Three-Dimensional Vector Analysis of the Human Vestibuloocular Reflex in Response to High-Acceleration Head Rotations
II. Responses in Subjects With Unilateral Vestibular Loss and Selective Semicircular Canal Occlusion

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SUMMARY AND CONCLUSIONS

1. We studied the three-dimensional input-output human vestibuloocular reflex (VOR) kinematics after selective loss of semicircular canal (SCC) function either through total unilateral vestibular deafferentation (uVD) or through single posterior SCC occlusion (uPCO), and showed large deficits in magnitude and direction in response to high-acceleration head rotations (head "impulses").

2. A head impulse is a passive, unpredictable, high-acceleration (3,000–4,000/s²) head rotation through an amplitude of 10–20° in roll, pitch, or yaw. The subjects were tested while seated in the upright position and focusing on a fixation target. Head and eye rotations were measured with the use of dual search coils, and were expressed as rotation vectors. A three-dimensional vector analysis was performed on the input-output VOR kinematics after uVD to produce two indexes in the time domain: magnitude and direction. Magnitude is expressed as speed gain (G) and direction as misalignment angle (δ).

3. G, after uVD, was significantly lower than normal in both directions of head rotation during roll, pitch, and yaw impulses, and were much lower during ipsilesional than during contralesional roll and yaw impulses. At 80 ms from the onset of an impulse (i.e., near peak head velocity), G was 0.23 ± 0.08 (SE) (ipsilesional) and 0.56 ± 0.08 (contralesional) for roll impulses, 0.61 ± 0.09 (up) and 0.72 ± 0.10 (down) for pitch impulses, and 0.36 ± 0.06 (ipsilesional) and 0.76 ± 0.09 (contralesional) for yaw impulses (mean ± 95% confidence intervals).

4. δ, after uVD, was significantly different from normal during ipsilesional roll and yaw impulses and during pitch-up and pitch-down impulses. δ was normal during contralesional roll and yaw impulses. At 80 ms from the onset of the impulse, δ was 30.6 ± 4.5 (ipsilesional) and 13.4 ± 5.0 (contralesional) for roll impulses, 23.7 ± 3.7 (up) and 31.6 ± 4.4 (down) for pitch impulses, and 68.7 ± 13.2 (ipsilesional) and 11.0 ± 3.3 (contralesional) for yaw impulses (mean ± 95% confidence intervals).

5. VOR gain (γ), after uVD, were significantly lower than normal for both directions of roll, pitch, and yaw impulses and much lower during ipsilesional than during contralesional roll and yaw impulses. At 80 ms from the onset of the head impulse, the γ was 0.22 ± 0.08 (ipsilesional) and 0.54 ± 0.09 (contralesional) for roll impulses, 0.55 ± 0.09 (up) and 0.61 ± 0.09 (down) for pitch impulses, and 0.14 ± 0.10 (ipsilesional) and 0.74 ± 0.06 (contralesional) for yaw impulses (mean ± 95% confidence intervals). Because γ is equal to |G* cos (δ)|, it is significantly different from its corresponding G during ipsilesional roll and yaw, and during all pitch impulses, but not during contralesional roll and yaw impulses.

6. After uPCO, pitch-vertical γ during pitch-up impulses was reduced to the same extent as after uVD; roll-torsional γ during ipsilesional roll impulses was significantly lower than normal but significantly higher than after uVD. At 80 ms from the onset of the head impulse, γ was 0.32 ± 0.13 (ipsilesional) and 0.55 ± 0.16 (contralesional) for roll impulses, 0.51 ± 0.12 (up) and 0.91 ± 0.14 (down) for pitch impulses, and 0.76 ± 0.06 (ipsilesional) and 0.73 ± 0.09 (contralesional) for yaw impulses (mean ± 95% confidence intervals).

7. The eye rotation axis, after uVD, deviates in the yaw plane, away from the normal interaural axis, toward the nasooccipital axis, during all pitch impulses. After uPCO, the eye rotation axis deviates in same direction as after uVD during pitch-up impulses, but is well aligned with the head rotation axis during pitch-down impulses. These misalignments can be explained by activation of the direct neural connections between the vertical SCCs and the extraocular muscles. During all pitch impulses after uVD, and during pitch-up impulses after uPCO, there is excitation and reciprocal inhibition of single, instead of pairs of, vertical SCCs, producing vertical as well as contralesional torsional eye rotations. The torsional eye rotations occur because the oppositely directed torsional eye rotations arising from stimulation of pairs of vertical SCCs are no longer canceled.

