# The cervico-ocular reflex

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# Cervico-Ocular Reflex in Normal Subjects and Patients with Unilateral Vestibular Hypofunction

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**Objective:** To determine whether the cervico-ocular reflex contributes to gaze stability in patients with unilateral vestibular hypofunction.
**Study Design:** Prospective study.
**Setting:** Tertiary referral center.
**Patients:** Patients with unilateral vestibular hypofunction (n = 3) before and after vestibular rehabilitation and healthy subjects (n = 7).
**Interventions:** Vestibular rehabilitation.
**Main Outcome Measures:** We measured the cervico-ocular reflex in patients with unilateral vestibular hypofunction before and after vestibular rehabilitation and in healthy subjects. To measure the cervico-ocular reflex, we recorded eye movements with a scleral search coil while the trunk moved at 0.3, 1.0, and 1.5 Hz beneath a stabilized head. To determine whether the head was truly stabilized, we measured head movement using a search coil.
**Results:** We found no evidence of cervico-ocular reflex in any of the seven healthy subjects or in two of the patients with unilateral vestibular hypofunction. In one patient with chronic unilateral vestibular hypofunction, the cervico-ocular reflex was present before vestibular rehabilitation only for leftward trunk rotation (relative head rotation toward the intact side). After 5 weeks of placebo exercises, there was no change in the cervico-ocular reflex. After an additional 5 weeks that included vestibular exercises, cervico-ocular reflex gain for leftward trunk rotation had increased threefold. In addition, there was now evidence of a cervico-ocular reflex for rightward trunk rotation, potentially compensating for the vestibular deficit.
**Conclusion:** The cervico-ocular reflex appears to be a highly inconsistent mechanism. The change of the cervico-ocular reflex in one patient after vestibular exercises suggests that the cervico-ocular reflex may be adaptable in some patients. **Key Words:** Cervico-ocular reflex—Vestibular hypofunction.
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The cervico-ocular reflex (COR) has been proposed as a mechanism of gaze stability for subjects with bilateral (1–5) and unilateral vestibular hypofunction (6,7). This hypothesis is based, in part, on the finding that COR gain in subjects with vestibular hypofunction is greater than COR gain in healthy subjects (3,8,9). Studies of subjects with healthy vestibular systems report COR gain values that vary from 0 to 0.4 (2,8–10). Of all these studies investigating COR in healthy individuals, only one has measured head movements and eye movements to control for head movement producing a vestibulo-ocular reflex during testing (10). In that study of eight healthy subjects, methods were used to reduce head movements to less than 0.04 degree, and COR gain was always less than 0.07. Given the variability among separate studies and the question of adequate stabilization of the head during measurement of COR, it is not clear whether the COR is present in healthy subjects or in patients with unilateral vestibular hypofunction (UVH).

The purpose of this study was to measure COR while head movement is monitored in patients with UVH and in healthy subjects. We also investigated the effects of vestibular adaptation exercises on COR gain in subjects with vestibular hypofunction. This article presents evidence that COR is not found in healthy subjects or in the majority of patients with unilateral vestibular loss. One patient with UVH was found to have a COR that increased after vestibular adaptation exercises.

## PATIENTS AND METHODS

### Subject Characteristics

Three patients with UVH and seven healthy controls provided informed consent in compliance with the Emory University Institutional Review Board. The patient subjects were grouped on the basis of diagnoses. Patients with UVH had greater than or equal to 25% unilateral weakness between the right and left sides on caloric and rotary chair tests (constant

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velocity rotation, 240 deg/s). Two of the patients with UVH were diagnosed as having vestibular neuritis on the basis of history. Magnetic resonance imaging (MRI) of the head was performed to rule out an acoustic neuroma in two of the patients with UVH. All normal subjects had normal caloric tests. Each of the subjects was screened for cervical abnormality.

