Scand J Rehab Med 25: 149–152, 1993

VESTIBULO-OCULAR REFLEX SUPPRESSION FOLLOWING HEMISPHERIC STROKE

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ABSTRACT. This study examined vestibular function following hemispheric ischemic strokes, by testing the suppression of the vestibulo-ocular reflex (VOR). Fifteen patients with cortical or subcortical infarcts several months after a unilateral hemispheric stroke were compared with control subjects. The results indicated impairment of VOR suppression in patients with cortical infarcts. It is suggested that cortical infarcts may induce a mild and symmetrical impairment of vestibular activity which may be responsible for mild and transient imbalance in patients who undergo stroke.

Key words: vestibulo-ocular reflex, stroke, VOR suppression.

Hemispheric stroke may be followed by equilibrium impairment, but imbalance rarely persists for a long period of time if the patient is adequately trained (1). The vestibular system has an important role in maintaining equilibrium and although this system is mainly depending on infratentorial structures, it is modulated by the cerebral hemispheres (7). Vestibular function may be impaired after cerebral lesions such as hemispherectomy (5), hemidecortication (10) and posterior parietal lesions (12). It is not known, however, if vestibular functions are impaired following stroke and if the transient balance impairment is related to vestibular dysfunction. In order to clarify the nature of the vestibular activity and its possible influence on balance following vascular cerebral insults, we examined the integrity of the vestibulo-ocular reflex (VOR) after unilateral hemispheric infarctions.

The VOR normally presents as ocular movements in a direction opposite to that of the turn of the head and enables ocular fixation on target during cephalic movement. It is initiated by stimulation of receptors in the labyrinthine semicircular canals. It must be suppressed when the subject views targets that are moving along with the head. The VOR gain (eye/head displacement) should approach a value of 1 when the VOR is fully activated, and is reduced, in healthy subjects, to values lower than 0.2 during VOR suppression (3, 7). Testing the VOR suppression by means of rotating subjects on a vestibular chair while fixating on a visual target rotating along with them, may be a useful clinical tool for evaluation of vestibular dysfunction. Examination of VOR suppression in patients after hemispherectomy or hemidectortication showed an asymmetric impairment of the VOR gain (5, 10).

In another experiment we found that during VOR activation, the mean VOR gain values were lower in stroke patients than in control subjects, although not with statistical significance. In addition, the patients' VOR gain correlated with equilibrium. To achieve VOR activation, the subjects were rotated in the dark while fixating on a fixed imaginary target. In the present paper, the effect of hemispheric stroke on VOR suppression is presented. The findings demonstrate symmetric impairment of the VOR suppression in patients with cortical infarcts.

PATIENTS AND METHODS

The VOR was studied in three groups: 9 patients with cortical hemispheric infarcts, 6 patients with subcortical hemispheric infarcts, and 7 control subjects. The 15 patients (Table I) were 11 males and 4 females and had a mean age of 57 years (range, 32 to 68 years). They were examined 3 to 17 weeks after a first stroke. All patients were hemiplegic or hemiparetic with significant dysfunction of the upper limb and/or lack of independent gait. They could stand with or without support and were able to cooperate fully during the examinations. Their CT scans showed unilateral single hemispheric infarcts. None of the patients had clinical or radiological evidence of infratentorial brain lesions or a history of vestibular impairment. None of them had hemianopsia and only one patient showed visual hemineglect (Patient N9 in Table I). Two patients (N 1 and N12) used to take mild sedative drugs which were withheld 24 hours before examination. The control subjects (4 males and 3 females) were volunteers with no

Table I. *Patient data*

HPL, Hemiplegia; *HPA*, Hemiparesis; *IC*, internal capsule; *P*, Parietal; *FP*, Fronto-parietal; *TP*, Temporo-parietal; *FPO*, Fronto-parieto-occipital; *C*, cortical; *SC*, Sub-cortical; *ll*, lower limb; *ul*, upper limb; *Rt*, right; *Lt*, left
>, indicates in which limbs the motor deficit was higher or lower; =, indicates that the motor deficit was the same in upper and lower limb.