8. The eye rotation axis, after uVD, deviates in the pitch plane away from the normal rostrocaudal axis toward the nasooccipital axis during ipsilesional yaw impulses. In contrast, the eye rotation axis remains well aligned with the head rotation axis during contralesional yaw impulses. We propose that the anatomic orientation of, and the direction the endolymph flow, in the remaining intact vertical SCCs can explain this misalignment. After uVD, the dominant excitation from the ipsilesional lateral SCC is absent. The relative magnitude of excitation from the intact posterior SCC is larger than that from the anterior SCC on the same side, which results in small horizontal, downward vertical, and large ipsilesional torsorial eye rotations.

INTRODUCTION

The human vestibuloocular reflex (VOR) has traditionally been measured in terms of VOR gain (γ) to assess the deficits after unilateral loss of semicircular canal (SCC) function (Aw et al. 1994, 1995a,b; Fletcher et al. 1996; Foster et al. 1994; Halmagyi and Curthoys 1988; Halmagyi et al. 1990; Maioli and Precht 1985; Paige 1989; Vibert et al. 1993). Because the VOR operates in three dimensions, an exact description of the total VOR performance requires the three-dimensional measurement and vector analysis of
its input-output kinematics in terms of both magnitude and direction. Simply recording a deficit in $\gamma$ during head rotations does not specify whether this is the result of an insufficient eye velocity magnitude (eye speed), or of misalignment of the eye rotation axis with respect to head rotation axis, or of both.

In this second of two papers, we studied the VOR in two groups of subjects with loss of SCC function after either unilateral vestibular deafferentation (uVD) or inactivation of one posterior SCC, in response to high-acceleration head rotations. A three-dimensional vector analysis algorithm (Aw et al. 1996) was used to quantify the deficits of the total VOR performance in terms of magnitude, expressed as speed gain ($G$), and direction, expressed as misalignment angle ($\theta$), in the time domain, during roll, pitch, and yaw head rotations in these subjects. Apart from the three-dimensional vector analysis, we also used the traditional one-dimensional analysis on the data from the same subjects during roll, pitch, and yaw impulses so that our data could be compared with those from previous studies. With the use of a vectorial resolution technique (Curthoys et al. 1977) and the published anatomic data on the human SCCs (Blanks et al. 1975), we calculated the relative unit magnitude of SCC stimulation during roll, pitch, and yaw head rotations to explain the eye rotation axis misalignments after the unilateral loss of SCC function.

METHODS

The methods were the same as in the companion paper (Aw et al. 1996). Only those parts that are specific to this study are presented in detail here.

Subjects

We studied 10 uVD subjects (35–65 yr, 51.4 ± 9.2 yr, mean ± SD), and 6 unilateral posterior SCC occluded (uPCO) subjects (52–67 yr, 63.5 ± 5.8 yr). The 10 (6 left and 4 right) uVD subjects had undergone uVD either during surgical removal of an acoustic neuroma (6) or as treatment for Ménière’s disease (4). They were tested >1 yr post-uVD (4.6 ± 2.3 yr, mean ± SD). One subject had bilateral vestibular deafferentation (bVD) during complete removal of large bilateral acoustic neuremas 10 yr previously and therefore had no vestibular function. As a result of the surgery, this subject was also totally deaf and had bilateral facial palsies.

The six subjects with uPCO (3 left and 3 right) had undergone surgical occlusion of one posterior SCC >1 yr previously (1.3 ± 0.2 yr, mean ± SD) as treatment for intractable benign positional vertigo. A technique similar to the procedure described by Parnes and McClure (1991) had been used. All the uPCO subjects were tested before and after the SCC occlusion with clinical audiometry (range: 125–8,000 Hz). Postoperatively, all the uPCO subjects had preserved hearing in the operated ear, and statistical analysis of the hearing threshold at 2 kHz showed no detectable change ($-2 ± 3$ dB, mean ± SD). All the uPCO subjects also had pre- and postoperative bithermal caloric tests, and yaw rotation tests with sinusoidal (0.1 and 0.33 Hz) stimuli and also stimulus at a constant acceleration of $20^\circ/\text{s}^2$ for 5 s. After surgery, none of the subjects had any significant change in their caloric or rotational test results. From these results we conclude that uPCO subjects had unaltered cochlear and lateral SCC function.