### Measurement of COR and Vestibulo-Ocular Reflex

During COR and vestibulo-ocular reflex (VOR) testing, the subject was seated in the rotary chair and the trunk was stabilized using a dynamic air splint (Kramer; Cramer Products, Gardner, KS, U.S.A.). Velcro bands were used to further stabilize the body in the chair. Eye and head position were measured using search coils. The COR was measured by recording eye movements during trunk oscillation while the subject's head was stabilized using a bite bar wrapped in dental wax (positioned in front of the subject). During COR testing, trunk and knees moved in the direction of the rotating chair. The VOR was measured by recording eye movements during whole-body sinusoidal rotation. Caution was taken to ensure comfort of the subject and that the head was positioned in 30 degrees of flexion (11).

### Protocol

Cervico-ocular reflex and VOR were recorded at 0.3, 1.0, and 1.5 Hz (peak velocities, 24, 31, and 34 deg/s, respectively). All tests were performed in complete darkness. The subjects were not given specific instructions during COR testing, although an investigator was present in the room to ensure alertness and to monitor the comfort of the subjects. Subjects were asked to perform naming tasks to ensure alertness during VOR testing. Subjects with vestibular hypofunction were tested at the time of the initial assessment, after 4 weeks of placebo exercises, and after 5 weeks of vestibular adaptation exercises.

### Protocol to Increase COR Gain with Mental Imagery

In addition to the above-described protocol, three of the healthy control subjects performed tasks to determine whether the COR could be enhanced through mental imagery. Specifically, the subjects were asked to 1) imagine a target on their knees; 2) concentrate on the motion of their trunks; and 3) imagine the direction of the relative head rotation.

### Data Capture and Analysis

Eye and head position was measured using 6-foot magnetic field coils (CNC Engineering, Seattle, WA). Search coils (Skalar, Delft, The Netherlands) were precalibrated by measuring changes in voltages occurring during known rotations. The system was 99% linear over an operating range of ±25 degrees. Horizontal and vertical eye and head position as well as chair tachometer and chair position were converted to digital signals using software written in LabVIEW (National Instruments, Austin, TX).

All data from the coil experiments were stored on the hard drive of a Dell Dimension Pentium III (Dell Computer Corporation, Austin, TX) desktop computer for offline analysis. Gaze and head position signals were filtered at 200 Hz using six-pole Bessel antialiasing filters before digitization at 1 kHz with 16-bit precision (Krohn-Hite Corp., Avon, MA). Horizontal and vertical eye and head velocities were differentiated from position signals using a two-point central difference algorithm. Horizontal and vertical eye and head accelerations were generated by differentiating velocity arrays. Eye in orbit velocity and position was determined by subtracting head velocity and position signals from gaze velocity and position signals.

Mean peak COR gain was calculated as the ratio of peak eye velocity to peak trunk (chair) velocity. In determining the mean peak COR gain, we corrected (because of unwanted head motion) for the contribution of VOR (ratio of peak eye velocity to peak head velocity) by multiplying the slow component eye velocity (SCEV) on the basis of the VOR gain measured at each of the three frequencies (0.3, 1.0, and 1.5 Hz). We then subtracted that value from the SCEV measured during COR testing.

Right and left hemicycles were analyzed individually. Only COR gain values greater than 0.06 were considered significant and used for statistical analysis. The value of 0.06 was chosen, as this represents the limit at which we could accurately detect the eye position signal.

