SHORT REPORT

Horizontal eye movement disorders after posterior vermis infarctions

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Abstract

The horizontal saccade, smooth pursuit, and vestibulo-ocular reflex gains were recorded in 19 patients with cerebellar infarction documented with MRI, and in a group of control subjects. Bilateral saccade hypometria and a decrease in ipsilateral smooth pursuit gain were found only in patients with a lesion affecting the posterior vermis. These results in humans support experimental findings suggesting that the posterior vermis controls both saccade accuracy and smooth pursuit velocity.

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In the monkey, two cerebellar regions, the flocculus and the posterior vermis, are known to be involved in smooth pursuit. Bilateral ablation of the flocculi and portions of the paraflocculi results in a reduction of smooth pursuit velocity to 65% of normal values, which decreases to 30–40% with the additional ablation of the posterior vermis (lobuli IV to VIII).[1] Neural activity related to smooth pursuit has been recorded in these two structures.[2] Furthermore, the posterior vermis and underlying fastigial nuclei are involved in the control of saccade accuracy, as ablation of these structures in the monkey results in saccade dysmetria.[3 4] Neural activity related to saccades has been recorded in vermal lobuli VI and VII in the monkey.[2] Anatomical data suggest that the oculomotor vermis projects predominantly to the ipsilateral mediocaudal fastigial nucleus.[5]

The ocular motor signs resulting from lesions affecting specific cerebellar regions have not yet been precisely defined in humans. Electro-oculographic studies of patients with cerebellar lesions have often involved degenerative, tumorous, or demyelinating processes, usually not confined to the cerebellum. Electro-oculographic studies of patients with focal and pure cerebellar lesions have rarely been performed.[6 7] Because infarction topography has been described in recent pathological and radiological studies, MRI may be used for the radioanatomical correlation of such lesions.[8 9] Therefore, the purpose of this study was to analyse quantitatively saccade accuracy and smooth pursuit velocity in patients with a recent and pure cerebellar infarction, documented by MRI.

Patients and methods

Nineteen patients (mean (SD) age 56 (15) years) with a cerebellar infarction were studied. Patients with a coexisting brainstem infarction or any clinical sign or radiological evidence of oedematous brainstem compression were not included. The location of the infarction was defined from MRI axial sections and previously published templates based on pathological studies of 64 cerebella.[8 10] Coronal and sagittal sections were also correlated with Courchesne's templates.[11 12] Two patient groups were constituted according to presence and absence of vermal involvement.

GROUP 1

This group comprised nine patients (mean (SD) age 65 (11) years) with an infarction involving the cerebellar vermis (figure). The vascular territory involved was that of the posterior inferior cerebellar artery, on the right side in six cases and on the left side in three (one case associated with an ipsilateral infarct involving the superior cerebellar artery territory). In the cerebellar vermis, the infarctions affected variably lobuli VI to X (the clivus, tuber, pyramis, uvula, and nodulus), corresponding to the posterior vermis. Lobuli VIII and IX were damaged in all cases and lobuli VI and VII in four cases. The inferior part of the ipsilateral cerebellar hemisphere was also damaged, including variably the tonsil, the lobuli biventer, gracilis, semilunaris inferior, and the inferior part of the lobulus semilunaris superior. The flocculus and the dentate nuclei were not affected by any of the infarctions. Brain MRI did not show recognisable fastigial nuclei. Eye movements were recorded on average 35 (SD 42) days after onset of the stroke.

GROUP 2

This group comprised 10 patients (mean (SD) age = 47 (12) years) with a cerebellar infarction sparing the cerebellar vermis (figure). The vascular territory was that of the right posterior inferior cerebellar artery in three cases and that of the superior cerebellar

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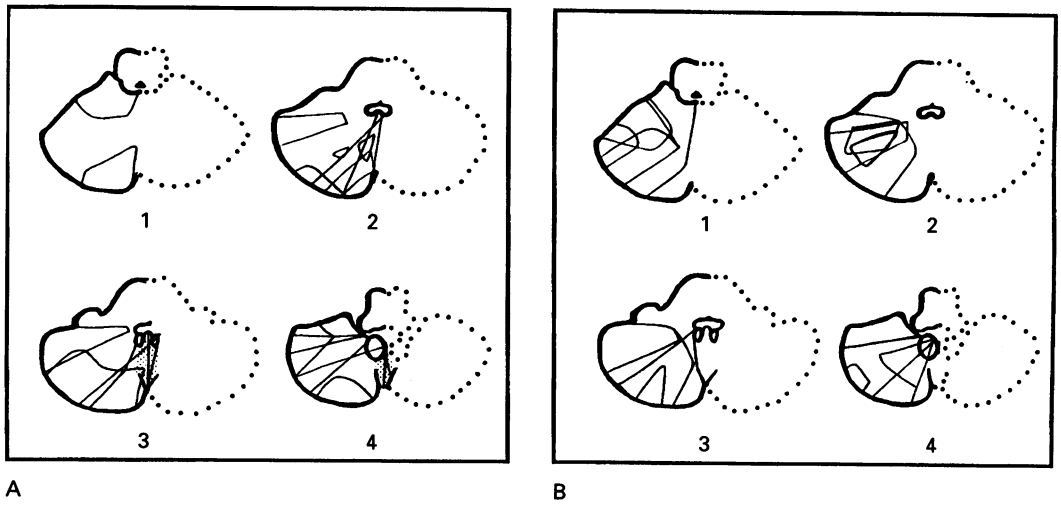

