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Three-Dimensional Analysis of Vertical and Torsional Vestibulo-Ocular Reflex

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Abstract

Objectives: This study aimed to quantify the nystagmus induced by four kinds of angular accelerations exerted on the head, and to clarify whether the semicircular canal theory can explain the mechanism of ocular counter-roll. **Methods:** Subjects were 12 healthy humans. Experiment 1: Head was passively moved back and forth for three cycles (pitch rotation). Experiment 2: Head was rotated 45˚ to the left, moved back and forth for three cycles. Experiment 3: Head was rotated 60˚ to the left, moved back and forth for three cycles. Experiment 4: Head was tilted laterally to the left and right for three cycles without axial rotation (roll rotation). In all experiments, the amplitude was approximately 30˚, and the frequency was 0.33 Hz. Eye movements in the dark were recorded and analyzed. **Results:** In Experiment 1, nystagmus was purely vertical. In Experiments 2 to 4, nystagmus was vertical/torsional. The intensity of the vertical component was greatest in Experiment 1, followed by Experiment 2, then Experiment 3, and was smallest in Experiment 4. The intensity of the torsional component was in reverse pattern. **Conclusion:** Considering the nystagmus direction and intensity, ocular counter-roll can be explained by the semicircular canal theory.

Keywords

Semicircular Canal, Law of Inertia, Ocular Counter-Roll, Angular Acceleration, Endolymph

1. Introduction

The vestibulo-ocular reflex (VOR) is an involuntary response that maintains gaze. As a result of this reflection, the eyes move relatively in the opposite direction of head rotation, stabilizing the image. VOR responds to angular acceleration in all directions, 360˚, and reacts to all frequencies from low to high.

Accurate reflection occurs when fixation is sustained on the spot in a well-lit environment, and the gain (eye speed/head speed) is exactly 1. However, the gain decreases when gaze fixation is impossible, such as in darkness. This finding means that the response of the inner ear alone is insufficient for effective gaze control. VOR serves solely as an adjunct, and precise fixation is unattainable without a target.

Angular acceleration is a physiological stimulus and is non-invasive, facilitating its use in clinical testing [1]; however, it delivers a bilateral stimulus, making it impossible to identify the affected side. Angular acceleration cannot stimulate only one semicircular canal (SCC) based on the principles of physics. Additionally, spontaneous nystagmus affects the outcomes. Owing to the limitations of the device, the only structure that could be stimulated by the rotation test was the lateral SCC. Stimulation of the lateral SCC induces eye movements parallel to that plane. This is called Ewald's first law. Horizontal nystagmus is induced during yaw rotation (horizontal rotation).

The video head impulse test has recently been introduced [2]. The device is compact and can measure gain, resulting in global adoption. A substantial advantage is that the rotation direction can be set arbitrarily, facilitating the assessment of the vertical SCCs. This test uses a potent stimulus while fixating on a single point in a well-lit environment. This complicates the elucidation of the function of the SCCs. In bright conditions, visual stimuli significantly affect eye movements. The assessment of the responses of the inner ear should be conducted in a dark environment with a weak stimulus, because the angular acceleration typically encountered by individuals is exceedingly weak and momentary.

Previous studies on the VOR have predominantly focused on the lateral SCCs, with few studies on the VOR induced by stimulation of the human vertical SCCs [3] [4]. Therefore, we attempted to quantitatively analyze the vertical and torsional VOR using a three-dimensional analysis software for eye movements. Ikeda *et al.* [5] developed this software, and it analyzes video images frame by frame on a Macintosh computer. Although it is unsuitable for long-term eye movement analysis, measuring maximum slow-phase velocity (MSV) in nystagmus can be performed easily. Therefore, quantitative analysis of the torsional component, previously unattainable with conventional electronystagmography, has become feasible.

