Semicircular canal plane head impulses detect absent function of individual semicircular canals

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Summary

We studied the human vestibulo-ocular reflex (VOR) in response to head 'impulses': brief, unpredictable, passive, high-acceleration (up to 4000°/s^2), low-amplitude (20–30°) head rotations. We delivered the head impulses approximately in the plane of the semicircular canal (SCC) being tested. To test the anterior and posterior SCCs, the head impulses were delivered in a diagonal plane, midway between the frontal (roll) and sagittal (pitch) planes. We recorded head and eye position in three dimensions with scleral search coils in nine normal subjects, seven patients following unilateral surgical vestibular neurectomy and three patients following unilateral posterior SCC occlusion. In the post-surgical patients we demonstrated a severe, permanent VOR gain deficit (0.2–0.3) for head impulses directed toward any single non-functioning SCC. The sensitivity of the test depends on the physiological properties of primary vestibular afferents, and its specificity depends on the anatomical orientation of the SCCs. The diagonal head impulse is the first test of individual vertical SCC function in humans, and together with the horizontal head impulse, forms a comprehensive battery of SCC-plane tests. These canal-plane impulses could be useful in evaluating patients with vertigo or other vestibular disorders.

Keywords: vertical vestibulo-ocular reflex; head impulse; semicircular canal

Abbreviations: LARP = left anterior SCC/right posterior SCC (plane); RALP = right anterior SCC/left posterior SCC (plane); SCC = semicircular canal; uPCO = unilateral posterior SCC occlusion; uVD = unilateral vestibular deafferentation; VOR = vestibulo-ocular reflex

Introduction

Vestibular disorders are common and often difficult to characterize, because there are no clinical or laboratory tests for all of the various elements of the vestibular end organ. The vestibular end organ consists of five elements in each ear: the lateral, anterior and posterior semicircular canals (SCCs), which transduce angular acceleration, and the two otolith organs, the utricle and saccule, which transduce linear acceleration. We can test the function of the lateral SCC using thermal or rotational stimuli, but currently there is no test for individual anterior or posterior SCC function in humans.

The main bedside test of SCC function is the head 'impulse' test, which is limited to the lateral canals (Halmagyi and Curthoys, 1988). To perform this test, the examiner rapidly turns the patient's head to the right or the left through 20–30°, and instructs the patient to keep staring straight ahead. The patient's ability to maintain visual fixation is a measure of their horizontal vestibulo-ocular reflex (VOR) gain. If the VOR gain is unity, the compensatory vestibular eye rotation has exactly the same speed as the head impulse, but in the opposite direction, so the patient will be looking straight ahead at the end of the impulse. If the VOR gain is low in one direction, the patient's gaze will be dragged in the direction of the head impulse. Because he had been told to look straight ahead, he will make a voluntary corrective eye movement back to the original fixation point, and it is this late saccadic eye movement that the clinician detects. We have confirmed these clinical findings with head and eye movement recordings, which demonstrate a severe VOR gain deficit (0.2–0.3) during horizontal head impulses directed

toward a lesioned lateral SCC (Cremer *et al.*, 1988). The two lateral SCCs are approximately co-planar with each other (Blanks *et al.*, 1975*a*), and the horizontal head impulse is therefore a 'canal-plane stimulus', because it is a head rotation in the plane of the lateral SCCs. This means that a horizontal head impulse will stimulate the lateral SCCs maximally. Despite the usefulness of the horizontal head impulse test, the corresponding vertical (pitch and roll) impulses have not been as helpful in diagnosing a lesion of individual vertical SCCs. Pitch head impulses are delivered in the sagittal plane about the inter-aural axis, and roll impulses are delivered in the frontal plane about the naso-occipital axis.

By recording head and eye rotation during pitch impulses (with scleral search coils), it is possible to demonstrate a modest reduction in VOR gain (~0.7) following ablation of an individual vertical SCC (Halmagyi *et al.*, 1992; Aw *et al.*, 1996*b*). Aw *et al.* (1996*b*) were also able to identify the side of the lesioned SCC, by analysing the direction of misalignment in the axis of eye rotation compared with the axis of head rotation. This was one of the first demonstrations of vertical SCC dysfunction in humans, but it required precise three-dimensional recordings and complex off-line mathematical analysis.

In this study we used a diagonal canal-plane head impulse stimulus, oriented approximately in the plane of the vertical SCCs, and we were able to test each vertical SCC separately. The vertical SCCs are aligned diagonally in the head, and they form two approximately co-planar pairs: the left anterior/right posterior SCC (LARP) pair, the right anterior/left posterior SCC (RALP) pair (Figs 1 and 2) (Blanks *et al.*, 1975*a*; Curthoys *et al.*, 1977). Because the LARP, RALP and lateral SCC pairs are roughly orthogonal to each other, a head impulse in the plane of one pair will stimulate mainly that pair, and not the other two SCC pairs. Furthermore, the asymmetric response of primary vestibular afferent nerve fibres dictates that the VOR during a canal-plane impulse toward a particular SCC is driven largely by that SCC, and not by its co-planar partner. Thus, for both anatomical and physiological reasons, diagonal canal-plane head impulses test each vertical SCC separately.

Methods
Subjects
Control subjects
We studied nine normal subjects (median age 28 years, range 25–48 years) without any history or clinical signs of vestibular disease.

Unilateral vestibular deafferented (uVD) subjects
We studied seven patients who had undergone surgical vestibular ablation for intractable vertigo, which was attributed to Ménière's disease (four), perilymph fistula (one),
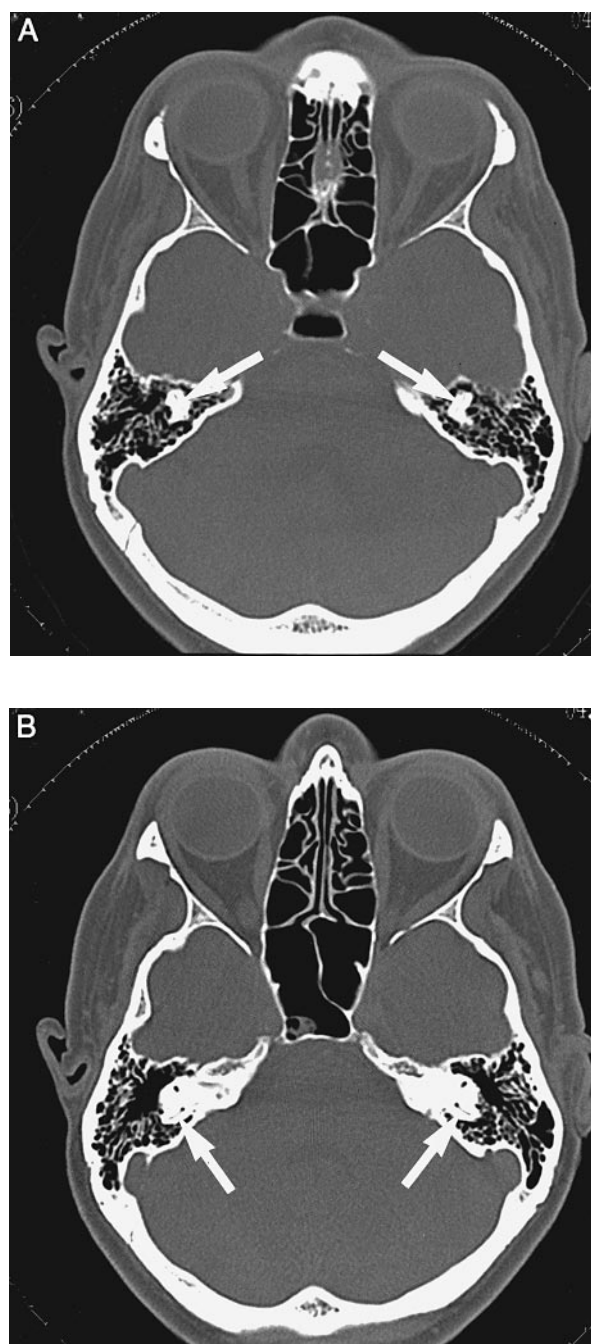
Fig. 1 Axial CT scans of the skull demonstrating that the anterior and posterior SCCs are oriented diagonally within the temporal bones. (**A**) The left and right anterior SCCs are shown (arrows). (**B**) CT scan of the same subject, 4 mm caudal to **A** which shows the left and right posterior SCCs (arrows).

cholesteatoma (one) and intractable paroxysmal positioning vertigo (one, before the widespread use of repositioning manoeuvres and canal occlusion surgery). The median age of the uVD subjects was 65 years (range 43–83 years), and the lesions were right-sided in four patients and left-sided in three. The patients had all recovered from the acute affects of surgery (an average of 8 years before testing, range 36–
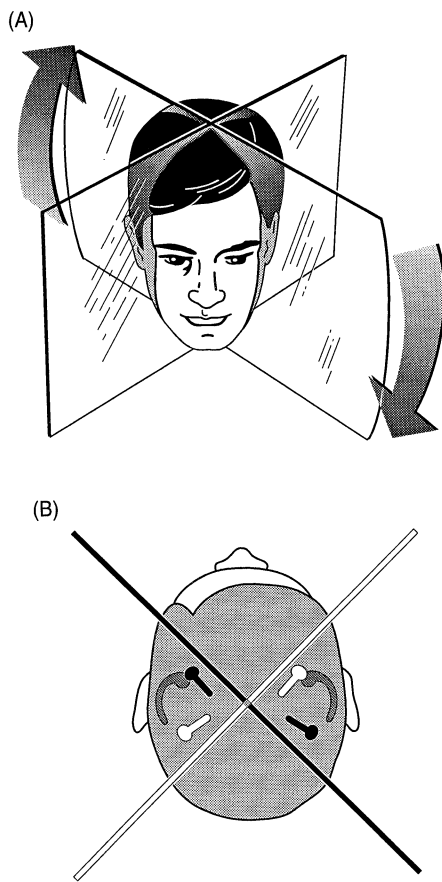
Fig. 2 (**A**) Diagram of the diagonal LARP and RALP planes in which the head impulses were delivered. The arrows show the head about to move in the LARP plane, in the direction of the left anterior SCC. (**B**) Axial view of the head from above, showing the LARP plane (black line) and the RALP plane (white line). The LARP plane is approximately co-planar with the left anterior and the right posterior SCCs, shaded black. The RALP plane is approximately co-planar with the right anterior and left posterior SCCs, which are white.

