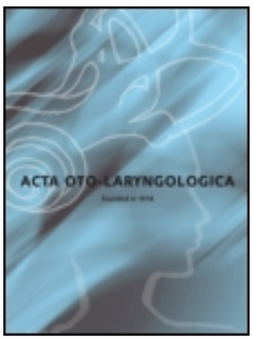

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Videonystagmoscopy: Its Use in the Clinical Vestibular Laboratory

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Vestibular function of a population including labyrinthine-defective patients and a control group of age-matched normal healthy volunteers was evaluated using videonystagmoscopy. This device is made of one or two CCD infra-red cameras mounted on diving glasses and allows observation of ocular movements on a video monitor and/or recording on a videotape. Eye movements are observed after rotations in a Bárány chair and during passive head tilts. With this simple and non-invasive test, a screening of vestibular function at bedside or during ENT clinical investigations can be performed. A further study with videonystagmography to quantify these results being prepared. *Key words: videonystagmoscopy, rotations, torsional nystagmus, cervico-ocular reflex.*

INTRODUCTION

Videonystagmoscopy is a very attractive tool allowing observation of the eyes during all movements of the head. This device allows complete vestibular screening of patients with balance disorders during ENT clinical examination or at bedside.

This device operates in complete darkness thus suppressing all visual motion cues, which enhances the responses of the vestibular system to a specific stimulation in a labyrinthine-defective patient.

MATERIAL AND METHODS

Patients

The population sample of 100 labyrinthine-defective patients participating in the tests included: 20 patients post vestibular neurectomy, 20 patients post surgery for acoustic neuroma, 50 patients with vestibular neuronitis (sudden vestibular loss) and 10 bilateral caloric areflexic patients after gentamycine treatment. These patients complained of balance disorders including true vertigo and/or unsteadiness. Most of them were seen at the acute stage of their vestibular lesion and they were followed up until they recovered and/or had attained vestibular compensation. All these patients were treated with vestibular rehabilitation.

These patients were also submitted to a complete neuro-otological assessment including: audiometry, impedance-audiometry, caloric test, electro-oculographic recordings of smooth pursuit and saccades, dynamic posturography (Equitest). Cerebral imaging (CT-scan and/or MRI) was performed when brain and/or hindbrain lesions were suspected.

A control group of 50 age-matched healthy volunteers was also tested with videonystagmoscopy.

Videonystamoscopy device

The device is made of one or two infra-red CCD camera mounted on a frame similar to diving glasses (Fig. 1). The clinician looks at the eye movements on a video monitor (Fig. 2) and can record them on a videotape. A third infra-red camera can be used to record the head displacement.

Procedure

The patients and the subjects sat in a Bárány chair. A protocol was created to assess peripheral vestibular function. This procedure was carried out as follows: *i)* Search of spontaneous nystagmus and gaze nystagmus. In the absence of visual cues, when the patient was asked to look in a specific direction, nystagmic eye movements could be observed (20 s has to pass before it could be certified that this nystagmus was pathologic). *ii)* Head-shaking nystagmus (HS). This test was performed by having the patient's head shaken in the horizontal plane as fast as possible for approximately 10 cycles (1). The eyes of the patient were closed during the test. HS should not provoke

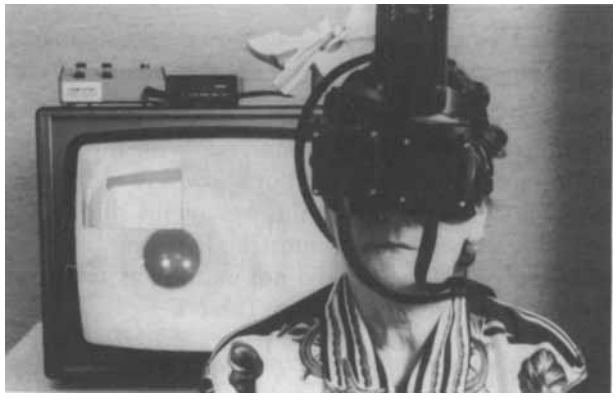

Fig. 1.