Ocular counter-roll was discovered approximately 200 years ago. It is the rotational movement of the eyes about their visual axes, rotating opposite to the direction of head tilt to maintain gaze. This reflex was thought to be mediated by the utricle. Several animal experiment studies have demonstrated that stimulation of the otolithic organs induces a few types of nystagmus [6] [7]. Clinical applications of video-oculography (VOG) analysis as an otolithic organ function test have been explored [8]. However, we have noticed a curious phenomenon. Following the Epley maneuver for benign paroxysmal positional vertigo (BPPV), pathological debris is stimulating the utricle. In spite of this situation, no nystagmus was observed in the sitting position. We have prompted the hypothesis that nystagmus

may not emerge from the utricle [9]. If this is accurate, it suggests that ocular counter-roll may originate from the SCCs (SCC theory).

This study aimed to quantify the nystagmus induced by four kinds of angular accelerations exerted on the head, and to clarify whether the SCC theory can explain the mechanism of ocular counter-roll.

2. Materials and Methods

2.1. Subjects

This study included 12 healthy individuals without a previous history of dizziness.

Experiment 1

Each subject's head was passively moved back and forth for three cycles while sitting (pitch rotation). The amplitude was approximately 30˚, and the frequency was 0.33 Hz.

Experiment 2

Each subject's head was rotated 45˚ to the left while seated, moved back and forth for three cycles. The amplitude was approximately 30˚, and the frequency was 0.33 Hz.

Experiment 3

Each subject's head was rotated 60˚ to the left while seated, moved back and forth for three cycles. The amplitude was approximately 30˚, and the frequency was 0.33 Hz.

Experiment 4

Each subject's head was tilted laterally to the left and right for three cycles without axial rotation while seated (roll rotation). The amplitude was approximately 30˚, and the frequency was 0.33 Hz.

Eye movements were captured in the dark using an infrared charge-coupled device camera and converted into three-dimensional VOG. Despite the darkness, the participants were directed to gaze as directly forward as possible.

In Experiments 1 to 3, we measured the MSV of the horizontal, vertical, and torsional components of nystagmus that manifested during downward (forward) head movement. In Experiment 4, we quantified the MSV of the horizontal, vertical, and torsional components of nystagmus induced by rightward head movement.

2.2. Eye Movements Analysis

Eye movements were recorded and converted to digital data. ImageJ version 1.36 software (a public domain, Java-based image-processing program developed at the National Institutes of Health) was utilized to perform three-dimensional VOG. The XY center of the pupil was calculated to analyze the horizontal and vertical components. For analysis of the torsional component, the entire iris pattern, rotated in steps of 0.1˚, was superimposed over the corresponding area of the subsequent iris pattern, and the angle at which both iris patterns exhibited the greatest match was calculated [5].

3. Results

Table 1 and Table 2 present the results. Actual nystagmus is presented in the Video [10]. Figure 1 depicts the VOG of subject 1.

Experiment 1

Horizontal component: No subject exhibited a horizontal component.

Vertical component: All subjects exhibited a downward vertical nystagmus. The mean MSV was 33.3˚/s, with a minimum value of 16.7˚/s and a maximum value of 63˚/s. The standard deviation was 13.8˚/s, indicating a significant variation.

Torsional component: No subject exhibited a torsional component.

Experiment 2

Horizontal component: Three subjects exhibited a rightward horizontal component.

Vertical component: Eleven subjects exhibited a downward vertical component. The mean MSV was 21˚/s, with a minimum of 0˚/s and a maximum of 52.4˚/s. The standard deviation was 13.2˚/s, indicating a significant variation.

Torsional component: Ten subjects exhibited a rightward torsional component. The mean MSV was 8.5˚/s, with a minimum of 0˚/s and a maximum of 13.9˚/s. The standard deviation was 6.6˚/s.

Experiment 3

Horizontal component: Eight subjects exhibited a rightward horizontal component. The mean MSV was 9.9˚/s, with a minimum of 0˚/s and a maximum of 37.2˚/s. The standard deviation was 11.6˚/s.

Vertical component: Eleven subjects exhibited downward vertical component, with a mean MSV of 18.2˚/s with a minimum of 0˚/s and a maximum of 35.9˚/s. The standard deviation was 10.8˚/s.