The results of these subjects were compared with the results of the 12 normal subjects (21–53 yr, 35 ± 11 yr) (Aw et al. 1996).

All the subjects gave informed consent. The protocol was approved by the Royal Prince Alfred Hospital Human Ethics Committee.

RESULTS

Responses from the uVD and uPCO subjects were analyzed in one dimension and three dimensions. One-dimensional analysis allows us to compare the results with data from previous studies. Three-dimensional vector analysis quantifies the input-output kinematics of the VOR as three-component vectors with magnitudes and directions. Temporospatial and spatial analyses define the misalignment in three dimensions in different views (roll, pitch, and yaw planes). The results from this study are compared with those from normal subjects (Aw et al. 1996). The impulse directions in roll, pitch, and yaw and the directions of the torsional, vertical, and horizontal components of the coil signals are expressed with respect to the subject’s viewpoint. All position data are displayed as rotation vectors.

One-dimensional VOR analysis

In one-dimensional analysis, we used only one of the three components of the vector for the analysis. During yaw impulses, only the horizontal head and eye velocity components were analyzed, and we referred to this with the term yaw-horizontal VOR. Likewise, the term pitch-vertical VOR meant that only the vertical components were analyzed during pitch impulses, and similarly the term roll-torsional VOR meant that only the torsional velocity components were analyzed during roll impulses.

Representative profiles of the VOR responses during roll, pitch, and yaw impulses in a left uVD subject are shown as time series of head, gaze, and eye positions, velocities, and accelerations in one dimension (Fig. 1, A and B). All the eye signals are inverted for comparison. In each graph, the arrows indicate the onset of the impulse, which is 20 ms after the beginning of the plot.

During an ipsilesional roll counterclockwise impulse, the compensatory torsional eye position, velocity, and acceleration were substantially lower than normal, which resulted in a larger than normal torsional position error. During a contralesional roll-clockwise (roll-CW) impulse, the compensatory torsional eye position, velocity, and acceleration increased up to the peak of torsional head velocity, but were still slightly lower than normal, resulting in a torsional eye position error larger than normal. During both pitch-up and pitch-down impulses, the compensatory vertical eye positions, velocities, and accelerations were symmetrically decreased for pitch-up and pitch-down impulses. Horizontal eye responses showed larger deficits during a yaw-left (ipsilesional) impulse than during a yaw-right (contralesional) impulse, resulting in a larger than normal horizontal eye position error.

Time series of means ± 95% confidence intervals of the averaged roll-torsional, pitch-vertical, and yaw-horizontal head and eye velocities for 10 uVD and 6 uPCO subjects during roll, pitch, and yaw impulses are displayed in Fig. 2. The responses to roll and yaw impulses were grouped into ipsilesional and contralesional responses. In each graph, an arrow marks the onset of the impulse at 20 ms. Means ±
95% confidence intervals of roll-torsional, pitch-vertical, and yaw-horizontal eye velocity as a function of its respective head velocity of the same data are displayed in Fig. 3.

The patterns of deficits after uVD to roll and yaw impulses were similar (Figs. 2 and 3, top rows). During ipsilesional roll impulses, torsional eye velocity initially increased for ~40 ms and then decreased despite increasing torsional head velocity. During contralesional roll impulses, torsional eye velocity increased until the peak of torsional head velocity or close to it. Likewise, the horizontal eye responses were similar in pattern during yaw impulses. Responses to both roll and yaw impulses were significantly lower than normal for both ipsilesional and contralesional roll and yaw impulses ($P < 0.05$), with more marked deficits for ipsilesional impulses. Pitch-down and pitch-up impulses after uVD produced symmetrical vertical eye responses ($P > 0.05$, 1-sample $t$-test), which increased until vertical head velocity peaked, but were nonetheless significantly less than normal. At 80 ms from the onset of the head impulse (i.e., near peak head velocity), $\gamma$ was $0.22 \pm 0.08$ (ipsilesional) and $0.54 \pm 0.09$ (contralesional) for roll impulses, $0.55 \pm 0.09$ (up) and $0.61 \pm 0.09$ (down) for pitch impulses, and $0.14 \pm 0.10$ (ipsilesional) and $0.74 \pm 0.06$ (contralesional) for yaw impulses (mean $\pm 95\%$ confidence intervals).

