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THREE-DIMENSIONAL ANALYSIS OF SPONTANEOUS NYSTAGMUS IN PERIPHERAL VESTIBULAR LESIONS

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The direction of spontaneous nystagmus was recorded in three dimensions with scleral dual search coils in three patients after vestibular neurectomy and in seven patients with vestibular neuritis. The rotation vectors of the spontaneous nystagmus clustered along the sensitivity vector of the lateral semicircular canal (SCC). The direction of the spontaneous nystagmus after resection of the whole eighth nerve was not different from that after resection of only the superior branch of the vestibular nerve. Deviations from this direction were observed only after resection of the inferior vestibular nerve and in one patient with vestibular neuritis. The absence of nystagmus components in direction of the vertical SCC reflects an anisotropy of oculomotor efferents of the vestibulo-ocular reflex arc rather than a lesion limited to the lateral SCC afferents. Therefore, the three-dimensional analysis of spontaneous nystagmus does not permit accurate localization of a peripheral vestibular lesion.

KEY WORDS — eye movements, search coil, semicircular canals, vestibular neurectomy, vestibular neuritis.

INTRODUCTION

The three pairs of semicircular canals (SCCs) are positioned in three nearly perpendicular planes in the head and act as angular accelerometers. They provide the sensory input for the generation of the vestibulo-ocular reflex (VOR), which stabilizes gaze during angular head movements by rotating the eyes oppositely to the head movements in all directions. A sudden unilateral lesion of the peripheral vestibular system not only eliminates one counterpart of each pair of the vestibular accelerometers, but also abolishes the tonic neuronal discharge (resting activity) in the nerve on that side. The resulting imbalance between the tonic input of the two ears leads to the generation of reflexive eye movements (spontaneous nystagmus). Acute peripheral vestibulopathy ("vestibular neuritis") is the prototype of a sudden unilateral peripheral lesion. The cause of this common disorder is still unclear, but there is good evidence that different parts of the labyrinth may be involved to a variable extent in different patients. The clinical parameters spontaneous nystagmus, caloric canal paresis (reflecting mainly the function of the lateral SCC), and deviation of the subjective visual vertical (a parameter of mainly otolithic function) may be dissociated.[1]

Electrical stimulations of single SCC nerves in animals have shown that the SCC afferents induce eye movements roughly in the corresponding anatomic plane.[2] From this, one would conclude that lesions of single SCCs induce spontaneous nystagmus also in the corresponding canal plane. Clinically, however, spontaneous nystagmus is usually analyzed only in its horizontal (and vertical) component, because of technical limitations. Recent developments[3] permit accurate analysis of the eye movements in three dimensions, ie, in all degrees of freedom, also in human patients. The aim of this study was to investigate whether the recording of the spontaneous nystagmus in three dimensions permits a more accurate localization of lesions of the vestibular end organ.

MATERIAL AND METHODS

Patients. Three-dimensional (3-D) spontaneous nystagmus was recorded in three patients after vestibular neurectomy (patients VN1 through VN3) and in seven patients suffering from acute peripheral vestibulopathy (patients APV1 through APV7). Surgery was performed for relief from incapacitating vertigo due to Meniere's disease. (We thank Prof U. Fisch for permission to perform these measurements in patients operated on by him.) In patient VN1, a translabyrinthine cochleovestibular neurectomy was performed. The internal auditory canal was reached by removing the SCCs. The intrameatal segment of the vestibular nerve, including Scarpa's ganglion, and the most peripheral part of the cochlear nerve were resected.[4] A selective supralabyrinthine vestibular neurectomy was performed in patient VN2, who had only a moderate hearing loss of the affected ear.