Torsional component: All subjects exhibited a rightward torsional component. The mean MSV was 12˚/s with a minimum of 1.8˚/s and a maximum of 22.3˚/s. The standard deviation was 5.8˚/s.

Experiment 4

Horizontal Component: Five subjects exhibited a rightward horizontal component. The mean MSV was 6.3˚/s, with a minimum of 0˚/s and a maximum of 24.8˚/s, with a standard deviation of 8.5˚/s.

Vertical component: Five subjects exhibited a vertical component, four subjects exhibited downward nystagmus, and one subject exhibited upward nystagmus. The mean MSV was 6˚/s, with a minimum of 0˚/s, a maximum of 18.3˚/s, and a standard deviation of 7.8˚/s.

Torsional component: All subjects exhibited a rightward torsional component. The mean MSV was 20˚/s, with a minimum of 11.8˚/s and a maximum of 40.3˚/s. The standard deviation was 8.3˚/s.

Figure 2 depicts the mean values and standard deviations of MSV of the horizontal component for each experiment. Those of the vertical component are illustrated in Figure 3, while Figure 4 depicts those of the torsional component.

Table 1. MSV (˚/s) of nystagmus.

			Experiment 1			Experiment 2		
Subject	Age (years)	Sex	H	V	T	H	V	T
1	66	M	0	54.6	0	19.8	20.7	11.4
2	65	F	0	22.1	0	13.4	0	13.5
3	37	F	0	36.5	0	12.5	27.6	1.3
4	66	M	0	30.4	0	0	15	11.1
5	31	F	0	63	0	0	52.4	0
6	43	M	0	34	0	0	25.4	1.3
7	45	F	0	33.3	0	0	14.8	13.9
8	73	M	0	33.6	0	0	29.2	13.1
9	60	M	0	33.6	0	0	27.7	19
10	52	F	0	24.7	0	0	19.3	4.9
11	45	F	0	16.8	0	0	9.4	12.8
12	62	F	0	16.7	0	0	10.2	0
Mean	53.8		0	33.3	0	3.8	21	8.5
SD			0	13.8	0	7.1	13.2	6.6

SD = Standard deviation, M = Male, F = Female, H = Horizontal component, V = Vertical component, T = Torsional component.

Table 2. MSV (˚/s) of nystagmus.

	Experiment 3			Experiment 4		
Subject	H	V	T	H	V	T
1	37.2	24	14.1	24.8	0	40.3
2	26.1	0	7	0	11.3	18
3	9.4	20.7	22.3	0	0	16
4	0	30	19.7	14.5	16	17.8
5	12.4	35.9	11.6	10.7	8.9	14.1
6	0	16.3	1.8	0	0	11.8
7	11.2	9.8	11.6	0	0	19.5
8	7.1	30.4	10.3	10	0	32.6
9	12.3	16.3	7	0	18.3	14.1
10	0	14.5	18.4	15.1	0	21.6
11	2.6	3.7	9.3	0	0	17

Continued

12	0	16.2	11.4	0	17.5	17.8
Mean	9.9	18.2	12.	6.3	6	20.
SD	11.6	10.8	5.8	8.5	7.8	8.3

SD = Standard deviation, H = Horizontal component, V = Vertical component, T = Torsional component.