After uPCO (Figs. 2 and 3, bottom rows), the VOR deficits were observed mainly during ipsilesional roll and pitch-up impulses; the ipsilesional roll VOR deficit was significantly less than normal, but was significantly more than after uVD. Responses to contralesional roll impulses were normal. The VOR response was similar to those in uVD subjects during pitch-up impulses and was normal during pitch-down impulses. The pitch vertical $\gamma$ during pitch up and pitch-down impulses was asymmetric, with the gain during pitch-up impulses significantly lower than during pitch-down impulses ($P < 0.05$, 1-sample $t$-test). Surprisingly, the ipsilesional and contralesional compensatory horizontal eye velocity responses to yaw impulses were slightly but significantly lower than normal. At 80 ms from the onset of the head impulse (i.e., near peak head velocity), $\gamma$ was $0.32 \pm 0.13$ (ipsilesional) and $0.55 \pm 0.16$ (contralesional) for roll impulses, $0.51 \pm 0.12$ (up) and $0.91 \pm 0.14$ (down) for pitch impulses, and $0.76 \pm 0.06$ (ipsilesional) and $0.73 \pm 0.09$ (contralesional) for yaw impulses (mean $\pm 95\%$ confidence intervals).

bVD subject

One bVD subject was tested with roll, pitch, and yaw head impulses. The compensatory eye velocity responses during roll, pitch, and yaw impulses were small, resulting in large eye position errors. Figure 4 shows typical examples of torsional, vertical, and horizontal eye responses during roll, pitch, and yaw head impulses, respectively, in the bVD subject. At 80 ms from the onset of the head impulse (i.e.,
near peak head velocity), $\gamma$ was $0.03 \pm 0.03$ (SD) for roll impulses, $0.05 \pm 0.01$ (SD) for pitch impulses, and $0.02 \pm 0.02$ (SD) for yaw impulses. In pitch and yaw impulses, corrective saccades occurred at $\sim 150$ ms after the onset of the impulse and acted to decrease the eye position errors. However, torsional fast phases were not observed during roll impulses. During the first 100 ms of the impulses of amplitudes $\sim 20^\circ$, the torsional eye position errors were $\approx 90\%$ in roll, and the vertical and horizontal eye position errors were $\approx 95\%$ in pitch and yaw, respectively.

Three-dimensional vector analysis of the input-output VOR kinematics after uVD

The three-dimensional input-output VOR kinematics during roll, pitch, and yaw impulses after uVD were analyzed with the use of a three-dimensional vector analysis algorithm (Aw et al. 1996). Unlike the one-dimensional analysis, all three components of the vector (e.g., head or eye velocity) were used in the three-dimensional vector analysis. This analysis quantifies magnitude expressed as $G$ and direction expressed as $\delta$. 

Figure 5 shows the means $\pm 95\%$ confidence intervals of the $G$, $\delta$, and $\gamma$ during roll, pitch, and yaw impulses. The first 10 ms of the data were not plotted because division of small values of eye velocity by small values of head velocity, during the initial part of the head impulse, can lead to large errors. Table 1 summarizes the means $\pm 95\%$ confidence intervals of the $G$, $\delta$, and $\gamma$ at 30 ms (10 ms from the onset of impulse) and at 100 ms (near the peak of the impulse).