### Exercises

Placebo exercises consisted of saccadic eye movements with the head stationary while viewing a Ganzfeld (blank surface). Vestibular exercises included adaptation exercises and eye-head exercises to targets (Table 1) (12). All exercises were to be performed four to five times daily for a total of 30 to 40

**TABLE 1.** *Progression of vestibular rehabilitation to improve gaze stability*

| Exercise weekly progression | Duration | Frequency |
|---|---|---|
| Week 1: X1 with target held in hand and also with target at distance, horizontal and vertical head movements | 1 min each exercise | Five times daily |
| Week 2: X1 with target held in hand and also with target at distance, horizontal and vertical head movements; also eye head movements between two targets with emphasis on seeing clearly | 1–2 min each exercise | Five times daily |
| Week 3: X1 with target held in hand and also with target at distance, horizontal and vertical head movements; X1 with checkerboard with target placed in center held in hand, horizontal head movements; eye head movements between two targets; imaginary target paradigm | 1 min each exercise | Four times daily |
| Week 4: X1 with checkerboard with target placed in center held in hand; X2 with target held in hand, horizontal and vertical head movements; eye head movements between two targets; imaginary target paradigm | 1 min each exercise | Four times daily |
| Week 5: X1 with target held in hand; horizontal and vertical head movements; X1 with checkerboard with target placed in center held in hand, horizontal and vertical head movements; X2 with target held in hand, horizontal and vertical head movements; eye head movements between two targets; imaginary target paradigm | 1 min each exercise | Four times daily |

X1, head rotates horizontally or vertically while subject views a stationary target; X2, head and target rotate in opposite directions (horizontal or vertical) while subject attempts to view target.

min/d. Individuals were provided with daily calendars to mark, ensuring compliance. Subjects brought the calendars to their weekly visits. Compliance for all of the subjects ranged from 50 to 100%, based on the calendars.

## RESULTS

### Subject Characteristics

Table 2 shows the individual characteristics for all subjects. Note that the age range for the normal subjects and the subjects with UVH are similar ($t$ statistic = 1.04, $p = 0.16$). None of the subjects had neck abnormality. Two of the three patient subjects had MRI or computed tomographic scans. There were no intracranial abnormalities noted. One patient (Subject UVL35) had an acoustic neuroma identified. That tumor was removed before participation in the study.

### COR in Healthy Controls and in Other Subjects with UVH

We found no evidence of COR in any of the normal subjects. This is illustrated in Figure 1 A for one subject. Table 3 summarizes the data from all controls. Attempts to enhance the COR in the healthy control subjects through mental imagery did not produce a COR. We found no COR in the other two patients with UVH. An example of this is shown in Figure 1 B. Note that any SCEV can be attributed to the small amount of head movement during testing.

### Subject (UVH81) with COR

An 81-year-old individual had a sudden onset of vertigo 11 months before testing (Subject UVH81). The vertigo resolved over the course of several days, but she continued to have complaints of disequilibrium and oscillopsia. In this patient, bithermal and ice water irrigation showed no response on the left side and normal response on the right side. Rotary chair testing showed lower gains for 60 and 240 deg/s constant velocity rotations to the left (0.49 and 0.21) compared with rotation to the right (0.59 and 0.34). Magnetic resonance imaging scans were normal. Audiography showed mild to moderately severe, symmetric sensorineural hearing loss at high frequencies bilaterally. The patient was diagnosed as having left vestibular neuronitis on the basis of history and examination.

Table 4 summarizes COR and VOR gain for this patient at initial assessment, after placebo exercises, and after vestibular adaptation exercises. Cervico-ocular reflex was evident during the initial assessment only for trunk rotation to the left at 0.3 Hz (relative head rotation to the right) (Fig. 2A). The mean gain of the COR was 0.10 ± 0.04 (range, 0.06–0.16). After 4 weeks of placebo exercises, there were no appreciable changes in COR. After 5 weeks of vestibular rehabilitation, during 0.3-Hz trunk rotations, mean COR gain had increased threefold (0.32 ± 0.13). Peak slow eye velocity during trunk rotation to the left had increased to 7.8 ± 3.0 deg/s, whereas the mean head velocity was only 0.6 ± 0.4 deg/s (Fig. 2B). COR was also identified at 1.0- and 1.5-Hz trunk rotation to the left (COR gain = 0.13 ± 0.04 and 0.13 ± 0.05, respectively). There was an indication of COR for rightward trunk rotation (relative head rotation to the left), but only in a few trials (Table 4).