300 months), and none had significant spontaneous nystagmus (>2°/s) in the dark.

Unilateral posterior SCC occluded (uPCO) patients

We studied three patients following surgical posterior SCC occlusion for intractable benign paroxysmal positioning vertigo. The surgical technique (Pohl, 1996) was similar to that described by Parnes and McClure (1991). The median age of the patients was 66 years (range 58–67 years). The surgery was performed >3 years prior to testing (mean 49 months, range 44–56 months). Two patients underwent left posterior canal occlusion and one had surgery on the right. All had postoperative caloric testing and audiometry, which revealed intact function in the lateral SCC and cochlea, respectively.

Consent

Written, informed consent was obtained from all subjects prior to testing, according to the declaration of Helsinki. The project was supported by the Medical Ethics Committee of the Central Sydney Area Health Service.

Recording system

The recording system was the same as that used by Aw *et al.* (1996*a*, *b*). Three-dimensional head and eye position was measured by the scleral search coil technique described by Robinson (1963) and Collewijn *et al.* (1985), using dual search coils (Skalar, Delft, The Netherlands). The eye coil, which had been calibrated in a Fick gimbal, was placed on the subject's left eye. The head coil was mounted on the nose-piece of a pair of lightweight spectacle frames, which were fastened securely to the subject's head with a velcro strap. We then placed a latex swimming cap on the subject's head to ensure that the spectacle frames did not slip. The subject was seated with the head in the centre of a (1.9×1.9×1.9 m) wooden frame housing the magnetic field coils.

The head position and gaze position (eye-in-space) signals were recovered in three orthogonal planes (yaw, pitch and roll) by phase detection, and passed through anti-alias filters with a bandwidth of 0–100 Hz. The six position signals were sampled at 1000 Hz by an IBM-compatible personal computer running the LabVIEW program (National Instruments, Version 4.0) under Windows 3.1. The recording system has 16-bit resolution and is able to resolve an angular position of 0.1 minute of arc.

Triggering of data sampling was controlled by DAOS software running under TSX-plus, on a PDP-11/73 processor (Digital Equipment Corporation). The PDP-11/73 also drove mirror galvanometers which controlled the position of a red laser fixation spot, which was back-projected onto a screen 94 cm in front of the subject's left eye.

System calibration
In vitro *calibration*

The head and eye search coils were mounted in a perspex Fick gimbal at the centre of the magnetic field. The gimbal was rotated in successive 5° steps from –20° to +20° in each of the yaw, pitch and roll planes. The corresponding output voltages at each angular location were sampled from both search coils simultaneously. The horizontal, vertical and torsional coil signals were divided by the sine of the calibration angle, and a linear regression analysis was performed to determine the gains and offsets. The linear regression coefficient was >0.99 in each case. Cross-talk between the orthogonal signals was <2%. These search coils are not responsive to translations which occur during head impulses (Aw *et al.*, 1996*a*).

Following gimbal calibration, the eye coil was cleaned

with detergent and water to remove proteinaceous material, and then sterilized in bleach for 10 min. The bleach was rinsed off with sterile saline solution.

In vivo *calibration*

The eye coil was calibrated again after it was placed on the eye. The subject was asked to fixate the laser target as it was moved across the screen in 5° horizontal, vertical and oblique steps through a range of ±15°. Although the gimbal calibrations were used in the final data analysis, the *in vivo* calibration served as a check that the eye coil was working well and that there was no significant coil misalignment on the eye.

Experimental protocol
The stimulus

The head impulse is a passive, unpredictable, low-amplitude (15–30°), high-velocity (200–400°/s), high acceleration (2000–4000°/s^2) head rotation (Halmagyi and Curthoys, 1988). For each subject we delivered 20 standard yaw (horizontal) head impulses, which are approximately co-planar with the lateral SCCs, and 40 head impulses approximately co-planar with the vertical SCCs, which we will refer to as 'diagonal' impulses. Diagonal head impulses have not been used previously, and they comprised 20 LARP impulses and 20 RALP impulses (Fig. 2). We define these diagonal planes to be earth-vertical, and to lie midway between the pitch and roll planes.

To deliver the diagonal head impulses, the operator placed one hand on the subject's forehead and the other hand on the occiput, along one diagonal meridian (either LARP or RALP). She then delivered each impulse as a brief, abrupt one-handed push or pull on the head along that meridian, using the other hand as a guide. This one-handed technique was important in eliminating components of horizontal rotation from the head impulse. A good diagonal head impulse comprises equal components of pitch and roll, without any yaw head rotation. The operator was able to view these individual components of angular head velocity on a small LCD screen immediately after each head impulse, in order to maintain quality control of the stimulus. The diagonal impulses were not easy to deliver and the operator was trained extensively on normal subjects. The test was robust, because even if a head impulse did not contain equal amounts of pitch and roll, or if it contained some component of yaw rotation, we were still able to detect absent function in a single SCC. We later plotted the relevant vector component of head and eye velocity for the particular head impulse. For example, during a LARP-plane impulse, only the LARP component of head velocity and the LARP component of eye velocity were plotted, thereby removing any 'out of plane' contribution to the VOR. This will be discussed in more detail later.

The diagonal head impulses were comfortable for all subjects, and none of the 19 subjects sustained any trauma from the test. Our protocol stipulates that the amplitude of the head impulses should not exceed 30°, in order to prevent damage to the cervical spine or vertebral arteries.

During each set of head impulses, the specific direction of the impulse within a canal-plane (left/right, LA/RP or RA/LP) was randomly selected by the operator. The timing of the impulse was determined by a set of dim light-emitting diodes, visible only to the operator, which were activated when the subject's head was repositioned inside a central head position 'window', set to ±1.5° in the yaw, pitch and roll planes. This ensured both that the head position was central at the onset of each impulse and that the timing was unpredictable to the subject.

In addition to these 60 scheduled head impulses, three normal subjects also received 10 roll and 10 pitch head impulses, about the naso-occipital and inter-aural axes, respectively.

At the beginning of the test, each subject was seated so that the left eye was positioned in the centre of the magnetic field. The room lights were dimmed and the subject was instructed to stare at a central red laser spot projected onto a screen 94 cm in front of the subject's left eye. The operator then delivered the head impulses and the subject's task was to maintain visual fixation on the red laser spot throughout the test.

Data analysis

For each head impulse, 1024 ms of data were acquired. Three-dimensional head and 'gaze' (eye in space) position were calculated as Fick angles and then expressed in rotation vectors with roll, pitch and yaw coordinates (Haslwanter, 1995). The rotation vectors were then passively rotated 45° about the yaw (earth-vertical) axis (Appendix, Fig. A1) by a matrix transformation (Appendix, Matrix 2) and expressed in canal-plane coordinates (LARP, RALP and yaw). Orientation of the eye in the head (referred to as 'eye' position) was calculated from the gaze and the head position coordinates (Haslwanter, 1995).