$G$ after uVD was lower than normal in both directions during roll, pitch, and yaw impulses (Fig. 5, top row). There were deficits in the magnitude of the input-output kinematics of the VOR during both ipsilesional and contralesional roll and yaw and pitch-up and down impulses. The $G$ deficits were also larger during ipsilesional than during contralesional roll and yaw impulses. Comparison of the roll ipsilesional $G$ with the yaw ipsilesional $G$ showed that whereas the roll $G$ decreased to a minimum of $\sim 0.22$, yaw $G$ decreased initially, then remained almost constant at $\sim 0.36$ as the yaw head speed increased to its peak. The pattern of ipsilesional yaw $G$ showed that as the yaw head speed increased, the compensatory eye speed was initially less than the yaw head speed and $G$ decreased initially for $\sim 50$ ms after the onset of the yaw impulse. Thereafter, $G$ remained constant at 0.36, showing that eye speed at this stage increased proportionally with head speed. During pitch impulses, $G$ was almost constant at $\sim 0.70$ from the onset of the impulse to the peak of head speed. Thus $G$ during both directions of roll, pitch, and yaw impulses was significantly lower than normal.

After uVD, $\delta$ was significantly different from normal during ipsilesional yaw and roll impulses and pitch-up and pitch-down impulses (Fig. 5, middle row). The $\delta$ increased to $\sim 30^\circ$ during ipsilesional roll and to a maximum of $69^\circ$ during ipsilesional yaw impulses as the head speed increased from onset to its peak. However, during contralesional yaw or roll impulses, $\delta$ remained small and was not significantly different from normal. Although the patterns of responses to pitch-up and pitch-down impulses were very similar, $\delta$ was slightly smaller, $\sim 24^\circ$ during pitch-up and $\sim 32^\circ$ during pitch-down impulses.

After uVD, $\gamma$ was significantly lower than normal in response to impulses in all six directions. Also, the large values of $\delta$ during ipsilesional roll and yaw and pitch-up and pitch-down impulses resulted in $\gamma$ being significantly different from its $G$ during these impulses [$\gamma = G \times \cos(\delta)$]. An important difference between $G$ and $\gamma$ during ipsilesional yaw impulses should be highlighted. Although the ipsilesional yaw $G$ was $\sim 0.36$ and $\delta$ was $\sim 69^\circ$, its corresponding yaw-horizontal $\gamma$ was only $\sim 0.14$.

Figure 6 shows the time series of the mean spatial misalignment angles, which are obtained from the projections of $\delta$ onto two appropriate orthogonal coordinate planes for roll, pitch, and yaw impulses in left and right uVD subjects. These spatial misalignment angles show in which coordinate plane the eye rotation axis deviates from the head rotation axis.

During ipsilesional roll impulses, the eye rotation axis
was misaligned by ~25° in the yaw plane (Fig. 6, bottom left, black dashed line and gray solid line). In addition to the expected small compensatory torsional eye velocity during ipsilesional roll impulses, there was a small but consistent positive vertical velocity in left and right uVD subjects, resulting in the eye rotation axis being rotated from the nasooccipital axis toward the interaural axis. Alignment of the cyc rotation axis during contralateral roll impulses was almost collinear, as shown by small spatial misalignment angles in the yaw plane of ~5° (Fig. 6, bottom left, black solid line and gray dashed line).

During pitch-up and -down impulses, the eye rotation axes were misaligned by ~25° in the yaw plane (Fig. 6, bottom middle) both in the left (black solid and dashed lines) and right (gray solid and dashed lines) uVD subjects. The spatial misalignment angles show that, in addition to the compensatory vertical eye velocities, there were consistent counterclockwise (CCW) torsional eye velocities in left uVD subjects and consistent CW torsional eye velocities in right uVD subjects, resulting in the eye rotation axis being rotated from the interaural axis toward the nasooccipital axis.

The cyc rotation axis was misaligned with the head rotation axis by up to 69° during ipsilesional yaw impulses in the pitch plane in left and right uVD subjects (Fig. 6, bottom right, black solid line and gray dashed line). This was due to the presence of large CCW torsional eye velocities in left uVD subjects and CW torsional eye velocity in right uVD subjects, which resulted in the eye rotation axis being rotated from the rostrocaudal axis toward the nasooccipital axis. During contralateral yaw impulses, the eye rotation axis remained well aligned with the head rotation axis, with only small spatial misalignment angles of ~5° (Fig. 6, bottom right, black dashed line and gray solid line).