## DISCUSSION

### COR in Healthy Individuals

We found no measurable COR in seven healthy individuals. This differs from previous reports (2,8,9). We believe the explanation for this difference is the degree of head stabilization during COR testing. We measured head stabilization using a search coil attached to the

**TABLE 2.** *Subject characteristics: initial assessment*

| Subject | Age (yr) | Time from onset (mo) | Head thrust test | Caloric asymmetry (% loss) | Rotary chair | |
|---|---|---|---|---|---|---|
| | | | | | VOR gain[a] | Tc (s)[b] |
| UVH | 35 | 2 | + Right | 100 | Left = 0.565<br>Right = 0.362 | Left = 3.8<br>Right = 5.4 |
| UVH | 52 | 5.5 | + Left | 100 | Left = 0.205<br>Right = 0.427 | Left = 10.1<br>Right = 6.5 |
| UVH | 81 | 11 | + Left | 100 | Left = 0.209<br>Right = 0.336 | Left = 7.9<br>Right = 10.3 |
| Normal | 31 | NA | –Bil | 20 | Not tested[c] | Not tested[c] |
| Normal | 36 | NA | –Bil | 4 | Not tested[c] | Not tested[c] |
| Normal | 40 | NA | –Bil | 16 | Not tested[c] | Not tested[c] |
| Normal | 83 | NA | –Bil | 4 | Not tested[c] | Not tested[c] |
| Normal | 40 | NA | –Bil | 3 | Not tested[c] | Not tested[c] |
| Normal | 34 | NA | –Bil | 7 | Not tested[c] | Not tested[c] |
| Normal | 27 | NA | –Bil | Not tested[d] | Left = 0.408<br>Right = 0.424 | Left = 13.5<br>Right = 24.4 |

[a]VOR gain for 240-deg/s step rotations to the right and left.
[b]Time constant for 60-deg/s step rotations to the right and left.
[c]In normal subjects, vestibular function was assessed usually with caloric testing.
[d]Caloric could not be performed adequately because of scarring of tympanic membrane.
+, Side of unilateral vestibular hypofunction; –Bil, negative head thrusts in both directions; VOR, vestibulo-ocular reflex; UVH, unilateral vestibular hypofunction; NA, not applicable.