Patient no.	Age/Sex	Infarct location (CT)			Infarct maximal diameter (mm)	Neurological findings		
						Motor deficit	Sensory Light touch	Position
1	66/M	Rt	IC	SC	20	HPA ul > ll	Impaired	Normal
2	52/M	Rt	IC	SC	17	HPA ul > ll	Normal	Normal
3	53/M	Lt	IC	SC	10	HPA ul = ll	Normal	Normal
4	62/M	Rt	IC	SC	10	HPA ul > ll	Normal	Impaired
5	42/F	Lt	P	SC	26	HPA ul > ll	Normal	Normal
6	68/M	Rt	IC	SC	18	HPA ul > ll	Impaired	Impaired
7	56/F	Rt	FP	C	59	HPA ul = ll	Impaired	Impaired
8	51/M	Rt	TP	C	59	HPA ul > ll	Impaired	Impaired
9	64/M	Rt	TP	C	67	HPL	Impaired	Impaired
10	66/F	Lt	TP	C	41	HPA ul > ll	Normal	Normal
11	66/M	Rt	FP	C	76*	HPA ul > ll	Normal	Normal
12	50/F	Rt	P	C	48	HPA ul > ll	Impaired	Impaired
13	67/M	Lt	FPO	C	103*	HPL	Impaired	Impaired
14	32/M	Lt	TP	C	**	HPA ul > ll	Normal	Normal
15	58/M	Rt	FP	C	68*	HPA ul < ll	Normal	Impaired

* A long but narrow infarct (the volume is not very large).
** Missing data.

history of neurological or vestibular impairment. Their mean age was 58 years (range, 48 to 72 years). All subjects had full range of ocular movements and could identify a visual target easily through a range of 40° from the center. Examinations were performed in a darkened room after at least a five minute dark-adaptation and training period. The subjects were seated in a vestibular chair with the head fixed to the chair and rotated in a sinusoidal rhythm. They were requested to fixate on a visible laser beam target which rotated along with them. Instructions were repeated over and over to ensure subjects' alertness and concentration. The rotations had an amplitude of 30° and a frequency of 0.2, 0.3 or 0.5 Hz.

The horizontal ocular movements were measured by electro-oculography (accuracy about one degree) (4). One eye was examined in each subject: the eye on the paralyzed side in the patients, and the right eye in the control subjects. Three surface Ag/AgCl electrodes (Sensormedics) were used, one on the inner canthus, one on the outer canthus and a reference electrode above the brow.

The amplified eye movements signals, and the target position and chair position signals were recorded on an FM tape (Model 30 ×, Teac). The total system and tape recorder bandwidth was higher than 300 Hz. The data were analyzed off-line using a home-made software package (11). Occasional rapid movements resembling saccades which were superimposed on the VOR response signal, were filtered at 80 Hz and subtracted to isolate the VOR. The rapid components-subtracted VOR signals were then further filtered at 50 Hz. Ten cycles of vestibular stimuli and ocular responses were recorded at each frequency. The results of the first one or two cycles at the beginning of each experiment were excluded to ensure that the chair had reached a sinusoidal rotation.

Statistical significance was calculated by analysis of variance (F test).

RESULTS

Examples of the suppressed VOR signals of subjects representing the three groups are illustrated in Fig. 1. The VOR signal of the control subject (Fig. 1A) has a very low amplitude and almost no gain. An occasional rapid eye movement is superimposed on it. The VOR signal of the patient with a cortical infarct (Fig. 1B) has an abnormally high amplitude and many superimposed rapid movements. The VOR signal of the patient with a subcortical infarct (Fig. 1C) has a slightly higher gain than that of the control subjects although it is still much lower than that in the patient with a cortical infarct.

The mean VOR suppression gain values in the control subjects (0.13–0.19; SD = 0.04–0.13) were similar to those reported in the literature for healthy subjects (2, 3). The mean values for the suppression gain in the control group, the group with cortical infarcts (0.26–0.35; SD = 0.09–0.23) and the group with subcortical infarcts (0.13–0.23; SD = 0.04–0.13) are detailed in Fig. 2A, 2B. The VOR suppression gain

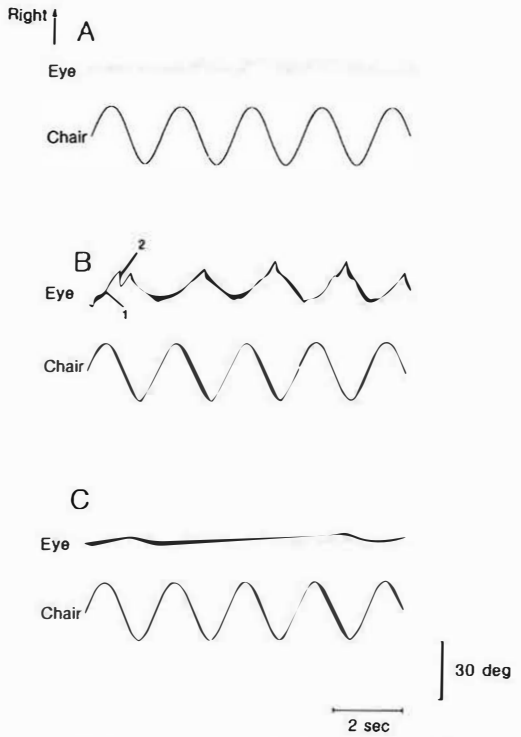

Fig. 1. Ocular response to chair rotation during VOR suppression. (A) In a healthy subject. (B) In a patient with right hemispheric cortical infarct. *Arrows*: 1, 2—superimposed rapid eye movements. (C) In a patient with right hemispheric subcortical infarct.