Drawing of the cerebellar areas involved by the infarcts as they appear in MRI on horizontal sections, independent of the infarct side. (Sections from the rostral to the caudal cerebellum: (1) and (2) sections through the upper pons, (3) section through the midpons, (4) section through the upper medulla.) A = group 1, B = group 2, L = left, R = right. The posterior vermis is represented by the hatched area.

artery in seven cases (two on the left side and five on the right side). The infarction area affected variably the tonsil, the lobuli biventer, gracilis, and the semilunaris inferior, superior, simplex, and anterior. The dentate nucleus was damaged in two patients. The flocculus was spared. Eye movements were recorded on average 26 (SD 19) days after onset of the stroke.

CONTROL GROUP

This group included nine normal subjects (mean (SD) age = 57 (8) years).

EYE MOVEMENT RECORDINGS

Eye movements were recorded by direct current electro-oculography in darkness, with the head immobilised. Three types of horizontal eye movements were studied in the patient and control groups.

For reflexive visually guided saccades, the subject was instructed to fixate a central point and then to look as quickly as possible at a lateral target suddenly occurring 25° left or right of the central point with unpredictable timing and direction. The saccadic gain (ratio of primary saccade amplitude to target eccentricity) was determined for four types of saccades: from the central position to the side of the lesion (ipsilateral centrifugal saccade) or the opposite side (contralateral centrifugal saccade), and from the side of the lesion (ipsilateral centripetal saccade) or the opposite side (contralateral centripetal saccade) to the central position. The results were averaged for each type of saccade with 15 consecutive saccades in each subject.

Smooth pursuit was tested by instructing the subject to follow a light emitting diode moving sinusoidally, with an eccentricity of ±20°, peak velocity of 23°/s (frequency = 0·28 Hz), and 45°/s (frequency = 0·37 Hz). The smooth pursuit gain (peak eye velocity over peak target velocity) was determined for each target velocity and each horizontal direction by averaging the gain existing in 10 consecutive cycles.

The vestibulo-ocular reflex (VOR) was tested by rotating the subjects sinusoidally in darkness while fixating an imagined earth-fixed target, with an amplitude of ±20° (frequency = 0·3 Hz, peak velocity = 35°/s). The peak eye velocity over peak rotation velocity (VOR gain) was determined for both horizontal directions by averaging the results of 10 consecutive cycles.

STATISTICAL METHODS

The saccadic, smooth pursuit, and VOR gains in each patient group were compared with those of the control group by analysis of variance, and multiple comparisons were made with the Newman-Keuls procedure. The comparison between the ipsilateral and contralateral values was made by Student's *t* test. The gain for each control subject and each type of eye movement was obtained by averaging the rightward and leftward values, as no significant difference was found between the two types of values in these subjects.

Results

Table 1 gives the mean results. In group 1 the saccadic gain was decreased in both centrifugal directions compared with that of controls

Eye movement results

	Saccade gain (mean (SD))				*Smooth pursuit gain (mean (SD))*				*VOR gain (mean (SD))*	
	Centrifugal		*Centripetal*		*23°/s*		*45°/s*		*35°/s*	
	Ips	*Cont*	*Ips*	*Cont*	*Ips*	*Cont*	*Ips*	*Cont*	*Ips*	*Cont*
Group 1 (n = 9)	0·82**† (0·08)	0·83*† (0·08)	0·89 (0·12)	0·89 (0·10)	0·68*† (0·24)	0·78 (0·19)	0·43**†† (0·24)	0·67 (0·27)	0·83 (0·15)	0·92 (0·27)
Group 2 (n = 10)	0·91 (0·04)	0·93 (0·06)	0·95 (0·04)	0·96 (0·05)	0·93 (0·04)	0·92 (0·08)	0·86 (0·15)	0·83 (0·15)	0·93 (0·19)	0·90 (0·12)
Controls (n = 9)	0·95 (0·03)		0·97 (0·03)		0·95 (0·07)		0·85 (0·06)		0·83 (0·11)	

*p < 0·01; **p < 0·001 *v* controls; †p < 0·01; ††p < 0·00 group 1 *v* 2. Ips = ipsilateral; Cont = contralateral.