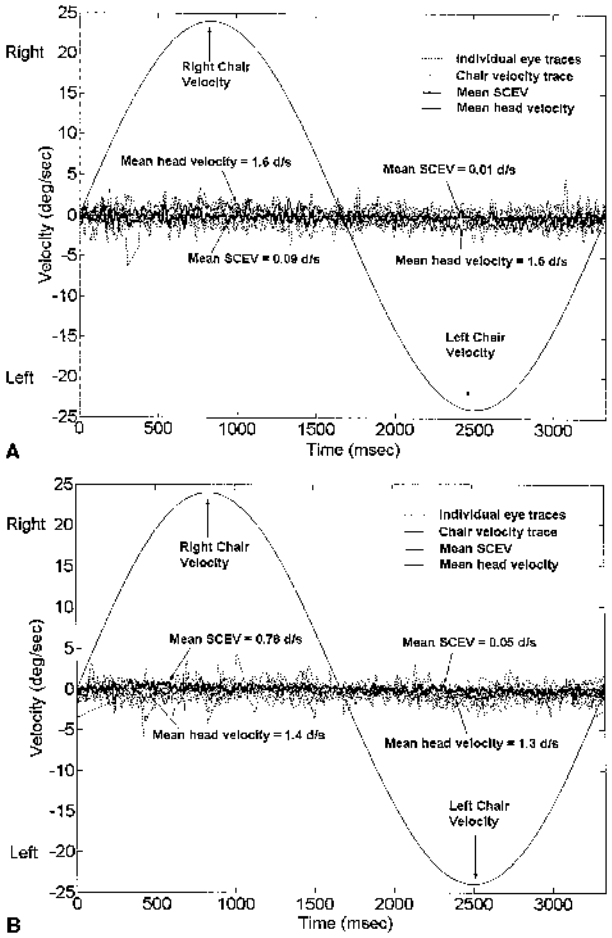


**FIG. 1.** No evidence of a COR is seen in a healthy control (*A*) subject. Chair velocity peaks at 24 deg/s. Neither eye nor head velocities are greater than 1.6 deg/s. All traces are of motion in the horizontal plane. Eye velocity trials have been desaccaded. Slow component eye velocity trials are not inverted because the head is intended to be stable. Positive numbers along the ordinate indicate rightward velocity rotation, whereas negative numbers indicate leftward velocity rotation. Dashed line placed at zero velocity is for reference. (*B*) Subject with complete right UVH. No evidence of a COR in a subject with complete right UVH. Neither eye nor head velocities are greater than 1.4 deg/s (see Fig. 1 A for legend).

head. In our pilot studies with healthy control subjects and with subjects with vestibular hypofunction, eye movements attributed initially to COR were all related to excessive head movements. Because of this, we took several steps to eliminate head movements. First, we found it necessary to wrap the lower trunk and hips in a dynamic air splint to stabilize the body. Second, we had to teach subjects how to bite down on the bite bar to prevent unwanted head movements. Finally, training sessions were performed to ensure the head remained stable. Even with all of this preparation, there was still some head movement present, which caused a VOR response. By subtracting the VOR eye response during trunk rotation, we feel we have been able to reliably measure COR alone. Our results of no recordable COR in healthy subjects is in agreement with Sawyer et al. (10), who also measured head movements in eight normal subjects.

### Effects of Mental Set

Some studies of normal subjects and of subjects with bilateral vestibular hypofunction have reported that mental set enhances the COR (9,13). We attempted to reproduce these results in a series of experiments on three of the healthy subjects. The conditions we used included asking the subjects to imagine a target on their knees, to concentrate on the motion of their trunks, and to imagine the direction of the relative head rotation. These three conditions were used in an effort to enhance COR. For example, imagining fixation on the knees would result in SCEV of COR in the direction of the knees. However, we were not able to identify measurable eye movements that could be COR in any of the conditions. Our results, therefore, are similar to those of Sawyer et al. (10), who also could not identify a COR, even when manipulating mental set. Again, we think that differences among these studies are related to the degree of head stabilization during COR testing.

### COR in Individuals with UVH

We found no COR in two of three patients with UVH before and after vestibular exercises. The single patient in whom we did find COR was an 81-year-old subject who developed sudden onset of vertigo 11 months before testing. We believe the SCEV generated by rotation of the trunk while the head was stabilized in Subject UVH81 was due to the COR. The SCEV (2.8 deg/s) generated was more than 10-fold the velocity of head movement (0.23 deg/s) generated during trunk on head rotation.

It is unlikely that other mechanisms such as spontaneous or gaze-evoked nystagmus or certain orienting strategies are responsible for the SCEV we found in Subject

**TABLE 3.** *Mean values for healthy controls*

| Test frequency (Hz) | Eye velocity (deg/s) (mean ± 1 SD) during COR | Head velocity (deg/s) (mean ± 1 SD) during COR | COR gain (mean ± 1 SD) | VOR gain (mean ± 1 SD) |
|---|---|---|---|---|
| 0.3[a] | 0.81 ± 0.82 | 0.89 ± 0.3 | 0.006 ± 0.04[b] | 0.66 ± 0.1 |
| 1.0 | 1.2 ± 1.3 | 1.17 ± 1.19 | 0.02 ± 0.02 | 0.68 ± 0.003 |
| 1.5 | 1.5 ± 1.7 | 1.94 ± 1.95 | 0.01 ± 0.01 | 0.84 ± 0.002 |

[a]COR was measured in all seven subjects at 0.3 Hz but in only four subjects at 1.0 and 1.5 Hz.
[b]This value is below the resolution of position signal from eye coil.
COR, cervico-ocular reflex; VOR, vestibulo-ocular reflex.