Using head, gaze and eye position data we calculated the three-dimensional angular velocity for head, gaze and eye. The method for calculating angular velocity takes into account the instantaneous head, gaze and eye positions as well as the mathematical differential of the position data (Hepp, 1990). Figure 3 shows some of our methods for data representation. Eye position and eye velocity have been inverted to allow comparison of the eye rotation response with the head rotation stimulus. If a subject had an ideal VOR with zero latency, the eye rotation traces would be superimposed on the head rotation traces. Head and eye position during RA and LP direction head impulses are plotted against time in row 1, and the corresponding angular velocity for each head impulse is plotted against time in row 2. We also plotted eye velocity versus head velocity for each head impulse (row 3), from
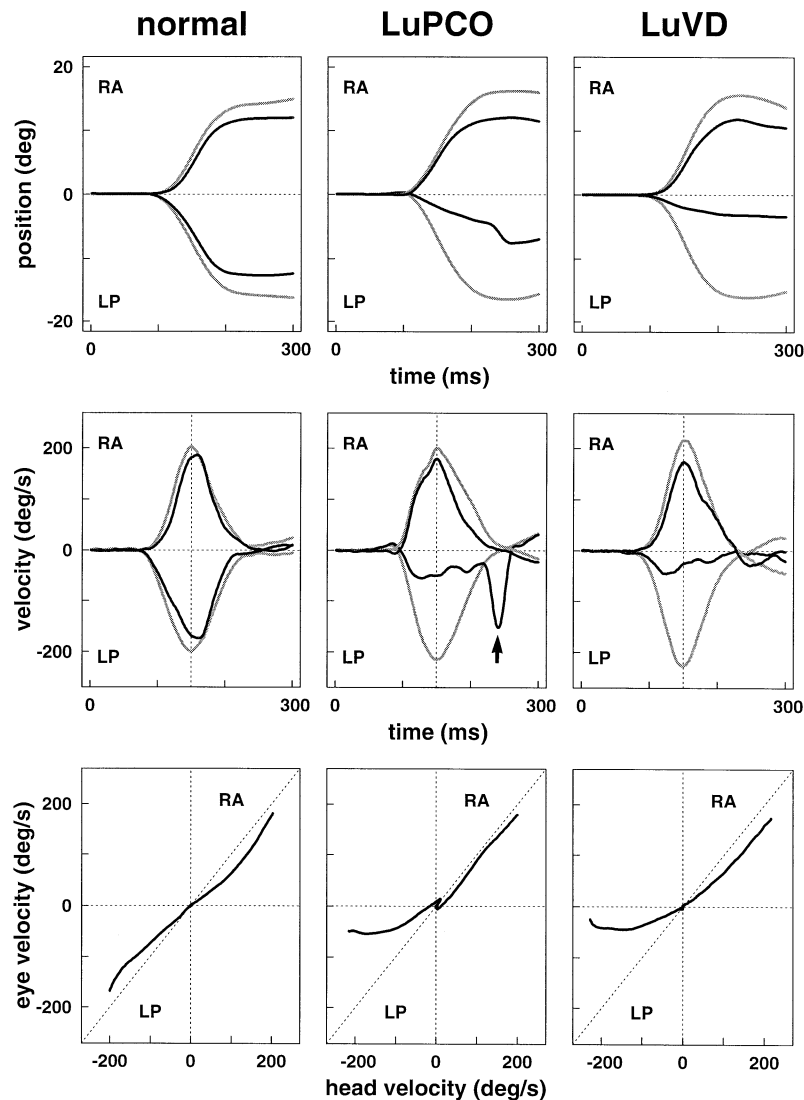
Fig. 3 Format of data representation in this study. Head impulses from one normal subject are shown in the left column (column 1), one patient following left uPCO (column 2) and one patient following left uVD (column 3). Head position and head velocity are displayed in grey. Eye position and eye velocity are displayed in black, and have been inverted for ease of comparison with the head rotation stimuli. Row 1 (top): head and eye position during one head impulse in the direction of the right anterior SCC (RA, top), and one head impulse in the direction of the left posterior SCC (LP, bottom), for each subject. Row 2: head and eye velocity plotted against time, corresponding to the head impulses in row 1. The vertical broken lines show the point of maximum head velocity. The sharp peak in eye velocity in row 2, column 2 (indicated by an arrow), is a 'catch-up' saccadic eye movement. Row 3: eye velocity plotted against head velocity for the same head impulses. A perfect VOR would yield an eye velocity trace superimposed on the diagonal line. The data are taken from the onset of the head impulse to maximum head velocity. For the two patients (columns 2 and 3), the VOR is deficient for head impulses in the LP direction.

the onset of the head impulse to the point of maximum head velocity (which is shown as the broken vertical line in row 2). In this representation, an ideal VOR would result in the eye velocity trace being superimposed on the diagonal broken line (in row 3). This early data plotted in row 3 corresponds to the first 50–100 ms of the head impulse, and was used in the analysis of VOR gain, because it is likely to be a true reflection of vestibular function. In this early phase, the short-latency VOR is active, but other visual following systems such as pursuit, optokinetic, cervico-ocular and predictive oculomotor systems are not operating, owing to their relatively long latencies (Halmagyi *et al.*, 1990).

The VOR gain was then calculated for each subject by dividing the length of the total eye velocity vector (eye speed) by the length of the head velocity vector (head speed) (Haslwanter, 1995; Aw *et al.*, 1996*a*).
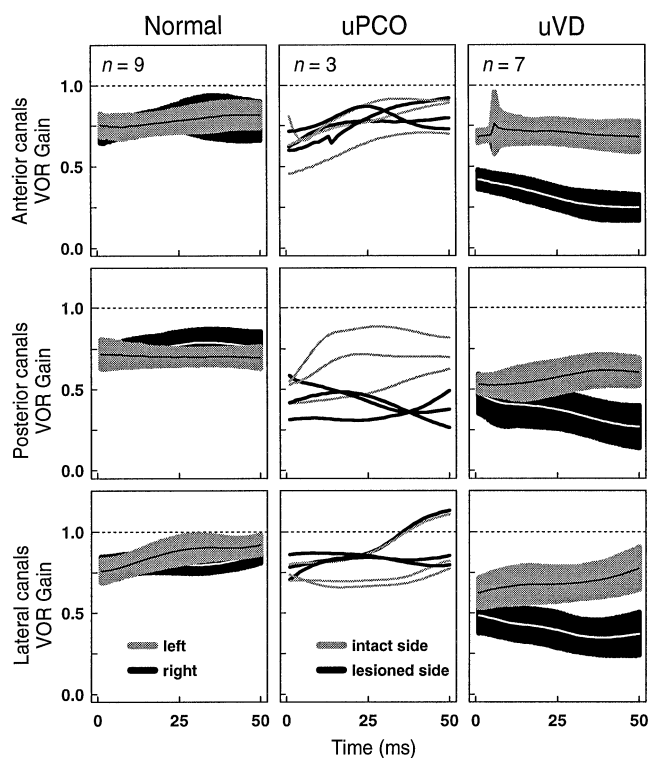
Fig. 4 Averaged VOR speed gain for nine normal subjects (column 1), three uPCO subjects (column 2) and seven uVD subjects (column 3). Instantaneous VOR gain is calculated for a 50 ms period prior to maximum head velocity. VOR gain during the first 10–20 ms of the head impulse is not shown because of erratic results due to dividing by small numbers. VOR gain is plotted for head impulses directed toward each anterior SCC (row 1), each posterior SCC (row 2) and each lateral SCC (row 3). For normal subjects and uVD subjects, the average VOR gain and 95% confidence intervals are plotted, and for uPCO subjects the mean gain for each of the three subjects is plotted separately. For normal subjects the VOR gain during head impulses toward the right ear is plotted in black, and the VOR gain during head impulses toward the left ear is plotted in grey. In uVD and uPCO subjects, the VOR gain during head impulses toward the lesioned side is plotted in black, and the VOR gain during head impulses toward the intact side is plotted in grey.

Results

VOR in normal subjects

Horizontal head impulses

The gain of the normal VOR during horizontal head impulses reaches a mean of 0.9 (±0.1, 95% confidence interval) at maximum head velocity (Fig. 4). In other words, during horizontal head impulses the eye rotates in the opposite direction to the head, with approximately the same speed.

Diagonal head impulses (LARP and RALP)

The average eye velocity responses (±95% confidence intervals) during diagonal head impulses in normal subjects are plotted against head velocity as the grey reference band in Fig. 7 (below). The VOR gain during diagonal head impulses is ~0.7–0.8 (Fig. 4). To investigate this deficient
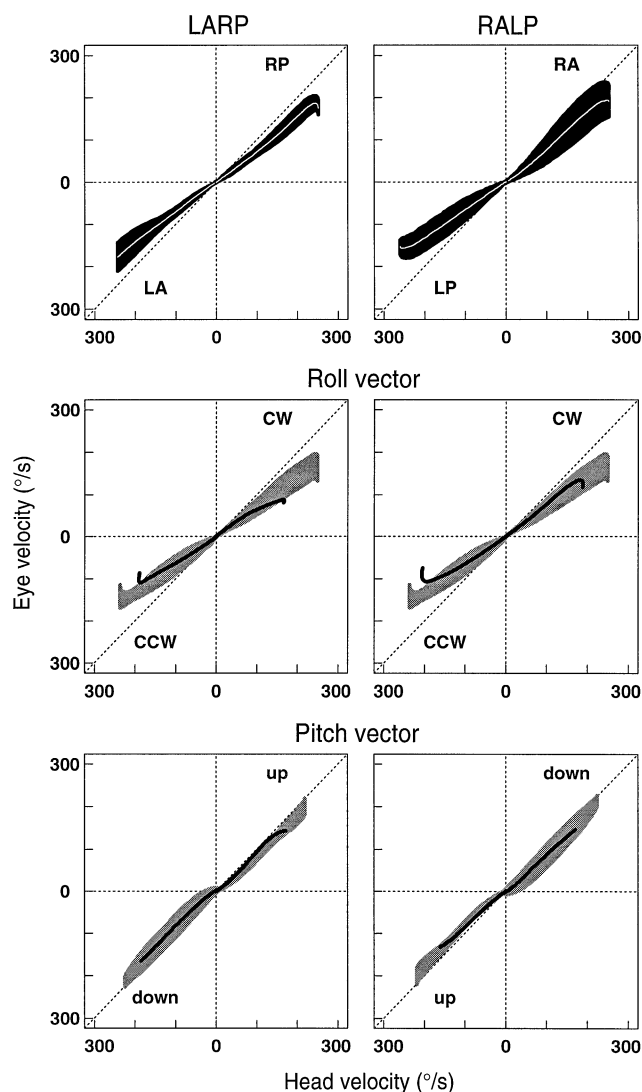
Fig. 5 Comparison of LARP- and RALP-plane with roll- and pitch-plane head impulses in three normal subjects. In all graphs eye velocity is plotted against head velocity. Row 1 shows the average (±1 SD) for LARP- and RALP-plane impulses. Rows 2 and 3 show the average roll and pitch component vectors of eye versus head velocity during LARP and RALP impulses (black lines). The grey bands in rows 2 and 3 represent the average (±1 SD) eye versus head velocity during roll- and pitch-plane impulses in the same three normal subjects. The roll and pitch VOR appear constant, irrespective of whether the stimulus is a purely roll or pitch rotation, or whether the roll and pitch components are part of a more complex (LARP or RALP) stimulus.

