in patients with distal occlusion of one superior cerebellar artery. In our patient with a proximal occlusion of the superior cerebellar artery (case 2), contralateral lateropulsion was markedly present during vertical saccadic eye movements, but cerebellar manifestations were more evident than other signs of lateropulsion: horizontal saccades showed the typical dysmetria associated with square wave jerks and horizontal gaze paretic nystagmus. Deeper and extended involvement of the cerebellum in our patient in the territory of the superior cerebellar artery, demonstrated by magnetic resonance imaging, may be the cause of the predominance of cerebellar signs. The damage of efferences from the cerebellum, especially the vermis to the fastigial and interpositus nuclei[12] and to the brainstem nuclei and superior colliculus[13], *i.e.*, cerebello-tectal projections, may be the origin of amplitude dysmetria seen in horizontal saccadic movements[12, 13].

The lateropulsion of the eye in WS and two kinds of contralateral lateropulsion in superior

cerebellar artery occlusion, *i.e.*, distal and proximal type, were demonstrated by comparative electrooculographic studies. The contralateral lateropulsion in vertical saccades associated with horizontal saccadic dysmetria indicated an extended damage of the cerebellum by proximal occlusion of one superior cerebellar artery.

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