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CLINICAL FINDINGS AND COMPONENTS OF NYSTAGMUS IN DESCRIBED PATIENTS

Patient	Sex	Age (y)	Side	Caloric Canal Paresis (%)*	Time of Nystagmus Recording (d)†	Spontaneous Nystagmus (°/s) x	y	z
Vestibular neurectomies								
VN1	M	40	R	43	5	4.3	1.9	−10.8
VN2	F	34	R	69	2	3.6	2.7	−11.1
VN3	M	62	R	100	4	2.9	−1.0	−3.2
Acute peripheral vestibulopathies								
APV1	M	57	R	43	2	5.3	2.2	−15.9
APV2	F	35	R	60	5	2.9	0.6	−18.7
APV3	M	67	L	100	6	−4.3	1.8	9.3
APV4	M	49	R	100	10	3.4	2.2	−17.0
APV5	M	73	L	100	10	−0.3	0.3	4.1
APV6	M	63	L	100	10	−0.1	1.7	3.8
APV7	M	45	L	100	11	−2.0	6.6	9.9

Patient VN1 had translabyrinthine cochleovestibular neurectomy, patient VN2 had supralabyrinthine vestibular neurectomy, and patient VN3 had translabyrinthine cochleovestibular neurectomy 16 years after homolateral supralabyrinthine vestibular neurectomy.

*In VN patients, before surgery; in APV patients, after onset of disease.

†In VN patients, days after surgery; in APV patients, days after onset of disease.

In this patient, the superior vestibular nerve was completely resected, including Scarpa's ganglion. Due to the very narrow internal auditory canal, only the posterior part of the inferior vestibular nerve was included in the resection in order to avoid disturbing the blood supply of the cochlear nerve. It is reasonable to expect that the saccular nerve fibers and some of the posterior SCC nerve fibers were, therefore, spared in this patient. Patient VN3 was submitted to a supralabyrinthine vestibular neurectomy (as in patient VN2) 16 years previously. He was completely relieved from vertigo, but still suffered from an incapacitating tinnitus. Therefore, the cochlear nerve was resected through a translabyrinthine approach as in patient VN1. Intraoperatively, a few residual fibers of the singular nerve (innervating the posterior SCC crista) and half the normal numbers of fibers of the ramus sacculi, but no residual fibers of the superior vestibular nerve, were found and removed. The preoperative clinical findings and time of postoperative eye movement recordings are shown in the Table.

Patients APV1 to APV7 experienced a sudden onset of vertigo with spontaneous nystagmus and unilaterally impaired caloric reaction (see Table). Exclusion of a specific ear disease or other neurologic deficits suggested the diagnosis of an acute peripheral vestibulopathy ("vestibular neuritis").

Three-Dimensional Recording of Eye Movements. The eye movements were recorded in three dimensions with dual search coils (manufactured by Skalar Instruments, Delft, the Netherlands), which were mounted in a soft contact ring placed on the locally anesthetized right sclera. The patients were sitting upright. The head rested on a chin bar, and was fixated by frontal and occipital blocks inside a coil frame (70 cm diameter) with earth-horizontal and earth-vertical magnetic fields. The measured right eye was exactly in the center of the frame. The angle between the stereotaxic horizontal plane of the head (defined by the outer acoustic meatus and the lower orbital rim) and the earth-horizontal plane was determined in order to translate the recorded eye movements from the earth-fixed recording coordinate system into head stereotaxic directions. Analog raw coil signals were obtained by a Skalar Eye Position Meter Type 3000, digitized with 833 Hz, and analyzed offline.

Calibration. The dual search coils were first calibrated "in vitro" on a three-axis gimbal system inside the magnetic frame. Measuring the induced voltages in both coils at well-defined 3-D gimbal positions allowed us to determine the angle between the two coils and their sensitivity vectors. "In vivo" calibrations were performed before each trial by asking the patients to fixate on small dots that were presented on a tangent screen (distance 164 cm) in the center and 20° in the horizontal and vertical directions with respect to the right eye. Measuring the induced search coil voltages at these positions allowed us to compute the instantaneous 3-D orientation of the coils — and of the eye — during subsequent periods of spontaneous eye movements. The precise algorithm of the procedure is described elsewhere.[5]

Experimental Procedure. After calibration, the patients were asked to fixate on the center dot on the tangent screen. Then the lights were turned off, and the eye movements were recorded for 10 seconds in complete darkness. During this time the patients were verbally encouraged to keep the current eye position. This procedure was repeated several times for central