canal-plane response in normal subjects, we resolved the LARP and RALP head and eye velocities into their separate pitch and roll vector components for three normal subjects (plotted as black lines in Fig. 5). The gain of the roll component was ~0.6, and the gain of the pitch component was ~0.9. This suggests that the deficiency in the diagonal canal-plane responses was due to a deficit in the gain of the roll component. We tested the same three subjects with pure roll and pure pitch head impulses, and demonstrated similar
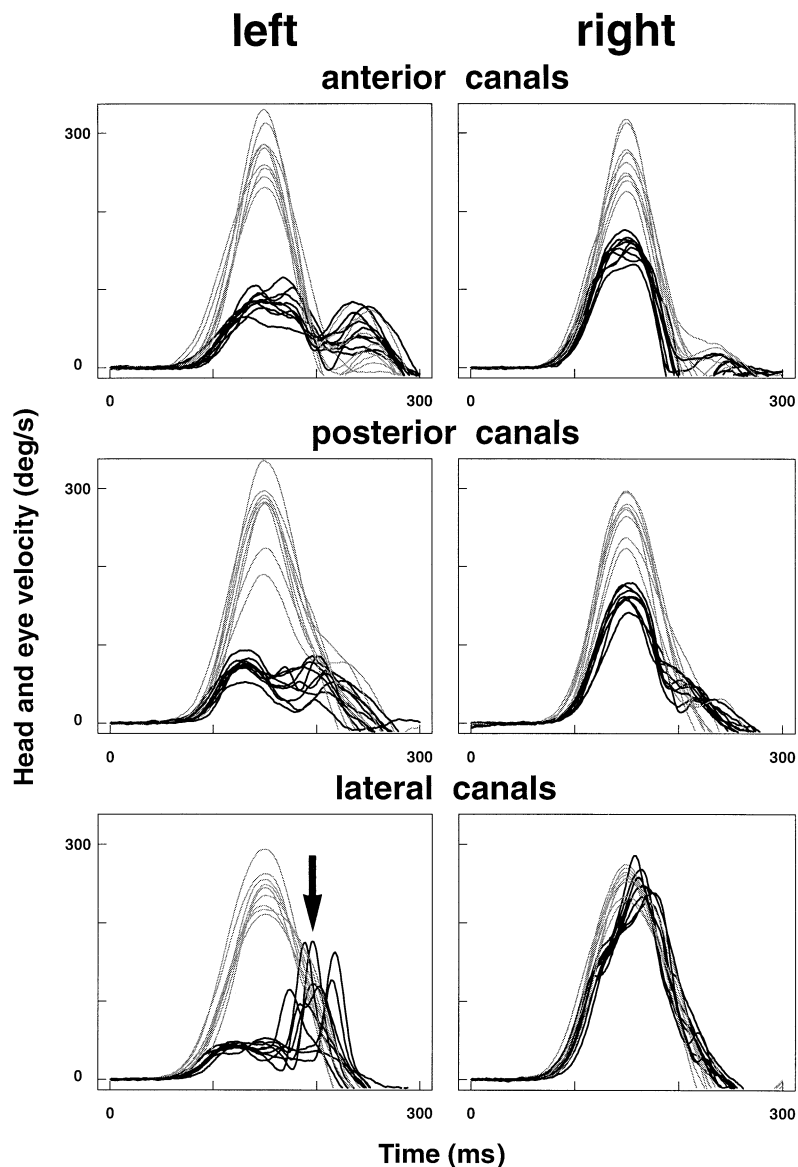
Fig. 6 Head velocity (grey) and eye velocity (black) are plotted against time; these responses are from a subject with a left vestibular neurectomy. Eye velocity has been inverted for ease of comparison with the head velocity stimulus. A total of 54 head impulses are shown, comprising nine head impulses toward each of the six SCCs. For head impulses directed toward the left anterior, left posterior and left lateral SCCs, the VOR is markedly deficient. The sharp peaks in the eye velocity traces in row 3, column 1 (indicated by an arrow), are 'catch-up' saccades.

responses to the roll and pitch components of the diagonal VOR (grey bands in Fig. 5).

VOR in uVD patients

The VOR during head impulses directed toward each of the three deafferented SCCs was consistently deficient. Figure 6 shows nine head impulses toward each SCC in one representative patient following left vestibular neurectomy. The VOR during head impulses toward affected SCCs on the left side was poor, but the responses toward the intact right side were within normal limits. In addition, the eye velocity response reached a maximum of 50–100°/s and

varied little for a wide range of stimulus magnitudes, from maximum head velocity of 180–320°/s. The VOR gain during head impulses toward the lesioned side was ~0.2–0.3 at maximum head velocity, regardless of which SCC was being tested (Fig. 4). Figure 7 (row 2) shows averaged eye velocity plotted against head velocity (in black), with 95% confidence intervals. There was a significant difference between the responses of the uVD subjects when tested toward their lesioned side and those in normal subjects (plotted in grey). For head impulses toward the intact side, the VOR gain was ~0.55–0.70, which overlaps the normal range at the low end.

Most subjects with a VOR deficit used saccades to refixate the target lost during the head movement. The subject shown
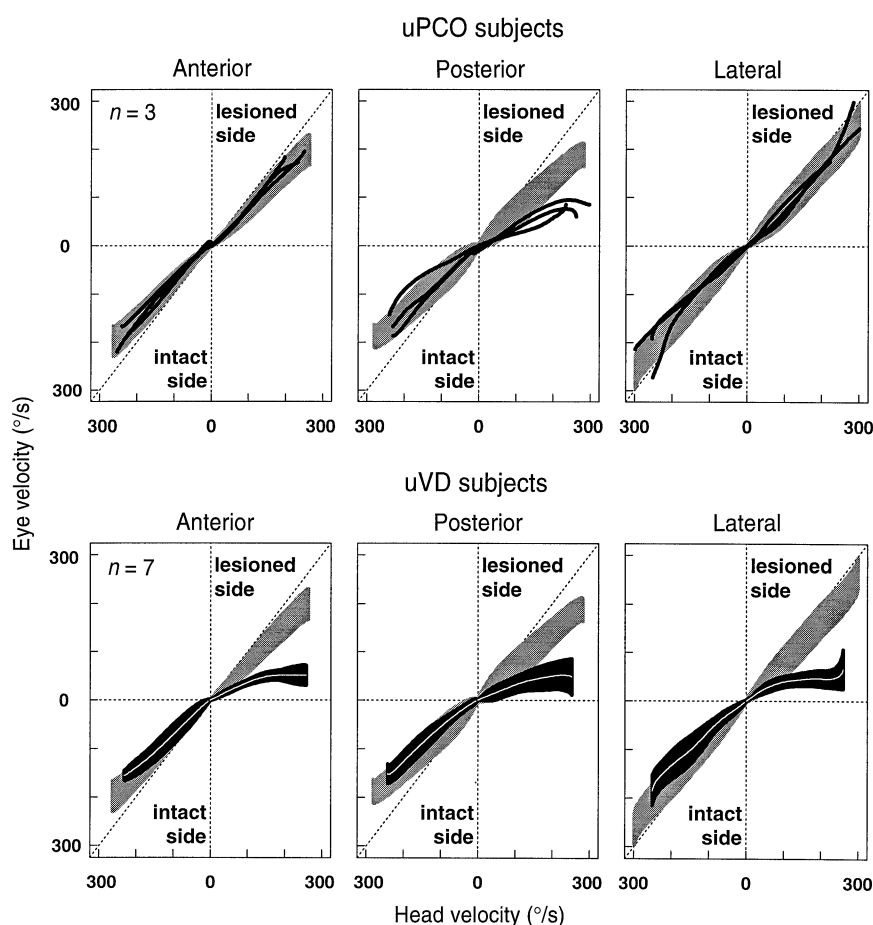
Fig. 7 Averaged eye velocity versus head velocity is plotted in black for the three uPCO subjects (row 1), and the averaged eye velocity (±95% confidence intervals) is plotted for uVD subjects (row 2). The VOR during head impulses directed toward the anterior SCCs is shown in column 1, toward the posterior SCCs (column 2), and toward the lateral SCCs (column 3). The normal range (average ±95% confidence intervals) is shown as a grey band in each plot. The VOR during head impulses directed toward the lesioned side is shown on the right side of each graph, and for head impulses toward the intact side on the left side of each graph. The eye velocity is limited to 50–100°/s during head impulses in the direction of any lesioned SCC.

in Fig. 6 used early saccades (indicated by an arrow) during yaw impulses, with latencies of 110–150 ms. Other subjects used early saccades during diagonal head impulses. Most subjects also used late saccades (>500 ms after the head impulse) to refixate the target.

VOR in uPCO patients

There was a clear deficit in VOR gain (0.3) for head impulses directed toward the lesioned posterior SCC, and the VOR gain during head impulses toward all other SCCs was normal (Fig. 4). Figure 8 shows nine head impulses directed toward each SCC in one subject following left posterior SCC occlusion. During head impulses directed toward the five intact SCCs, the eye velocity response matches the head velocity stimulus. Yet for head impulses in the direction of the inactivated posterior SCC, the eye velocity response is markedly deficient. The eye velocity reaches a maximum of 50–100°/s during head impulses with a maximum velocity

of 220–320°/s. In addition, there are early compensatory saccadic eye movements with latencies of 100–200 ms during head impulses toward the lesioned SCC.

Discussion
VOR in normal subjects
Horizontal head impulses
The gain of the normal VOR during horizontal head impulses reached 0.9 (±0.1, 95% confidence interval) at maximum head velocity. Therefore the eye rotation response compensates, in magnitude, for the head impulse stimulus. This confirms previous data for the horizontal VOR (Cremer *et al.*, 1988; Halmagyi *et al.*, 1990; Aw *et al.*, 1996a).