**TABLE 4.** *Cervico-ocular reflex and vesibulo-ocular reflex gain across frequencies for subject UVH81 with cervico-ocular reflex*

| | | Initial assessment | | After placebo exercises | | After adaptation vestibular exercises | |
|---|---|---|---|---|---|---|---|
| | Frequency | COR gain (mean ± 1 SD) | VOR gain (mean + 1 SD) | COR gain (mean ± 1 SD) | VOR gain (mean + 1 SD) | COR gain (mean ± 1 SD) | VOR gain (mean + 1 SD) |
| | 0.3 Hz | 0.01 ± 0.01 (0 of 18 trials) | 0.70 ± 0.11 | 0.03 ± 0.03 (0 of 11 trials) | 0.67 ± 0.05 | 0.09 ± 0.03 (2 of 14 trials) | 0.61 ± 0.06 |
| VOR - WBR (ipsilesional) | 1.0 Hz | 0.02 ± 0.15 (0 of 16 trials) | 0.62 ± 0.06 | 0.02 ± 0.02 (0 of 7 trials) | 0.78 ± 0.16 | 0.08 ± 0.04 (5 of 30 trials) | 0.44 ± 0.08 |
| COR (head relative left) | | 0.020 ± 0.02 (0 of 20 trials) | | 0.01 ± 0.02 | | 0.04 ± 0.04 | |
| | 1.5 Hz | | 0.64 ± 0.04 | (0 of 17 trials) | 0.59 ± 0.06 | (0 of 30 trials) | 0.90 ± 0.19 |
| | 0.3 Hz | 0.10 ± 0.04 (8 of 18 trials) | 0.97 ± 0.09 | 0.10 ± 0.04 (4 of 11 trials) | 0.92 ± 0.07 | 0.32 ± 0.13 (11 of 18 trials) | 0.78 ± 0.04 |
| VOR - WBR (contralesional) | 1.0 Hz | 0.06 ± 0.01 (0 of 16 trials) | 0.88 ± 0.05 | 0.06 (1 of 6 trials) | 0.89 ± 0.05 | 0.13 ± 0.04 (15 of 30 trials) | 0.90 ± 0.08 |
| COR (head relative right) | 1.5 Hz | 0.04 ± 0.02 (0 of 20 trials) | 0.85 ± 0.04 | 0.08 ± 0.01 (0 of 16 trials) | 0.98 ± 0.07 | 0.13 ± 0.05 (6 of 30 trials) | 0.95 ± 0.16 |

COR, cervico-ocular reflex; VOR, vestibulo-ocular reflex; WBR, whole-body rotation in the dark.

UVH81 during trunk on head rotation. Spontaneous nystagmus in a patient with a left UVH should generate an SCEV to the right. In Subject UVH81 the direction of the SCEV was to the left, but the quick phase component changed directions, which is not congruent with spontaneous nystagmus (Fig. 3). Gaze-holding nystagmus can occur when the eyes are positioned as little as 15 to 30 degrees eccentrically, and is absent when the eyes are centered in the orbit (14). In addition, in gaze-holding nystagmus, the direction of the slow component eye movements is dependent on eye in orbit position. In our patient, eye position during trunk rotations did not exceed 6 degrees eccentrically, which is not sufficient to elicit gaze-holding nystagmus. Furthermore, in our patient, the direction of the slow component eye movements was always to the left, regardless of eye in orbit position (Fig. 3). Thus, gaze-holding nystagmus does not explain the slow eye velocity we identified during trunk on head rotation in our patient. It is also unlikely that the SCEV in Subject UVH81 is due to an orienting strategy. One orienting strategy involves the generation of anticipatory smooth eye movements in response to target motion on the retina (15,16). Anticipatory smooth eye movements have not been found in the absence of a visual target, however (16), such as in our paradigm. In another orienting strategy, the eyes predict the eventual head/chair position and "jump" ahead, in the direction of the eventual head/chair position (17). As can be seen in Figure 3, just as the chair changes from rotating to the left to rotating to the right, the eyes quickly jump to the right. Similarly, quick-phase eye movements to the left occur just as the chair begins rotating to the left. The presence of the quick-phase eye movements of this particular orienting response, however, would not explain the slow-phase eye velocities during trunk on head rotation that we believe is COR.