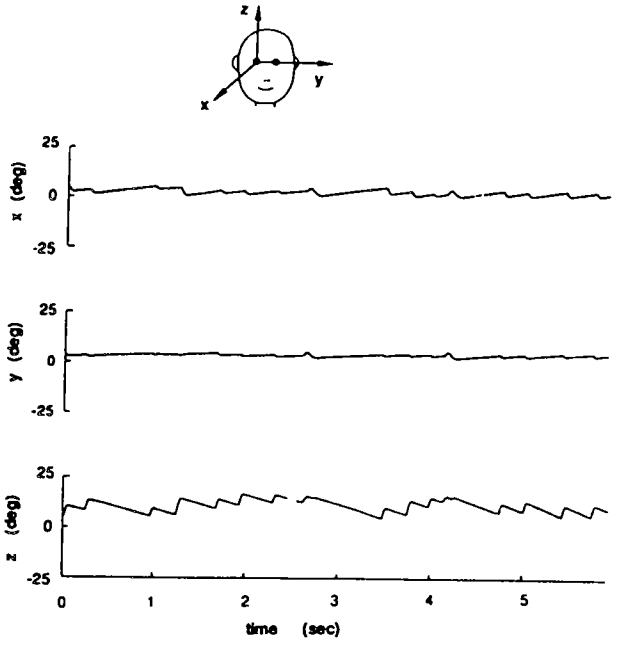

Fig 1. Sample of 6-second period of spontaneous nystagmus (eyes open in darkness, patient VN1). Eye movement components along three orthogonal axes are shown separately. x —rotation axis for torsional movements, y — vertical movements, z — horizontal movements. For signs of direction, see text.

gaze, as well as for gaze positions 20° to the right and 20° to the left, respectively.

Data Analysis and Presentation. For each gaze direction, the spontaneous nystagmus was analyzed from representative recording intervals of 6 seconds' duration. First, data points were converted into rotation vectors[6] and rotated from frame coordinates into stereotaxic head coordinates (Fig 1). Then, angular velocity vectors were computed.[7] An angular velocity vector is oriented parallel to the axis about which the eye bulb rotates in reference to a 3-D orthogonal head-fixed coordinate system, in which the torsional axis is parallel to the stereotaxic horizontal plane. Velocities are given in degrees per second. Horizontal eye movements are presented as vectors along the vertical (head) z-axis, vertical eye movements as vectors along the interaural y-axis, and torsional eye movements as vectors along the sagittal x-axis (see scheme at top of Fig 1). According to the right-hand rule, leftward and downward rotations are positive. Extorsional rotations of the upper pole of the right eye and intorsional rotations of the left eye correspond to a positive torsional vector. Figure 2 shows the instantaneous angular velocity rotation vectors during this 6-second period in the sagittal (zx) and the frontal (zy) head planes. From these data, the vectors at each