Diagonal head impulses
The VOR in response to diagonal plane head impulses does not mirror the stimulus as faithfully as it does for yaw plane
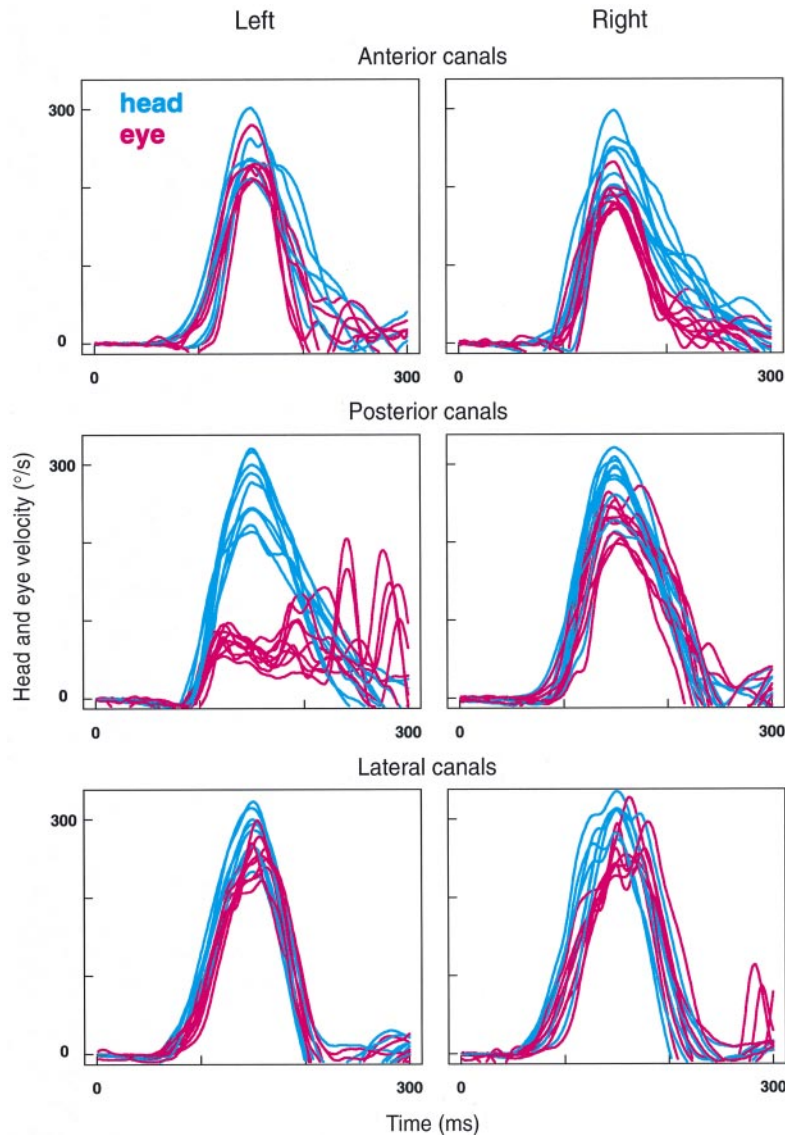
Fig. 8 Head velocity (blue) and eye velocity (red) are plotted against time in one subject following a left posterior SCC occlusion. Eye velocity has been inverted for ease of comparison with the stimulus (head velocity). A total of 54 head impulses are shown, comprising nine head impulses toward each of the SCCs. During head impulses directed toward the inactivated left posterior SCC the VOR is markedly deficient. Yet head impulses directed toward any of the five intact SCCs elicit a normal response. The sharp peaks in the eye velocity traces during head impulses directed toward the left posterior SCC are 'catch-up' saccades, which partially compensate for the deficient vestibular response.

stimuli. The gain of the response is only 0.7–0.8. This deficiency appears to be due to a physiological limitation of the roll VOR. The diagonal LARP and RALP planes lie midway between the pitch and roll planes, and the VOR during diagonal head impulses should be the vector sum of the pitch and roll VORs. The pitch VOR gain in normal subjects is 0.9–1.0, and the roll gain is 0.6–0.7 (Ferman *et al.*, 1987; Leigh *et al.*, 1989; Crawford and Vilis, 1991; Tweed *et al.*, 1994; Aw *et al.*, 1996*a*). We found the diagonal VOR gain to be between these values. To confirm this, we resolved the head and eye velocities of three normal subjects

into their pitch and roll components, and the VOR gains were ~0.9 and 0.6, respectively (black traces in Fig. 5).

In addition, we delivered 10 pitch-plane and 10 roll-plane impulses in each of these three subjects (grey bands in Fig. 5), and the resulting VOR closely matched the pitch and roll components of the response during diagonal impulses. In other words, the VOR in response to a head impulse which deviates from the pitch and roll planes is the vector sum of the responses to its pitch and roll components. Crawford and Vilis (1991) reached a similar conclusion after rotating alert monkeys in the LARP and RALP planes. Fetter *et al.*

(1994) confirmed this in humans by rotating their subjects sinusoidally in the LARP and RALP planes and finding that the VOR was a vector sum of the pitch and roll components, with the roll gain being two-thirds of the yaw or pitch gains. This has also been verified experimentally in canal-plugged monkeys (Yakushin *et al.*, 1995). This concept is not universally endorsed, since a study by Tweed *et al.* (1994) in humans showed that the roll VOR gain in response to a pure roll stimulus (0.37) was less than the gain when the torsional component was a small component of a predominantly yaw or pitch movement (0.52). This inconsistency might be due to the different stimuli used. Tweed *et al.* (1994) used sinusoidal oscillation with a maximum speed of 37.5°/s and torsional components of only 10°/s in their diagonal axis paradigm, whereas our head impulses reached velocities of 300–400°/s. It is possible that during the low-velocity stimulation of 10°/s, which is generally considered to be at the low end of the operating range of the angular VOR, other non-SCC systems, such as otolithic counter-roll, could boost the response.

VOR in uVD patients
'Off' direction VOR

The VOR during head impulses directed toward any one of the three deafferented SCCs was uniform (Fig. 7), and the gain was ~0.2–0.3 at maximum head velocity (Fig. 4). A similar VOR gain deficit has been shown in the horizontal plane, reflecting a deficiency of lateral SCC function on the lesioned side (Cremer *et al.*, 1988; Halmagyi *et al.*, 1990, 1991; Tabak and Collewijn, 1995; Aw *et al.*, 1996b; Haslwanter and Fetter, 1996). However, such a substantial gain deficit has not been shown for the anterior or posterior SCCs. Diagonal LARP- and RALP-plane impulses clearly detect a complete lesion of an individual anterior or posterior SCC.

Specificity of diagonal and yaw head impulses
Analysis of the stimulus plane

The specificity of diagonal head impulses (their ability to test individual SCC pairs) is predominantly due to the plane of head rotation being approximately co-planar with the SCC pair being tested. The specificity is also due to analysis of the data in the LARP and RALP planes. The LARP, RALP and lateral SCCs operate in a push–pull fashion, as three co-planar pairs. Because the SCC pairs are approximately orthogonal, a head impulse in the plane of one pair will have minimal stimulatory or disfacilitatory effect on the other two SCC pairs, both of which are out of the plane of head rotation. For any given head rotation, the amount of acceleration transduced by a particular SCC is determined by the geometric 'projection' of that SCC into the plane of the head rotation (Estes *et al.*, 1975). For example, a head rotation in the plane of a SCC causes maximum stimulation

of that SCC, but a head rotation in the plane perpendicular to a SCC causes no activation. Intermediate projections cause intermediate SCC activation, as determined by the cosine of the angle between the plane of head rotation and the plane of the SCC (Blanks *et al.*, 1975b; Bohmer *et al.*, 1985). This principle has been validated in SCC-plugged monkeys, whose VOR was shown to be a simple vector addition of the VOR generated by all six SCCs, weighted by their projection into the plane of head rotation (Yakushin *et al.*, 1995).

We calculated the relative contribution of each SCC to the canal-plane head impulses (Appendix). Using the canal-plane data from Blanks *et al.* (1975a), and geometrically projecting the data into LARP and RALP planes, we calculate that during a head impulse in the LA direction, the left anterior SCC is stimulated by 0.99 of the total head acceleration, the right posterior SCC is disfacilitated by 0.93, the left lateral SCC is stimulated by 0.37, and the remaining SCCs each transduce <0.15 of the total head acceleration. Similarly, during a head impulse in the RA direction, the right anterior SCC is stimulated by 0.99 of the total head acceleration, the left posterior SCC is disfacilitated by 0.93, the right lateral SCC is stimulated by 0.37, and the remaining SCCs each transduce <0.15 of the total head acceleration. During a leftward yaw impulse with the head erect, the left lateral SCC is stimulated by 0.91 of the total head acceleration, the right lateral SCC is disfacilitated by 0.91, the right posterior SCC is stimulated by 0.32, the left posterior SCC is disfacilitated by 0.32, and the anterior SCCs each transduce <0.02 of the total head acceleration. This selectivity of yaw head impulses accounts for their usefulness as a clinical test of lateral SCC function (Halmagyi and Curthoys, 1988).

Analysis of the data in diagonal planes

Further specificity of canal-plane impulses is derived from the plane of analysis of the head and eye rotations. We plotted the LARP component of head and eye velocity during LARP-plane impulses, the RALP component during RALP impulses and the yaw component during yaw impulses (Figs 6, 7 and 8). By plotting only the in-plane vector components of head and eye velocity, the data are not confounded by any out-of-plane components of the head velocity stimulus. Because the head impulses were delivered manually, they did not always lie exactly in a diagonal plane, and contained elements of yaw rotation or unequal components of pitch and roll. Yet if head and eye velocity are plotted in the LARP or RALP planes, these errors become unimportant, and a single defective SCC can be clearly identified (Fig. 8).