**Eye rotation axis misalignment during pitch impulses**

In Fig. 7 the spatial eye velocity responses during pitch impulses in a normal, a left uVD, a right uVD, and a left uPCO subject are compared. The results from all 10 trials during pitch-up and pitch-down impulses are displayed in the yaw plane view. Normally, the eye rotation axis almost mirrored the head rotation axis during pitch impulses. After uVD, in addition to the compensatory downward or upward eye rotations, there was a torsional eye velocity component that resulted in a deviation of the eye rotation axis from the interaural (Y) axis toward the nasooccipital (X) axis. After left uVD, torsional eye velocity was CCW, whereas after right uVD it was CW. The response after left uPCO was similar to that after left uVD during pitch-up impulses, but was normal during pitch-down impulses.

**Eye rotation axis misalignment during yaw impulses**

A comparison of the spatial characteristics of the head and eye rotation axes in the pitch plane during yaw impulses...
Roll Pitch Yaw

Relative unit magnitude of canal stimulation during head rotations

Measurements by Blanks et al. (1975) showed that the SCC planes do not lie along the planes of roll, pitch, or yaw head rotations. Therefore the relative magnitude of SCC stimulation during these head rotations will depend on the area of the SCC projection onto the plane of the head rotation and the direction of the endolymph flow in the SCCs during these rotations. Consequently, with the use of a vectorial resolution technique (Curthoys et al. 1977) that takes into account the SCC orientations and the direction of the endolymph flow, we calculated the relative unit magnitude of SCC stimulation during roll-CW, pitch-down, and yaw-left head rotation.

Our calculations, with the use of vector resolution techniques, take into account the fact that Reid’s stereotaxic plane was at 7° above the earth’s horizontal and we describe the pattern of excitation and inhibition in all six SCCs during the impulses. Figure 9 shows the relative unit magnitude of SCC excitation and inhibition during roll-CW, pitch-down, and yaw-left head rotations. Excitations are displayed as positive values and inhibitions as negative values.

Our calculations show that during a roll-CW impulse, the right anterior and posterior SCCs and also the right lateral SCC are excited, whereas the three left SCCs are inhibited.

During a pitch-down impulse, both anterior SCCs and both lateral SCCs are excited and posterior SCCs are inhibited.

During a yaw-left impulse, in addition to left lateral SCC excitation, it is the right and not the left posterior SCC that is excited (Curthoys et al. 1977). This is due to the backward tilt of the posterior SCC of 71° from Reid’s stereotaxic plane (Blanks et al. 1975) and the “amphullofugal” flow that results in posterior SCC excitation (Ewald 1892).

### TABLE 1. Means ± 95% confidence intervals of G, δ, and γ by three-dimensional vector analysis in uVD subjects

<table>
<thead>
<tr>
<th>uVD</th>
<th>At, ms</th>
<th>Roll</th>
<th>Pitch</th>
<th>Yaw</th>
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<tr>
<td></td>
<td></td>
<td>Ipsilesion</td>
<td>Contraslesion</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Up</td>
<td>Down</td>
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<tr>
<td></td>
<td></td>
<td>Ipsilesion</td>
<td>Contraslesion</td>
<td></td>
</tr>
<tr>
<td>G</td>
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<td>0.74 ± 0.04</td>
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<td>100</td>
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</tr>
<tr>
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<tr>
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</tr>
<tr>
<td>γ</td>
<td>30</td>
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<td>0.72 ± 0.04</td>
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</tr>
<tr>
<td></td>
<td>100</td>
<td>0.22 ± 0.08</td>
<td>0.54 ± 0.09</td>
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Data for 10 subjects. Note that the onset of the impulse is at 20 ms from the beginning of the impulse data. G, speed gain; δ, misalignment angle; γ, vestibuloocular reflex gain; uVD, unilateral vestibular deafferentation. Values in Roll, Pitch, and Yaw are means ± SD.
FIG. 6. Spatial misalignment angles obtained from the projection of $\delta$ onto the 2 orthogonal coordinate planes that intersect at the head rotation axis. Black solid and dashed lines: responses from left uVD subjects. Gray solid and dashed lines: responses from right uVD subjects. The terms "CCW" (dashed lines), "CW" (solid lines), "Up" (dashed lines), "Down" (solid lines), "Right" (dashed lines), and "Left" (solid lines) refer to the directions of impulses. Drawings illustrate the coordinate plane onto which $\delta$ is projected. Curved arrow shows the direction of the positive spatial misalignment angle. Plots show that the eye rotation axis is misaligned with the head rotation axis mainly during ipsilesional roll and yaw impulses, and during pitch-up and -down impulses (bottom row).