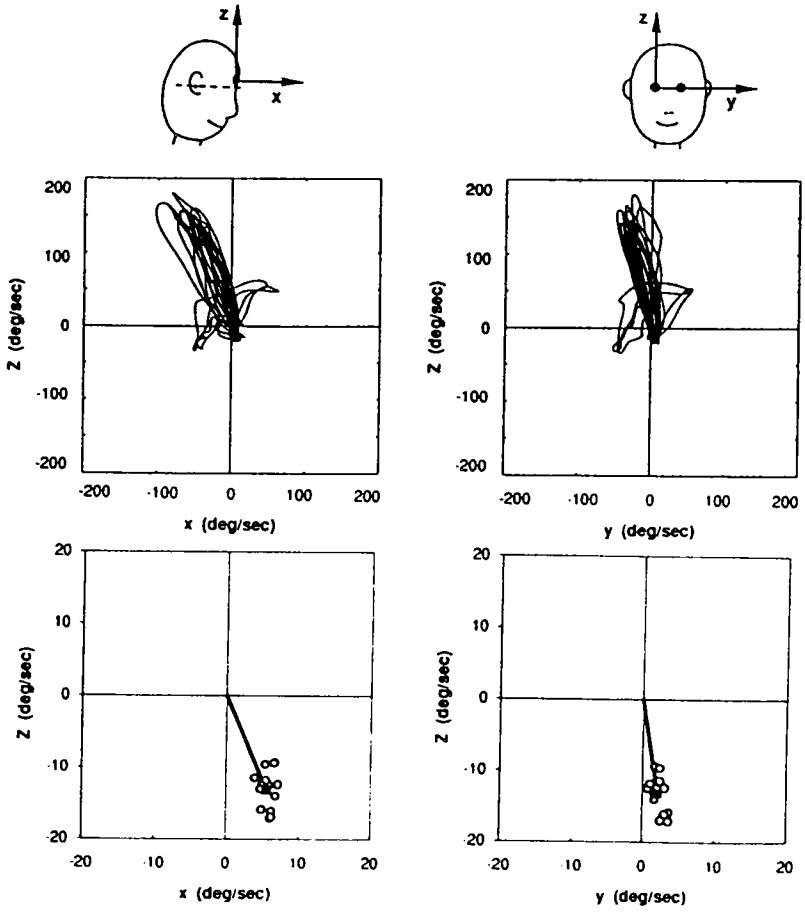

Fig 2. Angular velocity vectors during 6-second period of spontaneous nystagmus shown in Fig 1 in stereotaxic sagittal plane (left) and frontal plane (right). Upper row shows instantaneous vectors. Note vectors with angular velocity vectors around 15°/s (nystagmus fast phases) and oppositely directed vectors with angular velocities around 15°/s (nystagmus slow phases). Direction of these vectors indicates orientation of axis about which eye bulb rotates in reference to head-fixed coordinate system. Slow phase angular velocity vectors of each nystagmus beat (open circles) at instant 120 milliseconds after preceding fast phase and mean of these vectors (solid line and square) are shown in lower row. Note enlarged scale in lower graphs.

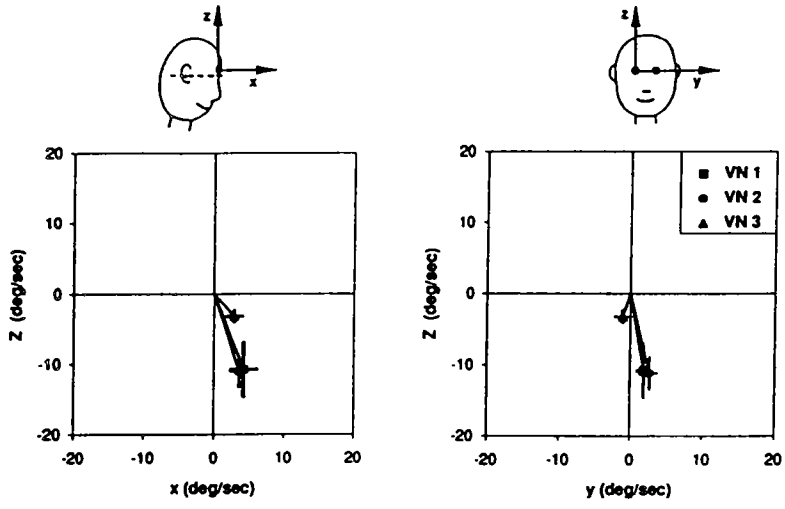

Fig 3. Mean slow phase angular velocity vectors, ±1 SD, of spontaneous nystagmus in three vestibular neurectomized patients. Patient VN1 had translabyrinthine cochleovestibular neurectomy, patient VN2 had supralabyrinthine vestibular neurectomy (superior branch of vestibular nerve), and patient VN3 had resection of inferior branch of vestibular nerve.

instant 120 milliseconds after the time when the vectorial velocity of a quick phase had dropped below 50°/s were selected as representative samples of each nystagmus slow phase (open circles in the lower graphs in Fig 2). The mean of these vectors (solid line and squares) represents the spontaneous nystagmus of a single recording period; several recording periods were averaged for calculating the direction and velocity of the spontaneous nystagmus of each patient.