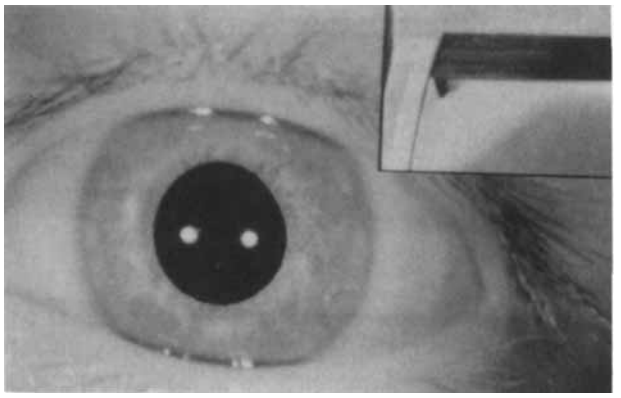

Fig. 2.

any nystagmus. When a nystagmus was observed, the lesion side was indicated by the direction of the slow phase (2). *iii)* Passive head tilt (45°) in the frontal plane (roll) at a velocity of 30°/s. In normal subjects, tilting the head toward the right shoulder causes the eyes to make a torsional nystagmus to the right. When the upper pole of the eye was rolling to the subject's left, the direction of the torsional nystagmus was counterclockwise. *iv)* Rotations of 180° provided by a Bárány chair manually driven during 5 s in one direction, with observation of the induced nystagmus during and after the rotations. After the rotations, when a nystagmus was noted, the clinician waited until it stopped. Then, a whole body rotation was done with the same protocol in the opposite direction.

These rotations were performed with the subject's head in standard position and with the head tilted on the shoulder (45°) to evaluate the responses of the semi-circular canals. *v)* Body rotation with respect to the head to assess the cervico-ocular reflex. This test was done when all the rotational tests previously described did not provoke any responses.

The results of the videonystagmoscopy protocol were compared with those of the classical otoneurological tests.

RESULTS

Normal subjects

Torsional nystagmus was seen during head tilt in both directions and was symmetric. With this stimulation, normal subjects did not show any nystagmus after the rotations, while the nystagmus during the rotations seemed to be symmetric. Rotating the body with respect to the head did not induce any response. These results are displayed in Table I.

Unilateral labyrinthine-defective patients

Patients compensated (Table II). These patients were said to be compensated because they did not complain anymore of balance disorders and demonstrated neither head-shaking nystagmus nor spontaneous nystagmus. A complete unilateral vestibular areflexia was seen at the caloric test. The electro-oculography (EOG) and the dynamic postugraphy of these patients were normal.

Table I. *Normal Subjects*

Spontaneous nystagmus: 0 Gaze nystagmus: 0 Head Shaking nystagmus: 0

	Left side	Right side
Torsional nystagmus	+	+
Nystagmus following the 180° rotation (head in standard position)	0	0
Nystagmus following the 180° rotation (head tilted on the shoulder)	0	0

+: presence; 0: absence.

Table II. *Unilateral labyrinthine-defective, compensated patients*

Spontaneous nystagmus: 0 Gaze nystagmus: 0 Head Shaking nystagmus: 0

	Intact side	Affected side
Torsional nystagmus	+	0
Nystagmus following the 180° rotation (head in standard position)	0	+
Nystagmus following the 180° rotation (head tilted on the shoulder)	+	0

+: presence; 0: absence.

Torsional nystagmus induced by head tilt was asymmetric in most of the cases with a decrease of the torsional nystagmus induced by tilting the head to the side of the lesion.

After the rotations toward the affected side with the head in standard position, a nystagmus was observed while there was no nystagmus after the rotations toward the intact side. After the rotations toward the intact side with the head tilted on the shoulder, the patients demonstrated a nystagmus, whereas no response was observed after the rotations toward the affected side.