FIG. 7. Misalignment of the eye velocity axis during pitch impulses. Projections of eye velocity and head velocity onto the yaw plane during pitch impulses for a normal, a left uVD, a right uVD, and a left uPCO subject. Ten trials are shown for each direction of impulse. In the normal subject, the eye rotation axis was almost perfectly aligned with the head rotation axis during pitch impulses. In the left uVD subject, the eye rotation axis was deviated away from the interaural ($Y$) axis backward, whereas in the right uVD subject it deviated forward, toward the nasooccipital ($X$) axis. In the left uPCO subject, the deviation of the eye rotation axis was similar to that in the left uVD subject during pitch-up impulses, but similar to that in the normal subject during pitch-down impulses.
**DISCUSSION**

**Permanent deficits in VOR magnitude and direction after loss of SCC function**

Although previous one-dimensional studies have shown that there is some, although incomplete, recovery in the VOR during the first 4 mo after uVD (Curthoys and Halmagyi 1995; Halmagyi et al. 1990), our present study further confirms that this recovery is still incomplete >1 yr after unilateral loss of the SCC function. These residual VOR deficits have time-varying components both in magnitude (speed) and in direction during high-acceleration head rotations (3,000–4,000°/s/s) in the frequency range of ~2 Hz and are present even with the functional loss of one SCC (Aw et al. 1995a,b; Barmack and Pettorossi 1988; Yakushin et al. 1995).

Significant deficits in $G$ were recorded during both directions of roll, pitch, and yaw impulses. The fundamental findings in $G$ deficits in the three-dimensional analysis can also be identified in the one-dimensional analysis as $\gamma$ deficits. These deficits exist for both the excitatory and the inhibitory directions of stimulation, suggesting that both excitation and disinhibition of the SCCs are essential for a normal VOR during high-acceleration head rotations. The results show more profound $G$ deficits during ipsilesional than during contralateral roll and yaw impulses, indicating that SCC excitation generates a larger component of the eye response than SCC disinhibition during these high-acceleration head rotations. The fundamental processes for the asymmetric responses during high-acceleration head rotations remain to be defined from further investigations with the use of such techniques as cellular recordings in animal models during these stimuli. $G$ in uVD subjects was also significantly symmetrically lower than in normal subjects during pitch impulses. This suggests that excitation and reciprocal inhibition of a pair of, instead of single, anterior and posterior SCCs, respectively, during pitch-down or pitch-up impulses are responsible for generating the normal pitch VOR.

The $\delta$ was significantly larger than normal during ipsilesional roll and yaw, and during pitch-up and pitch-down impulses, but was normal during contralateral roll and yaw impulses. This suggests that SCC excitation is primarily responsible for maintaining the normal alignment of the eye rotation axis during head rotation.

These results show that all six SCCs are essential for generating a normal angular VOR during high-acceleration head rotations. The contribution of each SCC to the angular VOR depends on the area of the SCC projection onto the plane of the angular head rotation and the direction of endolymph flow in the SCCs. Normally, the summation of excitation and inhibition from all six SCCs generates compensatory eye rotations that are well aligned with, and directed opposite to, the head rotations, with yaw and pitch $G$ close to 1.0 and a roll $G$ of ~0.7 (Aw et al. 1996).

**Mechanisms for eye rotation axis misalignment**

The two mechanisms proposed to explain the misalignments are 1) the geometric orientation of the SCCs in the head and 2) the direct neural connections between the SCCs and the extraocular muscles. The misalignment of eye rotation axis with respect to the head rotation axis during pitch impulses is probably due to excitation and reciprocal inhibi-
tion of single, instead of pairs of, anterior and posterior SCC's during pitch-down or pitch-up impulses. Normally, during pitch impulses, when pairs of anterior and posterior SCCs are excited and reciprocally inhibited, the opposing torsional eye rotations are canceled, resulting only in upward or downward eye rotations. However, during pitch-up impulses after uVD, excitation of one posterior SCC and reciprocal inhibition of one anterior SCC produces excitation of the ipsilateral superior oblique muscle and the contralateral inferior rectus muscle, producing downward and ipsilesional torsional eye rotations. After uPCO, the mechanism is similar, except with the added disinhibition from the anterior SCC on the lesioned side. Similarly, during pitch down impulses after uVD, excitation of one anterior SCC and reciprocal inhibition of one posterior SCC excites the ipsilateral superior rectus and the contralateral inferior oblique muscles, resulting in upward and ipsilesional torsional eye rotations. This torsional component causes the eye rotation axis to deviate from the normal interaural (Y) axis toward the nasooccipital (X) axis in the yaw plane.