RESULTS

Spontaneous Nystagmus Following Vestibular Neurectomy. A sample of spontaneous eye movements recorded in three dimensions in darkness in patient VN1 after resection of the (right) eighth nerve is shown in Fig 1. The most prominent components of this spontaneous nystagmus are fast phases to the left (+z) and slow eye movements to the right (−z). There are additional torsional (+x) and downward (+y) slow eye movements with saccades in the opposite direction. In Fig 2 the eye movements during this 6-second period are plotted as angular velocity vectors in the stereotaxic sagittal (xz) and frontal (yz) planes. The plots of the instantaneous angular velocity vectors (upper row) show fast eye movements of >150°/s with a negative x component in the sagittal plane and a negative y component in the frontal plane, as well as eye movements with much lower peak amplitudes (about 15°/s) in the opposite direction. These nystagmus slow phases are shown also as angular velocity vectors of each nystagmus beat in the lower graphs in Fig 2. In the sagittal plane, the mean angular velocity vector (which may be considered as the rotation axis of the eye bulb during the nystagmus slow phase) is tilted by the torsional (+x) eye movement components 23.0° ventrally. In the frontal plane there is a tilt of the mean angular velocity vector of 8.6° laterally to the left by the downward (+y) component.

After selective resection of the (right) superior vestibular nerve (patient VN2), the spontaneous nystagmus did not differ from that observed after total eighth nerve resection with regard to the angular velocity and to the direction of the slow component (Fig 3). Immediately after resection of the (right) inferior division of the vestibular nerve (16 years after removal of the homolateral superior vestibular nerve; patient VN3), a vigorous torsional, oblique left down-beating spontaneous nystagmus was seen with Frenzel glasses. The intensity of this spontaneous nystagmus declined rapidly during the next few days. When the 3-D recordings were performed on the fourth postoperative day, the mean slow phase velocity had declined to 4.7°/s. The mean angular velocity vector was directed more ventrally (relatively larger positive x component) than in patients VN1 and VN2 and was tilted right laterally (negative y component) instead of left laterally (Fig 3), confirming the clinical observation of a positive torsional (ie, extorsion of the upper pole of the right eye and intorsion of the upper pole of the left eye) and downward-beating nystagmus (fast phase).

Alexander's Law. The slow phase velocity of the spontaneous nystagmus varied as a function of the direction of gaze. In patient VN2, the horizontal slow phase velocity increased from 11.1°/s when looking straight ahead to 15.4°/s with lateral gaze 20° in the direction of the fast phase. With gaze of 20° in the opposite direction, the horizontal slow phase velocity decreased to 4.3°/s. There was, however, a similar increase and decrease of the eye movement components in the other directions; the direction of the nystagmus slow phase velocity vector thus did *not* change with changes of gaze direction (Fig 4).

Correlation With Orientation of Semicircular Canals. Figure 5 compares the direction of the spontaneous nystagmus with the stereotaxic orientation of

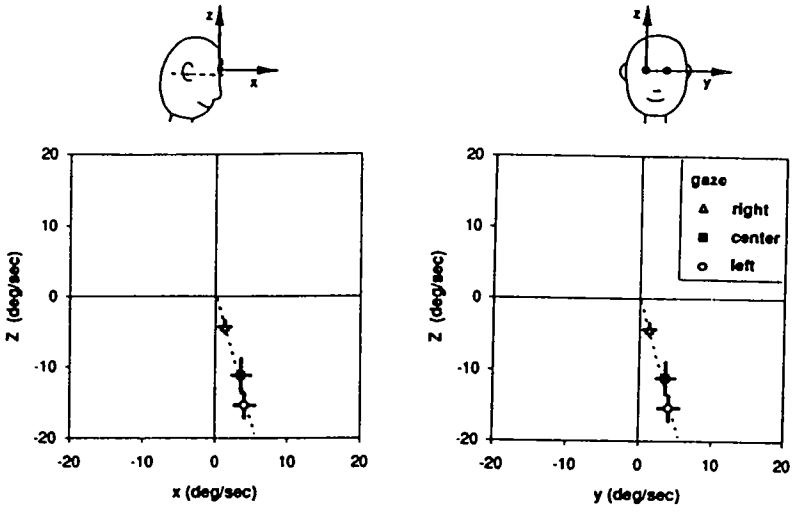

Fig 4. Mean slow phase velocity vectors, ±1 SD, of spontaneous nystagmus as function of direction of gaze (patient VN2). Note increase of angular velocity with gaze toward healthy ear (left) and decrease with gaze toward affected ear (Alexander's law), but no change of direction.

the SCC. The latter data were taken from Blanks et al[8] and are presented as (right) canal plane vectors. Head rotations about an axis parallel to these vectors induce maximal stimulation of a given SCC. The direction of the spontaneous nystagmus in patients VN1 and VN2 is closely collinear with the lateral canal plane vector. After resection of the inferior vestibular nerve (patient VN3), the direction of the nystagmus points in a direction corresponding to a vectorial addition of at least the lateral and the posterior canal vectors. (Only the loss of lateral canal function can induce a negative z component, ie, a left-beating nystagmus, and only the loss of the posterior canal can induce a negative y component, ie, a down-beating nystagmus.)