The physiological reason for this selectivity depends on the principle that each SCC generates eye rotations about an axis orthogonal to its own plane (Suzuki *et al.*, 1964). In other words, the posterior SCCs generate predominantly downward and torsional slow phase eye rotation, and the lateral SCCs generate mainly horizontal eye rotation with a smaller torsional component. For example, during an ideal LA-direction head impulse, even though the left lateral SCC

is stimulated by 0.37 of the total head acceleration, the resulting eye rotation generated by this SCC is largely horizontal. If we then plot the LARP component of eye velocity versus the LARP component of head velocity, the contribution of the left lateral SCC becomes very small. Using Robinson's methods (1982), and published VOR gain values (Aw *et al*., 1996*a*), we calculated that the left lateral SCC projects to the left medial rectus with a relative efficacy of 1.02, compared with 0.11 to the left superior rectus and 0.15 to the left superior oblique (Appendix). By projecting the plane of action of each extra-ocular muscle into the LARP plane, it appears that only the left superior rectus has a significant action in this plane, with a relative efficacy of 0.94, compared with 0.01 for the left medial rectus and 0.13 for the left superior oblique. In other words, even though the left lateral SCC is stimulated by 0.37 of the total LARP-plane head acceleration, the contribution of the left lateral SCC to LARP plane eye rotation is <0.04 (Appendix). This technique of excluding out-of-plane eye velocity, and therefore minimizing the contribution of out-of-plane SCCs, becomes even more important if a head impulse does not lie exactly in the LARP plane.

Even though plotting the component of head and eye velocity which corresponds to the intended plane of the head impulse increases the specificity of the test, the diagnostic ability of the diagonal impulses does not depend on analysing the data in canal planes. The calculation of VOR speed gain (Fig. 4) exposes the deficient SCCs, and is derived not from a particular component of head or eye velocity but from the total speed of eye rotation divided by the total speed of head rotation (Aw *et al*., 1996*b*).

Sensitivity of LARP, RALP and yaw head impulses

The sensitivity of canal-plane head impulses, in detecting total loss of an individual SCC, depends not on the anatomy but on the physiology of the VOR during high-acceleration stimuli. Rotating subjects in the LARP or RALP planes at low accelerations will expose only a small VOR gain asymmetry, despite complete functional loss of a single SCC (Kanayama *et al*., 1995).

Physiology of primary and secondary vestibular neurons

Ewald's Second Law (Ewald, 1892) implies that each SCC transduces angular acceleration in a non-linear manner. Yet the normal VOR is linear because each SCC is oppositely polarized to its co-planar partner, and the summed output is linear. Each SCC has a preferred 'on' direction response, and an opposite 'off' direction response which has a lower gain and which can be saturated. There is very little data on the behaviour of primary vestibular afferent neurons during high-acceleration stimuli, but Goldberg and Fernandez (1971)

have made neural recordings during moderate accelerations up to $150°/s^2$. They demonstrated that during 'on' direction stimulation of any individual SCC in squirrel monkeys, the firing of primary vestibular afferent neurons can exceed 350 spikes per second from a tonic resting level of 90 spikes per second. The upper limit of firing was not reached at these accelerations. During acceleration in the 'off' direction, firing in primary afferent neurons decreased from 90 spikes per second to zero, with a sensitivity less than that in the 'on' direction. It is likely to be a combination of the reduced neural sensitivity and neural silencing during high acceleration, 'off' direction stimuli, that explains the VOR asymmetry described by Ewald's Second Law. This non-linearity is propagated to, and probably enhanced in, vestibular nucleus neurons which receive input from one co-planar pair (Shinoda and Yoshida, 1974). Reisine and Raphan (1992) found that LARP or RALP plane vestibular nucleus neurons were less sensitive to contralateral (off-direction) acceleration than to ipsilateral (on-direction) acceleration and, more importantly, the contralateral response saturated at high stimulus accelerations. These secondary vestibular neurons then form excitatory connections predominantly with neurons of a single extra-ocular muscle in each eye whose action is roughly co-planar with the SCC driving the response, but they also synapse with other agonist ocular motor neurons to a lesser extent (Ezure and Graf, 1984; Graf and Ezure, 1986; Baker and Peterson, 1991). The VOR is therefore largely driven by the peripheral and central vestibular system ipsilateral to the head acceleration. In other words, the vestibular response to head acceleration in the direction of the right anterior, right posterior or right lateral SCCs is primarily driven by the right-sided SCCs and the right vestibular nuclei, with a weaker contribution from the SCCs on the left side. Under normal circumstances, this non-linearity in the VOR is overcome by co-planar vestibular nucleus neurons on opposite sides of the brainstem working synergistically to ensure that the VOR is symmetric regardless of the direction of head acceleration.

In uVD subjects the VOR in any plane is driven solely by the three SCCs on the intact side. It should therefore be possible to expose the inherent asymmetry of each remaining SCC by using a high-acceleration, 'off'-direction, saturating stimulus. This has been shown for the lateral SCCs only. Horizontal head impulses, with accelerations up to $4000°/s^2$, have demonstrated a permanent, severe deficit of lateral SCC function in patients following uVD (Cremer *et al*., 1988; Halmagyi *et al*., 1990; Tabak and Collewijn, 1995; Aw *et al*., 1996*b*; Fletcher *et al*., 1996). In contrast, other studies in humans using lower acceleration stimuli on rotational chairs have failed to detect such a substantial VOR deficit (Honrubia *et al*., 1982; Olson and Wolfe, 1984; Black *et al*., 1989). Paige (1983) and Fetter and Zee (1988) demonstrated in primates that the horizontal VOR following inactivation of a single lateral SCC was symmetrical for low acceleration stimuli, and only became deficient for high acceleration stimuli in the direction of the inactivated SCC. This is

presumably because low velocity rotational stimuli, such as those used in the routine clinical testing of patients, lack sufficient acceleration to silence the primary vestibular neurons on the intact side, in contrast to the horizontal head impulses and high acceleration rotations. Although low velocity stimuli cannot reliably be used to assess the integrity of individual SCCs, they do provide useful clinical information about the mid-to-low frequency responses of the VOR, and about central velocity storage.

Kanayama *et al.* (1995) studied three patients following uPCO and one patient following posterior SCC nerve section. The subjects sat in a large gimbal which was oscillated sinusoidally in the diagonal LARP and RALP planes, at a frequency of 0.55 Hz. Six months after surgery there was a modest (16%) asymmetry in the pitch component of the diagonal VOR in the plane of the lesioned SCC, compared with a 5% asymmetry for normal subjects. The torsional component of the LARP and RALP responses was highly variable. This failure to demonstrate a substantial deficit in posterior SCC function after surgical inactivation was probably due to inadequate stimulus acceleration and failure to silence primary afferents from the contralateral anterior SCC. Another problem with the stimulus was its predictability, allowing non-vestibular oculomotor systems, such as predictive vertical eye movements, to play a role. Fetter and Dichgans (1996) rotated 16 patients with vestibular neuritis in the LARP, RALP and yaw planes and found variable results. From the rotation axis of the spontaneous nystagmus, they concluded that both the lateral and anterior SCCs were deficient in many of their patients. Yet 14 out of 16 patients showed only modest asymmetry, or no asymmetry at all, in the VOR during LARP and RALP rotation. Again, the reason for this might be the low stimulus velocities used. Another possible explanation is that, because the anterior SCC lesions were pathological and not surgical, they might have been incomplete.

We used head impulses to examine the anterior, posterior and lateral SCCs, and we showed that in uVD subjects, the off-direction VOR is remarkably similar for any SCC being tested. This is probably because the behavior of primary afferent neurons from each SCC is uniform (Goldberg and Fernandez, 1971). During all head impulses in the direction of a deafferented SCC, there appears to be an eye velocity response limit of ~50–75°/s, regardless of whether the maximum head velocity stimulus is 225°/s or 350°/s (Fig. 6). This eye velocity limit might reflect the neural saturation as primary vestibular neurons from the intact co-planar SCC are silenced.

Comparison with pitch and roll head impulses

Previous efforts to study the anterior and posterior SCCs with the head impulse test have used pitch and roll impulses. Following uVD, the VOR gain during pitch-up and pitch-down impulses is symmetrical (0.5–0.9), regardless of whether the left or right vertical SCCs have been ablated

(Halmagyi *et al.*, 1992; Aw *et al.*, 1994, 1996*b*; Fletcher *et al.*, 1996; Haslwanter and Fetter, 1996). Analysing VOR gain during pitch head impulses is neither a sensitive nor a specific indicator of vertical SCC function in the uVD patient (Fig. 9). On the other hand, VOR gain during roll head impulses can indicate the side of the lesion, but does not provide any information about the function of individual SCCs (Fig. 9). Roll impulses elicit a severely attenuated VOR gain toward the lesioned side (0.2–0.3) versus 0.6 toward the intact side (Aw *et al.*, 1996*b*; Haslwanter and Fetter, 1996). Using detailed off-line three-dimensional analysis of the axes of head and eye rotation, Aw *et al.* (1996*b*) were able to infer that an individual anterior or posterior SCC was defective, since the eye rotation axis deviated toward the lesioned side. However, this result is not apparent to the examiner performing the test, nor is it evident from looking at the VOR gain during each head impulse. The reason for this is that all four vertical SCCs participate in pitch head impulses and all six SCCs participate in roll impulses, so that the effect of any one or two defective SCCs is masked by the remaining intact SCCs. Compared with pitch and roll head impulses, diagonal head impulses accentuate the VOR gain deficit due to loss of a single vertical SCC.