The effects of single SCC excitation have been demonstrated in cats and monkeys by electrical stimulation of the ampullary nerves of the vertical SCCs (Cohen and Suzuki 1963; Cohen et al. 1964; Suzuki et al. 1964). Although our results show that compensatory eye rotations from the contralesional roll and yaw impulses are well aligned with head rotations, suggesting that SCC excitation is largely responsible for the alignment of the eye rotation axis, these experiments could not separate the effect of single vertical SCC excitation from the effect of reciprocal inhibition of the other vertical SCC.

The eye rotation axis misalignment during yaw impulses can be explained by the geometric orientation of the vertical SCCs in the head (Blanks et al. 1975) and the vertical SCC excitation from ampullofugal endolymph flow (Ewald 1892). Figure 9, right, shows that during yaw-left impulses, only the left lateral SCC and the right posterior SCC are excited, and with inhibition from the right lateral SCC and the left posterior SCC. The anterior SCC, being almost vertical—89° from Reid's stereotaxic plane (Blanks et al. 1975a)—makes a negligible contribution. Thus, during an ipsilesional yaw impulse after uVD, the response is from disinhibition from the intact lateral SCC, and also the excitation of the intact posterior SCC. The downward eye rotations produced by the posterior SCC excitation and the small upward eye rotations produced by anterior SCC excitation on the intact side result in small net downward eye rotations, but both posterior and anterior SCCs produce ipsilesional torsional eye rotations (i.e., the eyes rolling toward the side of the lesion from the subject's viewpoint). Therefore the compensatory eye rotations are produced by a combination of a small horizontal (from disinhibition from the intact lateral SCC), a small vertical, and a large ipsilesional torsional eye rotation. Consequently the eye rotation axis deviates from the normal rostrocaudal (Z) axis toward the nasooccipital (X) axis in the pitch plane. Normally, the dominant excitation of the left lateral SCC produces an eye rotation axis that is well aligned with the head rotation axis, with only a small noncollinearity of ~5°.

During ipsilesional roll impulses, small net upward eye rotations were observed after uVD, which caused the misalignment of the eye rotation axis. These upward eye rotations probably arise from the geometric error due to the natural lateral displacement of the eye from the nasooccipital axis (Seidman et al. 1995). Because the compensatory torsional eye velocity is small (roll G is ~0.2), even a small net vertical eye velocity can result in a misalignment between eye and head rotation axes of ~30°.

Similarities between vestibular deafferentation and canal occlusion

We have confirmed our earlier finding that the VOR deficits after uVD and uPCO are essentially similar (Aw et al. 1994, 1995a,b), Barmack and Petrorossi (1988) also found no recovery in the yaw-horizontal γ in the rabbit after lateral SCC inactivation. Yakushin et al. (1995) also reported the same inability to adapt the spatial response of the angular VOR after canal-plugging in monkeys tested long after the lesion. In contrast, Paige (1983) reported that the compensation is adequate in monkeys after lateral SCC inactivation in the low-frequency and low-velocity stimulus range and showed that the yaw-horizontal γ increased from ~0.5 to ~0.8. Our data showed that loss of SCC function produces deficits in magnitude and direction during high-acceleration head rotations that are not fully compensated, regardless of the cause of the loss.

Potential clinical application

Vector analysis of the VOR in response to high-acceleration head rotations shows distinct patterns of deficits in uVD and uPCO subjects. This allows for a clear evaluation of the functional status of individual lateral or vertical SCCs when the subjects are tested with yaw and pitch impulses and the data are measured and analyzed in three dimensions. These methods have potential application in testing patients with diseases affecting individual SCCs.

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