Spontaneous Nystagmus in Acute Peripheral Vestibulopathies. All patients with acute peripheral vestibulopathies had a spontaneous nystagmus with a horizontal slow phase component toward the affected ear (nystagmus beating toward the healthy ear). Within 11 days after onset of the disease, the horizontal slow

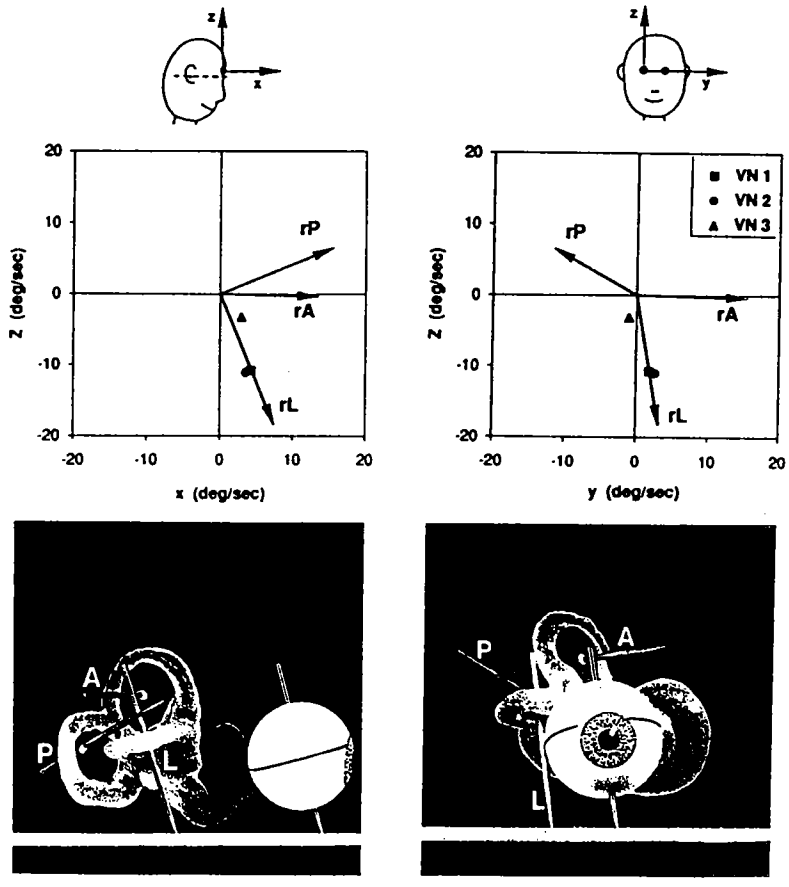

Fig 5. Mean slow phase velocity vectors induced by vestibular neurectomy compared to sensitivity vectors of semicircular canals. rL, rA, and rP indicate vectors of anatomic planes of right lateral, right anterior, and right posterior semicircular canals (data from Blanks et al[8]). These vectors are normalized; in this Figure, their unity value in direction of their maximum response corresponds to 20°/s. Polarity indicates direction of rotations that induce maximal inhibition of corresponding semicircular canal (corresponding to sudden loss of function). At bottom, these semicircular canal plane vectors are shown as bars attached to model of right labyrinth together with nystagmus angular velocity vector shown as rotation axis through model of eye bulb. A indicates right anterior, L right lateral, and P right posterior semicircular canal plane vector. White floor of this model represents stereotaxic horizontal (xy) plane.