It is interesting that the COR was present initially only during ipsilesional trunk rotation (head relative right) and that initially we did not find a COR for relative head rotations left (toward the side of the lesion). If the COR serves a compensatory role as a substitute for the VOR, one would expect a COR for relative head rotations toward the left, the side of this subject's vestibular lesion. One possibility is that the COR was present in this patient before the onset of her vestibular deficit. The COR was then lost for one direction with the occurrence of the unilateral vestibular loss.

### COR Adaptation

Our results are the first demonstration of COR adaptation in a patient with UVH. Mean COR gain toward the unaffected side did not change during a 4-week period of placebo exercises, but increased from 0.1 to 0.32 (0.3 Hz) after 5 weeks of vestibular adaptation exercises. In addition, COR was now present for 1.0 and 1.5 Hz. Before the initiation of vestibular exercises, a COR was not present for ipsilesional head movement (relative). This increased to a gain of 0.1 after rehabilitation. Heimbrand et al. (13) demonstrated adaptation of the COR using magnifying lenses in patients with bilateral vestibular loss. Their adaptation paradigm is similar to the stimuli inherent in the exercises performed by our patient which, like magnifying glasses, are designed to produce retinal slip. It is possible therefore that the exercises designed to enhance adaptation of the vestibular system may induce adaptation of the COR. Our findings of a COR at frequencies of 1.0 and 1.5 Hz indicate that the COR may be useful with some activities.

### Contribution of COR to Gaze Stability

Although the number of patients in this study is small, our data suggest that the COR may not contribute significantly to gaze stability during head movements in patients with UVH. First, it appears to be an inconsistent response. It was present in only one of three subjects and, even in that subject, was not found consistently. Second,