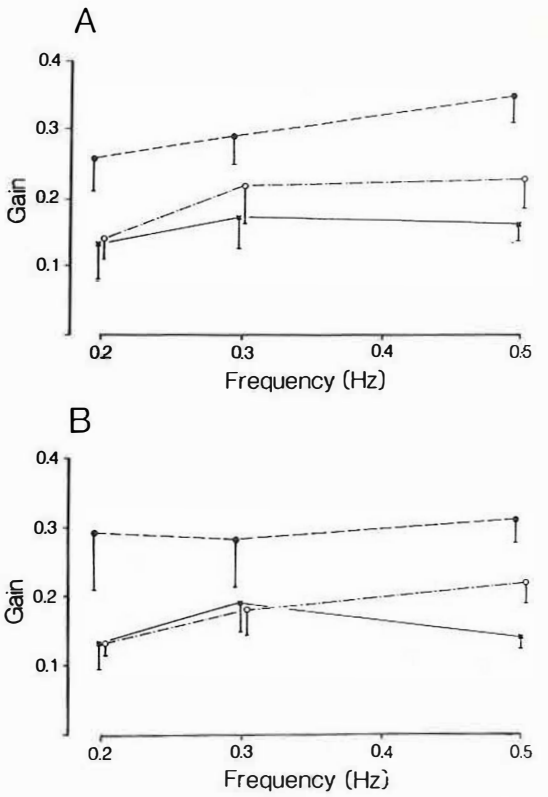

Fig. 2. Mean VOR suppression gain in the control group (×), in the group with cortical infarcts (●), and in the group with subcortical infarcts (○). The vertical line represents the standard error of the mean. (A) Ipsilateral VOR (rightward in the control group). (B) Contralateral VOR (leftward in the control group).

was affected by the frequency of rotation and by the subject group ($p < 0.05$). The gain at the lowest frequency (0.2 Hz), was significantly lower than the gain at 0.3 Hz and 0.5 Hz ($p < 0.02$). The mean gain in the group with cortical infarcts (0.26–0.35) was significantly higher than that in the control group (0.13–0.19) ($p < 0.025$), whereas the gain in the group with subcortical infarcts was not significantly different from that in the control group ($p > 0.6$). As shown in Fig. 2A, 2B the VOR gain was independent of the direction of the horizontal eye movement; it was similar with ocular responses towards the damaged hemisphere (ipsilateral) or towards the intact hemisphere (contralateral) ($p > 0.6$).

DISCUSSION

This study shows for the first time impairment of the VOR suppression in a group of patients with cortical infarcts. The results demonstrate decreased suppression of both ipsilateral and contralateral VOR in this group. When these patients attempted to fixate on a target rotating along with the head, their mean VOR suppression gain was significantly higher that that of the control group ($p < 0.025$) (Fig. 2).

Our finding of an abnormal VOR is consistent with previous results in patients and monkeys with iatrogenic unilateral hemispheric damage (5, 10, 12). The present study is also in agreement with previous publications with respect to the correlation found between the VOR gain and stimulus frequency (2, 3). In earlier studies, however, VOR suppression was impaired with contralateral eye movements (gain 0.5–0.6), but intact with ipsilateral eye movements (gain 0.1–0.14) (5, 10). In these studies, the contralateral VOR gain was higher than in our patients with cortical infarcts, and the ipsilateral was lower. This inconsistency might stem from differences in lesion size. Only

extensive lesions, such as hemidecortication or hemispherectomy, caused a persistently asymmetric VOR (5, 10, 12), while the cortical lesions in our patients, although severe enough to induce a VOR impairment, were limited. The partial cortical sparing may be the reason for the relatively small decrease in contralateral VOR suppression. Some spared cortical areas might also have a role in modulating the ipsilateral VOR, enabling symmety of VOR.

Previous studies have shown that reduced alertness (that may be expected after stroke) may impair VOR suppression, and that the act of choosing an imaginary fixation point may per se suppress the VOR (2, 8). Therefore, reduced ability of concentrating upon the fixation point (may be due to slightly reduced alertness) should also be considered as a possible mechanism behind the effect of cortical lesions on VOR.

The relative integrity of the VOR in the group with subcortical infarcts probably expresses the smaller size of the infarcts in this group and emphasizes the importance of the cortex in VOR modulation.

In conclusion, the present study demonstrates a mild and symmetrical reduction in VOR suppression in stroke patients with cortical infarcts. This finding suggests that a hemispheric vascular lesion may cause a mild and symmetrical impairment of vestibular activity. Such an impairment may be responsible for mild and transient imbalance following hemispheric stroke.

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