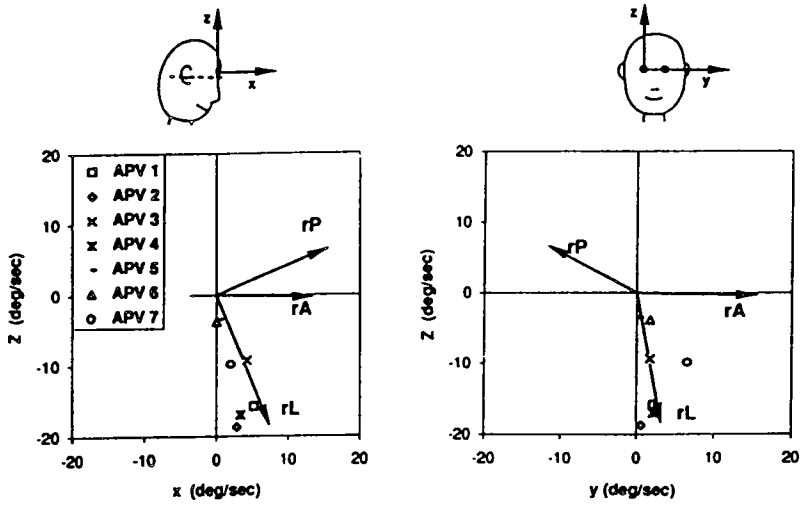

Fig 6. Mean slow phase velocity vectors of spontaneous nystagmus in patients with vestibular neuritis together with sensitivity vectors of (right) semicircular canals. All patients are treated as if right ear was affected. Note alignment of vectors along lateral semicircular canal plane vector with exception of patient APV7. For abbreviations, see Fig 5.

phase velocity was between 9°/s and 19°/s in five patients and 4.1°/s and 3.8°/s in patients APV5 and APV6, respectively. For better comparison, the directions of the nystagmus components were reversed in the patients with left-sided disorders, as if the right ear had been affected. The direction of the mean angular velocity vectors closely coincided with the lateral canal vector in all but one patient (Fig 6). The latter patient (APV7) had a relatively stronger positive y component (slow phase down) of his spontaneous nystagmus when recorded 11 days after onset of the disease.

DISCUSSION

Clinicians typically describe a spontaneous nystagmus as movements of the pupil relative to the orbit with horizontal, vertical, and torsional components. However, slow phase eye movements are generally produced by angular rotation of the eye bulb about a *single, head-fixed axis* running through the center of the eye *bulb*.[9] This axis may change over time, but — as shown in this study — its orientation in the head is relatively stable in spontaneous nystagmus after peripheral vestibular lesions and, furthermore, is independent of gaze position. A change of gaze position means a movement of the pupil to another place in the orbit. With a spontaneous nystagmus about a head-fixed axis, the pupil will thus perform different movements, depending on the direction of gaze. Presume, for example, rotations of the eye bulb about an axis parallel to the vector of the posterior SCC, which runs in an approximately horizontal head plane some 45° antero-ipsilaterally to the sagittal head plane. During ipsilateral gaze, the axis of gaze will be nearly parallel to the axis of eye rotation and the pupil will perform essentially torsional movements. When the gaze is directed, however, to the contralateral side, the axis of gaze will be orthogonal to the axis of rotation and the pupil will perform essentially vertical movements in the orbit. Conventional electro-oculographic recording techniques mimic this gaze dependency,[10] because they record the horizontal and vertical movement of the electrical axis of the eye bulb (coinciding with the gaze axis) relative to the electrodes attached around the orbit, while torsional movements (along the axis of gaze, ie, nearly parallel to the electrical axis of the eye bulb) are completely missed by this technique. The 3-D search coil technique used in this study, on the other hand, allows for accurate determination of the rotation axis of the eye bulb independent of the direction of gaze. The analysis of Alexander's law has shown that the rotation axis of the spontaneous nystagmus induced by vestibular neurectomy does not change with different horizontal gaze directions.

This provides the means to investigate how (or whether) an underlying vestibular lesion influences the direction of spontaneous nystagmus. Each SCC seems to induce eye movements that are roughly in the direction of its anatomic plane.[2] On physiological stimulation, the three pairs of SCCs, which are oriented in three nearly orthogonal planes in the head, generate compensatory eye movements (VORs) approximately in the direction of the head movement. This suggests vectorial addition of the sensory information originating from the six SCCs. The spontaneous nystagmus in peripheral vestibular disorders is attributed to the loss of the neuronal resting activity in the diseased vestibular afferents. One therefore may hypothesize that the angular rotation axis of spontaneous nystagmus could reflect the vectorial addition of the remaining SCC afferents.