We calculated the contribution of individual SCCs to pitch and roll head impulses using published canal-plane coordinates in humans (Blanks *et al.*, 1975*a*) projected onto the pitch and roll planes (Appendix). During a pitch-down head impulse each anterior SCC is stimulated by 0.75 of the total head acceleration stimulus, each posterior SCC is disfacilitated by 0.56, and each lateral SCC is stimulated by 0.16. Similarly, during a roll-clockwise head impulse, the right anterior SCC is stimulated by 0.65 of the total head acceleration, the right posterior SCC is stimulated by 0.76, the right lateral SCC is stimulated by 0.37, and the left-sided SCCs are disfacilitated by corresponding amounts. In other words, pitch head impulses test all four vertical SCCs in normal subjects and the two remaining vertical SCCs in uVD subjects, and roll head impulses test all six SCCs in normal subjects and the three remaining SCCs in uVD subjects. For these anatomical reasons, pitch and roll head impulses have not been as useful in the clinic as horizontal head impulses in testing the function of individual SCCs.

Could the failure to silence primary vestibular afferent neurons from the intact vertical SCCs also have contributed to the failure of pitch impulses to detect a severe deficit in vertical VOR gain following uVD (Fig. 9)? The answer is probably yes. It is likely that the intact anterior SCC afferents are silenced during pitch-up impulses, and that the intact posterior SCC afferents are silenced during pitch-down impulses. From the canal orientations, we calculated that during a typical pitch-down head impulse with a maximum acceleration of $2500°/s^2$, the intact anterior SCC is being stimulated by a component equal to $1883°/s^2$ and the intact posterior SCC is being disfacilitated by $1403°/s^2$. Tabak and Collewijn (1995) demonstrated that head impulses with a maximum acceleration of $700–1300°/s^2$ were sufficient to
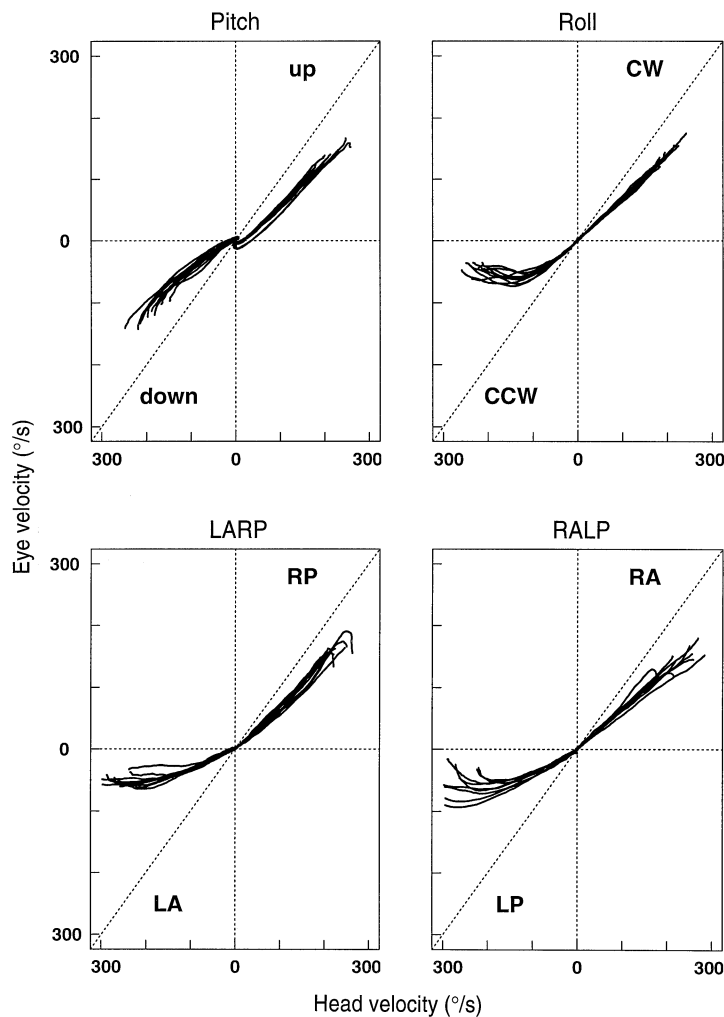
Fig. 9 Comparison of pitch-, roll-, LARP- and RALP-plane head impulses in two patients following left vestibular neurectomy. Eye velocity is plotted against head velocity for 10 head impulses in each direction. The VOR during pitch-up and pitch-down impulses is modestly reduced and symmetric. The VOR during roll-CCW (counter-clockwise) is markedly reduced. During LARP- and RALP-plane impulses toward the lesioned left anterior and left posterior SCCs, the VOR is markedly deficient. The pitch- and roll-impulse data were kindly provided by Dr Aw (Aw *et al.*, 1996*b*).

expose severe asymmetry in the function of a single lateral SCC. This implies that these accelerations were sufficient to silence the intact primary vestibular afferent nerve fibres during head rotation toward the lesioned side. Therefore, during a pitch-down head impulse the primary afferents from the intact posterior SCC are probably being silenced, and at the same time the intact anterior SCC is being stimulated in its 'on' direction by an acceleration of $1883°/s^2$, and it is capable of generating a VOR gain of ~0.7 (Aw *et al.*, 1996*b*). Similarly during a pitch-up head impulse, primary afferents from the intact anterior SCC are probably being silenced, while the intact posterior SCC is being stimulated in its 'on' direction by an acceleration of $1403°/s^2$. The simultaneous 'on'-direction stimulation of one SCC masks the effects of the 'off'-direction response from the other vertical SCC on the intact side, rendering pitch impulses ineffective in detecting a severe VOR gain deficit in the uVD patient.

Similar analysis reveals that a typical roll head impulse in a uVD patient toward the lesioned side disfacilitates the intact anterior SCC with an acceleration of $1630°/s^2$, the intact posterior SCC with $1893°/s^2$ and the intact lateral SCC with $913°/s^2$. These stimuli are in the 'off' direction for all three SCCs, since all SCCs on one side contribute to the ipsilateral roll VOR. This explains why there is a severely limited roll VOR after uVD (Fig. 9). It also explains why the roll head impulse is a sensitive test of unilateral vestibular failure, but not a specific test of any individual SCC.

It is interesting that the roll VOR toward the lesioned side was similar to the LARP and RALP VOR toward the lesioned side in two patients following left-sided uVD (Fig. 9). In other words, the result of silencing the primary afferents from three SCCs (during roll impulses), is not different from silencing the primary afferents from one SCC (during LARP and RALP impulses).

The VOR in uPCO patients

There was a clear deficit in VOR gain (0.3) for head impulses directed toward the lesioned posterior SCC, with the VOR gain during impulses toward all other SCCs falling within normal limits. The magnitude of the deficit in uPCO patients was similar to that found during head impulses toward the lesioned posterior SCC in uVD patients. This demonstrates the ability of LARP and RALP head impulses to detect a lesion in a single SCC, when the remaining five SCCs are normal. Previous studies using pitch and roll head impulses (Aw *et al.*, 1996*b*) and using low velocity LARP and RALP rotation (Kanayama *et al.*, 1995) have shown only a modest VOR gain deficit in uPCO subjects.

Use of LARP and RALP impulses as a clinical test

LARP and RALP canal-plane head impulses expose a severe VOR gain deficit in the direction of a lesioned anterior or posterior SCC, much the same as the horizontal head impulses do for the lateral SCCs. Horizontal head impulses have gained acceptance as a useful bedside and laboratory investigation, and we believe that LARP and RALP head impulses complement the horizontal impulses, forming a comprehensive battery of canal-plane tests. Potentially, this battery will be useful in assessing all patients with vestibular disorders, especially when pathology (e.g. vestibular neuritis) selectively affects certain elements of the vestibular end-organ (Fetter and Dichgans, 1996).

The success of LARP and RALP impulses as a bedside test (i.e. without measuring head and eye rotation) depends on the examiner seeing a voluntary re-fixation saccadic eye movement after completion of the head impulse. Clinically, we were able to detect an abnormal response during head impulses toward lesioned SCCs in the clinic, prior to measuring the VOR in this experiment. However, the predictive value of the these diagonal head impulses as a bedside test needs to be evaluated formally.

Conclusions

LARP- and RALP-plane head impulses have exposed a severe, permanent deficit in anterior and posterior SCC function following surgical lesions. These canal-plane impulses are currently the only available method to evaluate each vertical SCC separately. Together with horizontal head impulses, they form a comprehensive battery of clinical SCC tests. The sensitivity of the head impulses in demonstrating a complete lesion of one SCC depends on exposing the inherent asymmetry in primary vestibular afferent neuron physiology, and the specificity of the test depends on the anatomical orientation of the SCCs.