Patients seen at the acute stage (Table III). The same population had been seen a few days (2 to 7) after surgery or, for the 50 vestibular neuronitiscares, a few days (2 to 4) after their acute vertigo.

Table III. *Unilateral labyrinthine-defective patients at the acute stage*

Spontaneous nystagmus: + Gaze nystagmus: 0 Head shaking nystagmus: +

	Intact side	Affected side
Torsional nystagmus	+	0
Nystagmus following the 180° rotation (head in standard position)	0	+
Nystagmus following the 180° rotation (head tilted on the shoulder)	+	0

+: presence; 0: absence.

Table IV. *Bilateral labyrinthine-defective patients*

Spontaneous nystagmus: 0 Gaze nystagmus: 0 Head shaking nystagmus: 0

	Left side	Right side
Torsional nystagmus	0	0
Nystagmus following the 180° rotation (head in standard position)	0	0
Nystagmus following the 180° rotation (head tilted on the shoulder)	0	0
Nystagmus following body rotation with respect to the head	+	+

+: presence; 0: absence.

These patients still had a spontaneous nystagmus beating opposite to the lesion. The caloric test demonstrated a non-compensated unilateral vestibular areflexia. EOG was normal. Dynamic posturography showed equilibrium scores reduced to 0 in conditions 5 and 6 (3).

The patients did not demonstrate any torsional nystagmus when tilted toward the affected side while the torsional nystagmus induced by tilting the head toward the intact side was normal or increased. In the few days following the vestibular loss, the spontaneous nystagmus was surperimposed onto the post-rotatory responses in both directions.

Bilateral labyrinthine-defective patients. These 10 bilateral caloric areflexic patients complained of oscillopsia and had a story of previous aminoglycoside treatment. They did not demonstrate any response to the caloric test, but EOG was normal. Dynamic posturography showed equilibrium scores in conditions 5 and 6 reduced to 0. They demonstrated neither torsional nystagmus nor nystagmus after the rotations. Rotating the body with respect to the trunk induced a nystagmus mimicking the vestibulo-ocular reflex (Table IV).

DISCUSSION

With our procedure, normal subjects did not have any nystagmus after rotations. All the unilateral labyrinthine-defective patients, even while compensated according to classical neuro-otological tests, demonstrated a nystagmus after rotations toward the affected side when the head was in standard position, and after rotations toward the intact side when the head was tilted on the shoulder.

Slow phase of torsional nystagmus or ocular counter-rolling (OCR) is torsion of the eyes and is evoked in the direction opposite to that of the head, even in darkness (4). Static OCR is considered to be caused by the utriculus, but it cannot be studied with videonystagmoscopy. The dynamic component of counterroll is said to involve stimulation of the otolith organs, vertical canals (because of the angular acceleration of the movement), and neck receptors.

Conflicting results have been reported following a unilateral neurectomy; it appears that there is no clear relationship between the side of the neurectomy and OCR (5, 6, 7). In some cases of our population, an absence of torsional nystagmus during passive head tilt was noted while these patients behave as normal subjects according to their responses after the rotations. Soon after a vestibular neurectomy, patients still demonstrate a spontaneous nystagmus beating toward the intact side. When the head of the patient is tilted to the intact side, the spontaneous nystagmus is superimposed onto the torsional induced nystagmus. After section of the vestibular nerve, torsional nystagmus induced by head tilt was not observed when the head of the patient was tilted to the side of the lesion.

CONCLUSION

Videonystagmoscopy allows a screening of vestibular function at bedside. In any condition, the presence of a nystagmus following rotations is abnormal with this procedure. As far as asymmetry of post rotatory responses is concerned, any degree of asymmetry reveals a vestibular deficit. These are preliminary results and further study using videonystagmography is ongoing to measure this behavioural asymmetry.

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