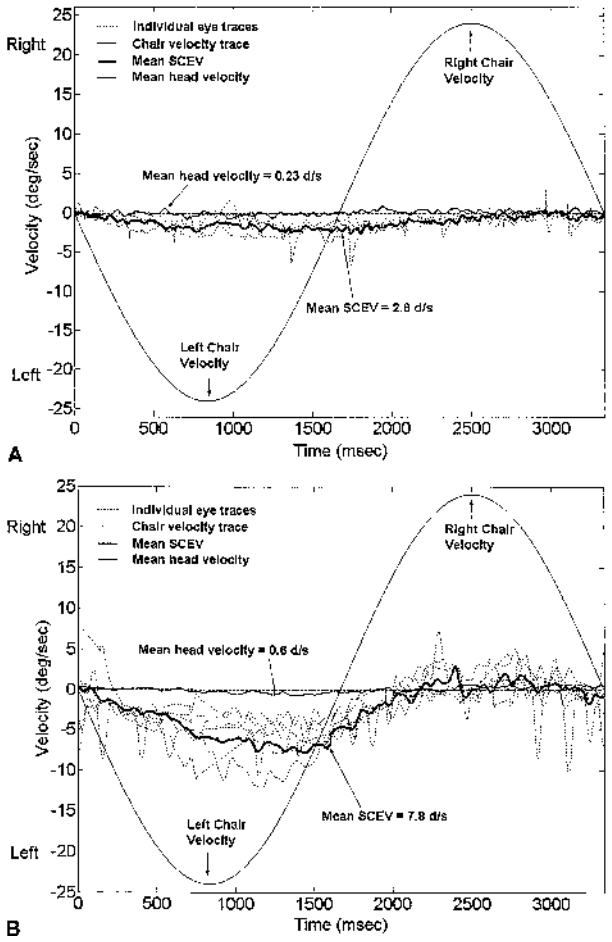


**FIG. 2.** (*A*) Subject with complete left UVH before vestibular rehabilitation. Evidence of COR during 0.3-Hz chair rotation to the left in a patient with left UVH (Subject UVH81) at the time of the initial assessment. After chair (trunk) velocity peaks to the left at 24 deg/s (relative head rotation to the right), an SCEV occurs to the left with a mean 2.8 ± 0.7 deg/s. Mean head velocity is stable at 0.23 ± 0.3 deg/s. All traces are of motion in the horizontal plane. Eye velocity trials have been desaccaded. Positive numbers along ordinate indicate rightward velocity rotation and negative numbers indicate leftward velocity rotation. Dashed line placed at zero velocity for reference. (*B*) Subject with complete left UVH after vestibular rehabilitation. Increased COR gain during 0.3-Hz rotation to the left after 5 weeks of vestibular rehabilitation in Subject UVH81. Mean SCEV increased to 7.8 ± 3.0 deg/s, although head velocity was stable at 0.6 ± 0.4 deg/s (see Fig. 2 A for legend).

COR gain was quite low. The gain of the COR therefore would not prevent significant retinal slip during most normal head movements. Third, the velocities at which COR was present were quite low (24–34 deg/s). The velocity of head movements during many activities of daily living typically exceeds 100 deg/s (18). Finally, it is not clear in what way COR would aid gaze stability when it was not in phase with the relative head movement. It is possible, however, that COR might contribute to gaze stability in some way during slow head movements.

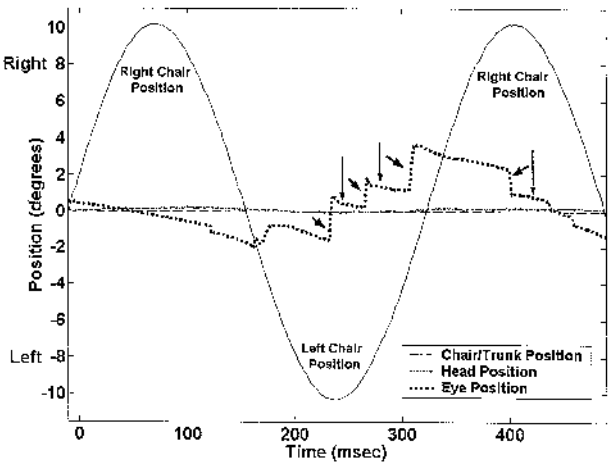


**FIG. 3.** Position trace of subject with complete left UVH. Position plot of eye and head in Subject UVH81 during trunk on head COR trial. (*Bold arrows*) Quick-phase eye position, which changes orientation as the trunk position changes. (*Vertical arrows*) Slow-phase eye position, which does not change. All traces are of motion in the horizontal plane. Positive numbers along ordinate indicate rightward position and negative numbers indicate leftward position. Dashed line are placed at zero velocity for reference.

## CONCLUSION

The COR is difficult to elicit with passive trunk on head rotation in normal subjects and in subjects with unilateral vestibular hypofunction. The difference between our results and earlier studies on COR appears to be the degree of head stabilization, confirmed by measurement. We found evidence of a COR in only one patient with a unilateral vestibular hypofunction. Initially, the COR was present only for trunk rotation to the left (relative head rotation toward the intact side). Vestibular adaptation and eye-head movement exercises appear to produce an increase in the gain of the COR for both directions, although the asymmetry remained. If the COR contributes to gaze stability, it would be in a very limited way and only for lower velocity head rotation.

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