Acknowledgements

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Appendix

Analysis of the projections of the SCCs into the LARP, RALP, pitch, roll and yaw planes

Blanks *et al.* (1975*a*) calculated the equations which describe the normals to the plane of the six SCCs in cardinal coordinates: roll, pitch and yaw (x, y, z). We have expressed these equations as matrices, representing the directional sensitivity of each SCC to head rotation in the roll, pitch and yaw planes; (the individual canal matrix [Ci]: Matrix 1). We have maintained the same Cartesian coordinate system: superior ($+z$), left ($+y$) and anterior ($+x$), but we have re-defined the direction of the SCC-sensitivity vectors to follow the 'right hand screw' convention. In other words, a SCC is stimulated by angular acceleration in the direction of the advance of a right hand screw, where the axis of rotation is the directional sensitivity vector of that SCC. For any head rotation, the change in firing of primary afferent neurons from a particular SCC is calculated by multiplying the roll, pitch and yaw components of head velocity by the SCC sensitivity matrix (Matrix 1) (Robinson, 1982). It is important to note that this method calculates the geometric projection of the head acceleration vector onto the SCC-sensitivity vector, but does not account for the physiological non-linear firing in primary vestibular afferent neurons. For example, during a leftward ($+z$) head rotation, the left lateral SCC is stimulated by 0.905 of the total head acceleration, the right lateral SCC is disfacilitated by 0.905, the right posterior SCC is stimulated by 0.320, and the left posterior SCC is disfacilitated by 0.320 of the total head acceleration. During a pitch-down head impulse with maximum head acceleration of 2500°/s², both anterior SCCs are stimulated by a component of 1883°/s², both posterior SCCs are disfacilitated by 1403°/s², and both lateral SCCs are stimulated by 395°/s². During a roll-clockwise head impulse, the right anterior SCC is stimulated by 1630°/s², the right posterior SCC is stimulated by 1893°/s², and right lateral SCC is stimulated by 913°/s²; and the left-sided SCCs are disfacilitated by the corresponding amounts.

Fig. A1 Axial view of the head from above, showing the axes about which the head impulses were delivered. The actual motion is perpendicular to these axes. The roll (x) and pitch (y) axes are passively rotated $+45°$ about the earth-vertical z-axis, to become the RALP and LARP axes, respectively. Arrows indicate the positive direction for each axis, and they follow the 'right hand' convention. For example a head impulse about the RALP axis in a positive direction, corresponds to a head impulse in the direction of the right anterior SCC (shaded white).

Matrix 1 *The individual canal matrix [Ci]*

	la	lp	ll	ra	rp	rl
x	−0.652	−0.757	−0.365	0.652	0.757	0.365
y	0.753	−0.561	0.158	0.753	−0.561	0.158
z	0.017	−0.320	0.905	−0.017	0.320	−0.905

This is a directional sensitivity matrix for each SCC, expressed in roll, pitch and yaw coordinates (x, y, z). la = left anterior SCC; lp = left posterior SCC; ll = left lateral SCC; ra = right anterior SCC; rp = right posterior SCC; rl = right lateral SCC.

In order to express the individual canal matrix in canal-plane coordinates (RALP, LARP, z), we have passively rotated the (x, y, z) coordinate system by $+45°$ about the earth-vertical z-axis (Fig. A1). This is equivalent to rotating all points $-45°$ about the z-axis, and is achieved by multiplying Matrix 1 by Matrix 2 (Haslwanter, 1995).

Matrix 2 *Rotation matrix which passively rotates the* x- *and* y-*axes by* $+45°$ *about the z-axis.*

cos (−45°)	−sin (−45°)	0
sin (−45°)	cos (−45°)	0
0	0	1

Matrix 3 *[Ci'] the modified individual canal matrix*

	la	lp	ll	ra	rp	rl
RALP	0.071	−0.932	−0.146	0.993	0.139	0.370
LARP	0.993	0.139	0.370	0.071	−0.932	−0.146
z	0.017	−0.320	0.905	−0.017	0.320	−0.905

This is the directional sensitivity matrix for each SCC, expressed in canal-plane coordinates (RALP, LARP, z). By convention, the LA-and RA-directions are positive.

Using canal-plane coordinates (Matrix 3) we calculate that during head acceleration in the direction of the right anterior SCC ($+$RALP), the right anterior SCC is stimulated by 0.99 of the total head acceleration, the left posterior SCC is disfacilitated by 0.93, the right lateral SCC is stimulated by 0.37 and the left lateral SCC is disfacilitated by 0.15 of the total head acceleration.

Geometric alignment of the extra-ocular muscles

The six extra-ocular muscles in each eye act as three pairs of approximately co-planar muscles, with each muscle pulling in the opposite direction to its partner. The average plane of action for each pair (in the left eye) was calculated by Robinson (1982), and we have represented that data in our coordinate system (Matrix 4).

Matrix 4 *The muscle matrix [M]*

	lmr	sir	sio
Ex	0.015	0.424	0.788
Ey	−0.005	−0.906	0.600
Ez	0.999	0.016	0.140

Plane of action of the extra-ocular muscles in the left eye (adapted from Robinson, 1982). lmr = lateral and medial rectus; sir = superior and inferior rectus; sio = superior and inferior oblique. Ex, Ey, and Ez refer to the vector components of eye velocity in the roll, pitch and yaw planes, respectively. The first named muscle pulls in the direction indicated by the sign of the vector, and the second named muscle pulls in the opposite direction.

We can also express the muscle matrix in canal-plane coordinates (Matrix 5) by multiplying Matrix 4 by the rotation matrix (Matrix 2).

Matrix 5 *The modified muscle matrix [M′]*

	lmr	sir	sio
ERALP	0.007	−0.341	0.981
ELARP	−0.014	−0.940	−0.133
Ez	0.999	0.016	0.140

Plane of action of the extra-ocular muscles in the left eye. lmr = lateral and medial rectus; sir = superior and inferior rectus; sio = superior and inferior oblique. ERALP, ELARP, and Ez refer to the vector components of eye velocity in the RALP, LARP and yaw planes, respectively. The first named muscle pulls in the direction indicated by the sign of the vector, and the second named muscle pulls in the opposite direction.

From this representation of the muscle matrix of the left eye, it is clear that the obliques pull predominantly in the RALP plane (0.981), the horizontal recti pull almost entirely in the horizontal plane (0.999), and the vertical recti pull predominantly in the LARP plane (0.94), but they also have some action in the RALP plane (0.341). By considering the direction of muscle action, the left superior oblique pulls in the RA direction (producing downward rotation and intorsion). The left superior rectus pulls mainly in the RP direction (producing upward rotation and intorsion) but also has a lesser action in the LP direction; the result is a largely upward eye rotation (0.906) with a smaller degree of intorsion (0.424) (Matrix 4).

The brainstem matrix
Robinson (1982) derived the 'brainstem matrix' which determines the pattern and relative strength of the neural projection from each pair of SCCs onto each pair of ocular motor neurons. The VOR is a product of three matrices: the canal-pair sensitivity-matrix [C], the brainstem matrix [B], and the muscle matrix [M]. The canal-pair sensitivity-matrix [C] was calculated as the average plane for each SCC pair. Thus if V_E is the eye velocity output and V_H is the head velocity stimulus, then

$$V_E = [M][B][C]V_H \qquad \text{(Equation 1)}$$

Robinson derived the brainstem matrix by assuming that the VOR is ideal, in other words, $V_E = -V_H$. If this is true, then

$$[M][B][C] = [-I] \qquad \text{(Equation 2)}$$

where [−I] is the negative of the identity matrix.

Matrix 6 *[–I] the negative of the identity matrix*

−1	0	0
0	−1	0
0	0	−1

Robinson then calculated the brainstem matrix by rearranging Equation 2.

$$[B] = [M^{-1}][-I][C^{-1}] \qquad \text{(Equation 3)},$$

where $[M^{-1}]$ and $[C^{-1}]$ are the inverse of their respective matrices.

We have modified the VOR gain element [−I] to reflect a more realistic value for the roll VOR gain of 0.7 (Aw *et al.*, 1996*a*).

Matrix 7 *[–VOR] the VOR-gain matrix*

−1	0	0
0	−1	0
0	0	−0.7

[−VOR] was used in our calculation of the brainstem matrix.

$$[B] = [M^{-1}][-VOR][C^{-1}] \qquad \text{(Equation 4)}.$$

Using Equation 4, we calculated the brainstem matrix.

Matrix 8 *The brainstem matrix [B]*

	Clrl	Clarp	Cralp
lmr	−1.016	0.230	−0.159
sir	0.106	0.877	0.331
sio	0.152	0.089	−0.740

Clrl = canal-pair sensitivity vector for the left and right lateral SCCs; Clarp = canal-pair sensitivity vector for the left anterior/right posterior (LARP) SCC pair; Cralp = canal-pair sensitivity vector for the right anterior/left posterior (RALP) SCC pair. A positive value indicates that the first named SCC excites the first named extra-ocular muscle, and the second named SCC excites the second named extra-ocular muscle. A negative value indicates that the first named SCC excites the second named extra-ocular muscle, and vice versa.

Specificity of canal-plane head impulses
Because the vertical SCCs do not lie exactly in the diagonal LARP and RALP planes, and because the SCCs are not exactly orthogonal

to each other (Blanks *et al.*, 1975*a*), head impulses in these canal planes will stimulate out-of-plane SCCs to some extent. For example, during a LA-direction head impulse, the left lateral SCC is stimulated by 0.37 of the total head acceleration (Matrix 3). In this case, how much of the resulting eye velocity that we plot in the LARP plane is due to left lateral SCC stimulation?

From the brainstem matrix (Matrix 8), we can see that stimulation of the left lateral SCC causes contraction of the left medial rectus with a relative efficacy of 1.016, contraction of the left superior rectus (0.106) and left superior oblique (0.152). By multiplying the muscle matrix (Matrix 4) by column 1 of the brainstem matrix (Matrix 8), we calculate that the resulting eye rotation is rightward (–0.992) with some intorsion (0.150), without any vertical component (which is cancelled by the opposing actions of superior oblique and superior rectus). To calculate the LARP plane contribution, we multiply the modified muscle matrix (Matrix 5) by column 1 of the brainstem matrix (Matrix 8). The relative LARP vector component of the eye velocity is 0.106 in the RP direction. However, during a LA-direction head impulse, the left lateral SCC is only stimulated by 0.37 of total head acceleration, so the relative LARP vector component of the VOR due to the left lateral SCC is only 0.039.