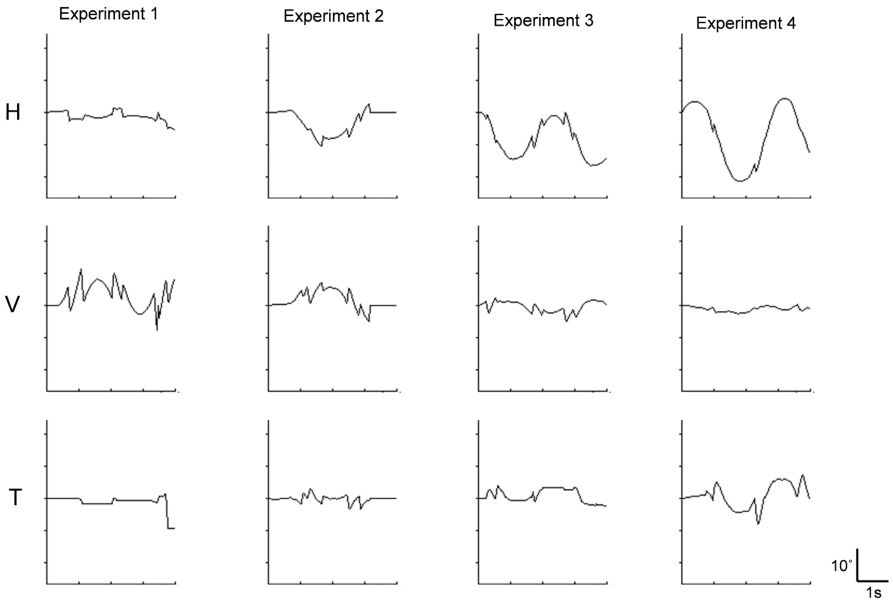

Figure 1. Video-oculography (position trace) of subject 1. The upward deflections in horizontal (H), vertical (V), and torsional (T) eye movements are indicated as being toward the right, upward, and right, respectively. Note the gradual change of the vertical and torsional components.

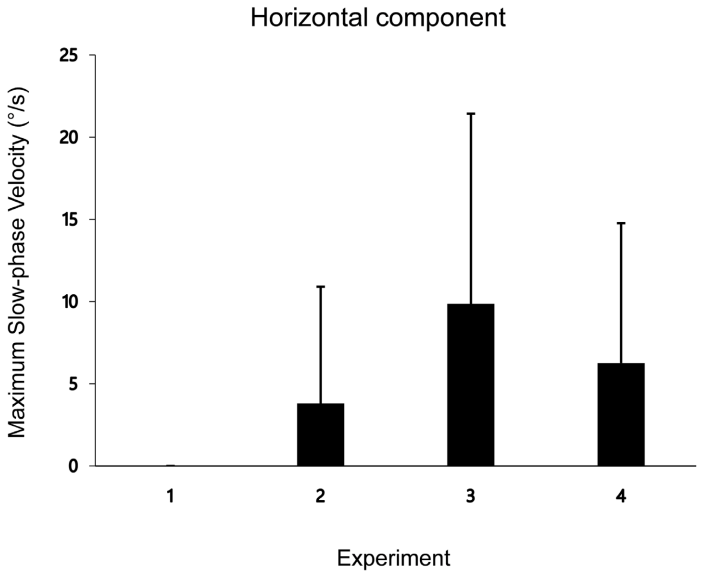

Figure 2. Mean value and standard deviation of maximum slow-phase velocity of the horizontal component of nystagmus.

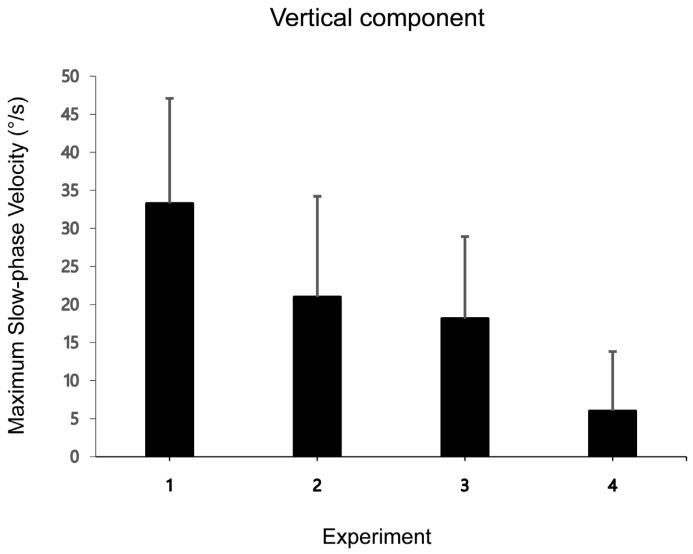

Figure 3. Mean value and standard deviation of maximum slow-phase velocity of the vertical component of nystagmus. The mean value was greatest in Experiment 1, followed by Experiment 2, then Experiment 3, and was smallest in Experiment 4.