A vestibular neurectomy represents a relatively well-controlled lesion that permits the evaluation of this hypothesis. Following deafferention of all three SCCs (cochleovestibular neurectomy), the eye movement vector coincided with the lateral SCC vector.

Theoretically, one would have expected a larger positive x (torsional) component induced by the simultaneous loss of the resting activity of the anterior and posterior SCC afferents. The vectors of these two SCC have similar x (torsional) directions, but oppositely directed y (vertical) components (Fig 5). Following deafferentation of the lateral and anterior SCCs and of the utricle (supralabyrinthine selective vestibular neurectomy), the angular velocity vector of the induced nystagmus also coincided with the lateral SCC vector. No significantly stronger positive y component (vertical downward slow component) was seen as one would expect from a vectorial addition of the loss of the lateral and anterior SCC afferent activity. Some effect of a sudden deafferentation of the posterior SCC, however, was seen after resection of the residual fibers of the inferior vestibular nerve in patient VN3 (cochleovestibular neurectomy following selective vestibular neurectomy). In this case, the relatively larger positive x component and the negative y component look like a vectorial addition resulting from a loss of the posterior and lateral SCCs, although the lateral SCC had been already deafferentated at the first operation 16 years earlier.

The complete absence of caloric responses in the diseased ear in five of the patients with acute peripheral vestibulopathies suggests a severe lesion of the labyrinth, which is very unlikely to be limited to the lateral SCC. Nevertheless, the angular velocity vector of the spontaneous nystagmus in these patients coincided closely with the lateral SCC vector (or had an even smaller x component). As in the patients with vestibular neurectomies, no nystagmus components in the directions of the vertical canals could be detected, with the exception of the patient with the largest delay between onset of disease and time of recording (APV7). A different organization of the horizontal, vertical, and torsional VORs is probably the reason for these unexpected findings. The six SCCs only differ in respect to the direction of maximal sensitivity to angular acceleration (a cosine function of the angle between the plane of the SCC and the rotation axis of the stimulus). Otherwise, all the SCCs have very similar physiological properties.[11] This suggests that a rotation about any axis gives rise to an equally weighted activity in the brain stem, ie, to an isotropic vestibular input. The oculomotor output of vestibular stimulation, on the other hand, is anisotropic; ie, the horizontal VORs differ from the vertical and torsional VORs in regard to gain (maximal slow phase velocity) and time constant. This anistropy of the oculomotor output even leads to a disalignment of the direction of reflexive eye movements from the direction of head rotation when the latter occur about an oblique axis.[12] Furthermore, the velocity storage mechanism in the brain stem, which prolongs the time constant of the vestibular signals, introduces another anisotropy by enhancing predominantly the horizontal eye movements.[13] This may also tilt the eye angular velocity vector of spontaneous nystagmus toward the lateral SCC vector. Therefore, estimating the contribution of each SCC to the generation of spontaneous nystagmus by mere vectorial addition is insufficient and an oversimplification. Furthermore, additional effects of utricular and saccular lesions may have to be considered. These are even more difficult to assess, because these organs do not have uniform sensitivity vectors, and therefore no distinct directions of eye movements can be attributed to their stimulation or loss of function.

In conclusion, the 3-D angular velocity vectors of the spontaneous nystagmus induced by vestibular neurectomy and in acute peripheral vestibulopathy were found to be surprisingly strongly clustered along the sensitivity vector of the lateral SCC. Significant deviations from this direction were only observed after resection of the inferior branch of the vestibular nerve and in one out of seven patients with acute peripheral vestibulopathy. The absence of nystagmus components in directions of the vertical SCCs may reflect an anisotropy of the oculomotor efferent part of the VOR arc rather than lesions limited to the lateral SCC afferents. The 3-D analysis of spontaneous nystagmus therefore does not permit accurate localization of a peripheral vestibular lesion. Supplementary dynamic stimulation, ie, analysis of VORs in response to angular accelerations in various directions, may give further information in this regard.

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NEUROMONITORING IN OTOLARYNGOLOGY AND SKULL BASE SURGERY

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