($p < 0.001$ for ipsilateral saccades and $p < 0.01$ for contralateral saccades) or to that of group 2 ($p < 0.01$ in both directions). The mean undershooting (17–18%) was similar for both centrifugal directions. Centripetal saccades were only slightly hypometric in group 1. In group 2, the centrifugal and centripetal saccade gains were similar to those of controls.

In group 1, the smooth pursuit gain was decreased, slightly for the contralateral direction but significantly for the ipsilateral direction, compared with the values of the control group ($p < 0.01$ at 23°/s and $p < 0.001$ at 45°/s) or with those of group 2 ($p < 0.01$ at 23°/s and $p < 0.001$ at 45°/s). In group 1, the smooth pursuit gain was variably decreased, explaining the relatively large SDs in this group. It was not possible, however, to correlate this gain to the variable extent of the lesions within the vermis (or in the paravermal structures) because the resolution of MRI is insufficient to quantify the volume of such small lesions. A significant asymmetry between the ipsilateral and contralateral gains existed in group 1 ($p < 0.05$). In group 2, the smooth pursuit gain was similar to that of controls.

For each patient group and each direction, the VOR gain was not significantly different from that of the control group. No abnormal eye movements, such as square wave jerks or nystagmus, were found in either group.

Discussion

Saccade hypometria and a decrease in the smooth pursuit gain existed in group 1 but not in group 2. The main difference between the two groups was damage to the posterior vermis in group 1. Furthermore, in all patients, the infarction was purely cerebellar, without brainstem compression. The flocculus was never damaged. Therefore, eye movement abnormalities observed in group 1 probably resulted from damage to the posterior vermis.

It should be noted that, in the posterior vermis, lobuli VI to IX were variably damaged in group 1, with only lobuli VIII and IX as areas of common damage. In monkeys, lobulus VIII may be involved in smooth pursuit, but lobuli VI and VII seem to be the main lobuli involved in control of eye movement.[2] Even though lobuli VI and VII were not damaged in all the patients of group 1, their afferent tracts from the pontine nuclei or their efferent tracts to the fastigial nuclei[5 13] could, however, have been impaired by the nearby lobulus VIII lesion.

SACCADES

Hypermetria of centripetal saccades has been reported in the monkey after experimental lesions affecting either the posterior vermis and the underlying fastigial nuclei[4] or these nuclei only.[14] Hypermetria has also been reported in patients with midline cerebellar lesions affecting the posterior vermis and probably also the fastigial nuclei.[15 16] Therefore, in the case of midline cerebellar lesions, it may be that hypermetria results mainly from damage to the fastigial nuclei.

Bilateral hypometria of mainly centrifugal saccades was the pattern of dysmetria in our patients with damage to the posterior vermis. In most of them, the strictly unilateral nature of the lesion could not be ascertained with MRI, as the vermis is located on the midline. Furthermore, infarctions affecting this structure often extend slightly beyond the midline.[8] Such hypometria of centrifugal saccades has previously been found in experimental studies in the monkey, after bilateral posterior vermal lesions.[3 17] Although it was not possible in our patients to determine with MRI whether the fastigial nuclei were affected by the infarctions, such damage seems unlikely as: (1) hypermetria was not found in these patients; (2) the vascular territory of the fastigial nuclei is that of the superior cerebellar artery, and not that of the posterior inferior cerebellar artery,[8 9] involved in group 1. Therefore, it may be hypothesised that hypometria of centrifugal saccades in our patients was due to damage to the posterior vermis and not to the fastigial nuclei.

SMOOTH PURSUIT

Because the brainstem, cerebellar peduncles, and flocculus were spared in our patients, the smooth pursuit deficit found in group 1 confirms that the posterior cerebellar vermis is involved in the control of this eye movement in humans.[8] The smooth pursuit gain was decreased bilaterally, but only significantly so for ipsilateral movements. This is consistent with electrophysiological studies in the monkey, in which it has been reported that 60% of the posterior vermis cells show a response during ipsilateral and 40% during contralateral smooth pursuit.[1]

In conclusion, these results confirm that the posterior vermis is involved in eye movement control in humans. Although small vermal infarctions are rare, further human studies with such lesions and other eye movement stimuli are required to determine the specific role of the different vermal lobuli and the other cerebellar structures in this control.

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