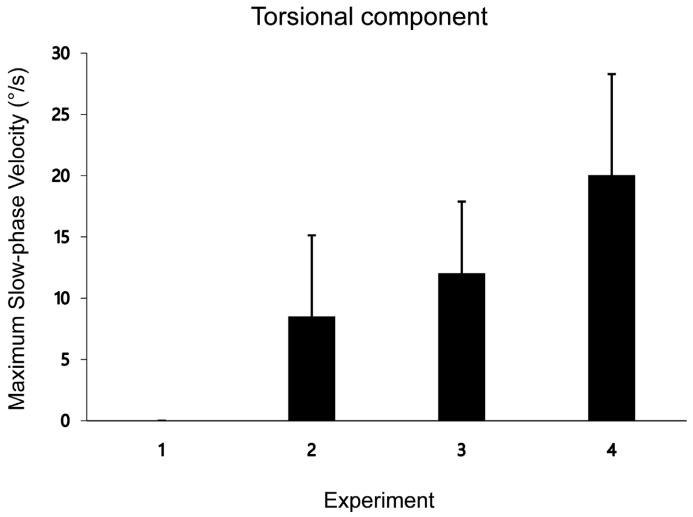

Figure 4. Mean value and standard deviation of maximum slow-phase velocity of the torsional component of nystagmus. The mean value was greatest in Experiment 4, followed by Experiment 3, then Experiment 2, and 0 in Experiment 1.

4. Discussion

We conducted a quantitative analysis of the vertical and torsional VOR in darkness and found that their features and intensity are heterogeneous and exhibit significant individual differences. For instance, in Experiment 1 (pitch rotation), the minimum value of MSV was 16.7˚/s, while the maximum value was 63˚/s, and the standard deviation was 13.8˚/s, indicating significant variability. The great variations in responses complicate the establishment of a normal range. For instance, although the MSV was 17˚/s, which is below average, it remains unclear whether this value represents a pathological decrease or is simply an individual variation.

Therefore, the use of MSV as a measure of SCC function is inappropriate in clinical applications.

The magnitude of response in the caloric test significantly differs among individuals [11]. Therefore, a normal range could not be established; however, in the caloric test, it is possible to determine the difference between the left and right sides. Therefore, the asymmetry (canal paresis) is used as a clinical criteria.

The torsional nystagmus observed during the roll rotation, termed ocular counter-roll (Experiment 4), exhibited variability in both the intensity of the response and the direction of eye movements. Although it sometimes exhibited solely torsional movement, significant individual differences were observed in cases of horizontal/torsional type or vertical/torsional type.

What accounted for the significant individual difference? The cause is attributable to insufficient visual input. When there is a clear target in a bright place, one may focus accurately on that point; however, in a dark place where the target is absent, we speculate that the control function of eye movements through the central nervous system decreases, resulting in gain reduction. Conversely, it might be asserted that performing the examination in a dark place would highlight the function of the inner ear. The caloric test is typically performed in the dark.

What causes jerky nystagmus to manifest in the dark? It is a reflex originating from the SCCs, and the cupula is indeed displaced. How do the SCCs sense angular acceleration? It is obvious that the law of inertia is involved in this mechanism. This principle is Newton's first law, which states that an object at rest tends to remain at rest. Inertial forces act in the endolymph within the long arm, causing a relative movement of endolymph, thereby displacing the cupula.

A previous study [12] demonstrated that the farther the object is from the center of rotation, the higher its acceleration and the stronger the inertial force it experiences. The direction of the inertial force is tangential.

Figure 5 depicts an overview of the movement of the inner ear in Experiment 1. An essential point is that the center of rotation is not the center of SCC. The center of rotation in each experiment is the neck. The inner ear is approximately 0.1m from the center of rotation. Because the inner ear has an eccentric rotation, endolymphatic flow centered on the center of the SCC does not occur. This point was overlooked until now.

Based on the findings of a previous study [12], we propose the "difference of inertial forces" theory to explain how SCC senses angular acceleration. Below is a detailed explanation of our proposed mechanism.

Assume that only the right inner ear is stimulated (**Figure 5**). Focus on one molecule of water at the upper pole of the right anterior SCC. In the case of forward bending in Experiment 1, the water molecule at the upper pole of the anterior SCC receives an inertial force of magnitude ma_1 posteriorly; where m is the mass of one molecule of water, and a_1 is the acceleration of the upper pole of the anterior SCC. An inertial force also arises in the posterior SCC. One molecule of water at the upper pole of the posterior SCC receives an inertial force ma_2; where a_2 is the ac-

celeration of the upper pole of the posterior SCC. The water molecule at the lower pole of the posterior SCC also receives an inertial force of magnitude ma_3; where a_3 is the acceleration of the lower pole of the posterior SCC.

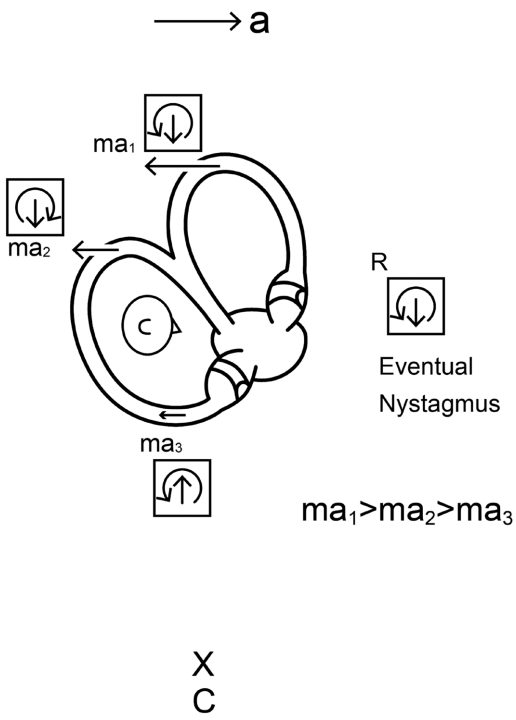

Figure 5. Illustration depicting the "difference of inertial forces" theory. The figure depicts the right anterior and posterior SCCs. When bowing forward, the cupula is displaced due to the difference in the magnitude of the inertial forces exerted on the endolymph. The squares represent the direction of theoretical nystagmus. The lateral SCC is not shown. m = Mass of one molecule of water in the endolymph. a = Acceleration of the head. a_1 = Acceleration of the upper pole of the anterior SCC. a_2 = Acceleration of the upper pole of the posterior SCC. a_3 = Acceleration of the lower pole of the posterior SCC. C = Center of rotation (neck). R = Right.

It was concluded that the acceleration increases as the radius of rotation increases. Since $a_1 > a_2 > a_3$, $ma_1 > ma_2 > ma_3$. The inertial force ma_1 causes an upward movement of the cupula of the anterior SCC and a nerve impulse is generated that causes downbeat nystagmus (torsional component is rightward). The inertial force ma_2 causes a movement to displace the cupula of the posterior SCC forward. The inertial force ma_3 causes a movement to displace the cupula of the posterior SCC backward. However, since $ma_2 > ma_3$, the cupula of posterior SCC is displaced forward, and a nerve impulse is generated that causes downbeat nystagmus (torsional component is leftward). Since $ma_1 > ma_2$, downbeat nystagmus (torsional component is rightward) eventually occurs.

If this hypothesis holds, it provides a compelling explanation of the correlation between the axis of rotation and the stimulated SCCs. For instance, in the traditional rotational test (yaw rotation), the axis of rotation of the head is vertical. The plane of the lateral SCCs is perpendicular to the axis in the chin-down 30˚ posi-

tion. This spatial relationship yields the maximum stimulation.

In Experiment 1, both inner ears were stimulated equally, resulting in a symmetrical activation by the same mechanism. As the torsional components from each side canceled each other out, purely vertical nystagmus eventually occurred. Figure 6 depicts the endolymph movement in four SCCs and the corresponding theoretical nystagmus during forward bending in Experiment 1.

In Experiment 2, the head was rotated 45˚ to the left, which predominantly stimulated the right anterior and left posterior SCCs (Figure 7). Video head impulse test is based on this principle. Unfortunately, it is impossible to stimulate only one SCC; both SCCs are stimulated at the same time. During forward bending, an inertial force is generated backward in the right anterior SCC (Stimulation in Ewald's third law. Same direction as in Experiment 1), and downbeat nystagmus occurs (torsional component is rightward). At the upper pole of the left posterior SCC, an inertial force is generated posteriorly (Inhibition in Ewald's third law. Same direction as in Experiment 1), and downbeat nystagmus occurs (torsional component is rightward). As a result, downbeat nystagmus occurs (torsional component is rightward).

This pattern was observed in nine subjects, where eye movements matched the predicted nystagmus. However, two subjects exhibited no torsional component, and one subject exhibited no vertical component. In three subjects, horizontal components were observed.

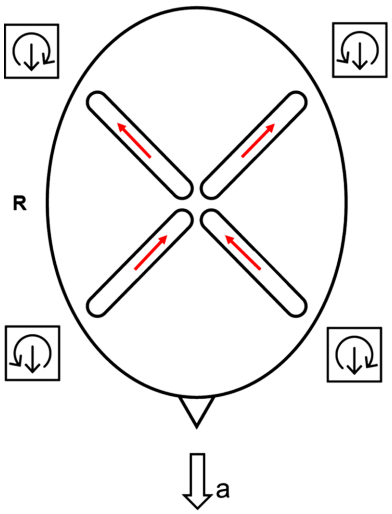

Figure 6. The direction of inertial forces (red arrows) experienced by the endolymph in the upper pole of SCCs during forward bending in Experiment 1. The small squares represent the theoretical nystagmus direction. *a* = Acceleration of the head. R = Right.

Why might the nystagmus have the horizontal component in Experiment 2? One likely explanation is anatomical variation. Della Santina *et al.* and Ichijo reported that the positions of the SCCs exhibited great individual variation [13] [14]. The angle between the anterior SCC and posterior SCC on one side is not always 90˚. Therefore, even with a 45˚ head rotation, the stimulated SCCs may not be pre-

cisely aligned with the direction of motion. Figure 7 depicts the right anterior and left posterior SCCs in the same plane; however, in reality, individual variations exist, and there are subtle angular misalignments. If the head rotation axis deviates from perfect perpendicularity to the posterior SCC plane, it is plausible that a horizontal component emerged during stimulation [15]. The three-dimensional analysis of nystagmus caused by posterior canal type BPPV revealed that many cases exhibited a horizontal component [16]. This finding can also be explained by the same reasoning.

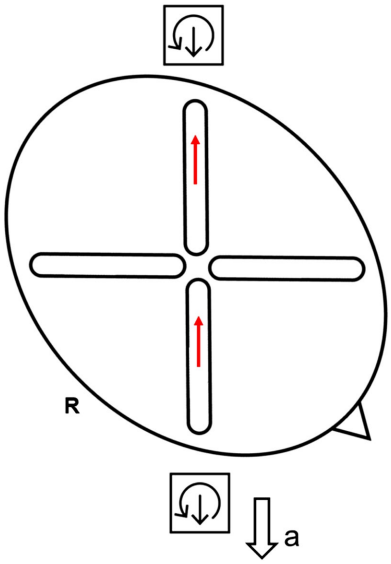

Figure 7. The direction of inertial forces (red arrows) experienced by the endolymph in the upper pole of SCCs during forward bending in Experiment 2. The small squares represent the theoretical nystagmus direction. *a* = Acceleration of the head. R = Right.

In Experiment 3, the head rotation angle was increased to 60˚ (Figure 8). Compared to Experiment 2, there was a difference of 15˚; it is improbable that the right anterior and the left posterior SCCs would suddenly stop being stimulated, indicating that endolymph movement persisted. Acceleration induces inertia forces that affect all objects, including the water molecules in the right anterior and left posterior SCCs.

Similarly, it is obvious that endolymph movements occur in the right posterior and left anterior SCCs. These stimuli trigger nerve impulses that generate upbeat nystagmus (with a rightward torsional component). Figure 3 supports this explanation, indicating that the vertical component of Experiment 3 was smaller than that of Experiment 2. Conversely, Figure 4 depicts that the torsional component has increased.

In Experiment 4, the head rotation was increased by another 30˚ from that of Experiment 3 (Figure 9). According to physics, such a change would not suddenly eliminate stimulation of the right anterior and left posterior SCCs. The right posterior and left anterior SCCs are naturally stimulated. Therefore, there is no essential difference between Experiments 3 and 4; they merely differ by a 30˚ shift

in the direction of movement. Figure 3 supports this explanation, illustrating that the vertical component is smaller compared to that of Experiment 3. However, Figure 4 depicts a further increase in torsional component. The direction of nystagmus that appears from each SCC is the same in Experiment 3 and Experiment 4. We support the interpretation that ocular counter-roll is a reflex driven by SCCs stimulation.

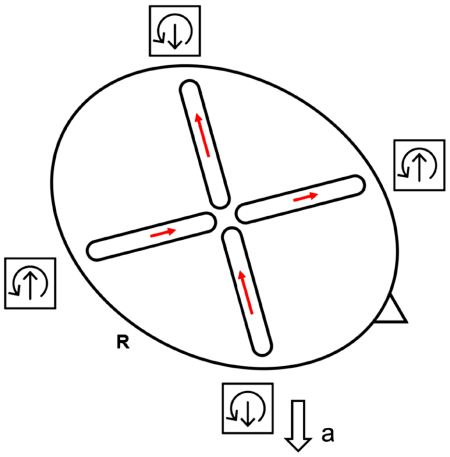

Figure 8. The direction of inertial forces (red arrows) experienced by the endolymph in the upper pole of SCCs during forward bending in Experiment 3. The small squares represent the theoretical nystagmus direction. *a* = Acceleration of the head. R = Right.

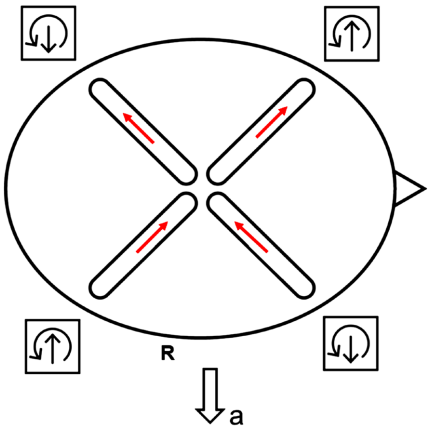

Figure 9. The direction of inertial forces (red arrows) experienced by the endolymph in the upper pole of SCCs during roll rotation to the right in Experiment 4. The small squares represent the theoretical nystagmus direction. *a* = Acceleration of the head. R = Right.

5. Conclusion

Although vertical and torsional VOR in the dark have a significant individual variation, considering the nystagmus direction and intensity, ocular counter-roll can be explained by the SCC theory.

Ethical Approval

All procedures performed in this study were in accordance with the ethical board

of the Hirosaki Medical Association (certificate number is 2024-19).

Acknowledgments

We thank Dr. Kazunori Futai for preparing some of the figures.

Informed Consent

Informed consent was obtained from all individual participants included in this study.

Conflicts of Interest

The authors declare that there is no conflict of interest.

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