Modern Health Science; Vol. 8, No. 2; 2025
ISSN 2576-7291 E-ISSN 2576-7305
https://doi.org/10.30560/mhs.v8n2p1

Is Ocular Counter-Roll Otolith-Ocular Reflex?

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Received: February 25, 2025; Accepted: March 11, 2025; Published: March 12, 2025

Abstract

Purpose

Ocular counter-roll has been thought to be an otolith-ocular reflex. However, there is a hypothesis that it is a semicircular canal (SCC) ocular reflex. This study aimed to validate whether ocular counter-roll can be explained by the SCC theory.

Methods

Experiment 1: Two springs with plastic balls (different turning radius) were fixed to a swivel chair and were rotated suddenly. Experiment 2: Subject was seated and the entire upper body was bent forward 60 degrees with the neck fixed. Experiment 3: Subject was seated, the head was twisted 45 degrees to the right and the entire upper body was bent forward 60 degrees with the neck fixed. Experiment 4 (roll rotation): Subject was asked to tilt his upper body 60 degrees to the left. Eye movements in the dark were observed and recorded using an infrared camera. In Experiments 2, 3 and 4, subjects were four healthy humans.

Results

Experiment 1: Immediately after the movement, the two balls moved tangentially. The ball on the outside spring moved more compared to the ball attached to the inside spring. Experiment 2: In all subjects, nystagmus was vertical. The direction was downward. Experiment 3: In all subjects, nystagmus was vertical/torsional. The direction was downward (torsional component was leftward). Experiment 4: In all subjects, nystagmus was torsional, and the direction was leftward.

Conclusion

Since the role of SCCs is to sense angular acceleration in all directions of 360 degrees in a coordinated manner, bilateral vertical SCCs are stimulated in roll rotation. Therefore, SCC theory can explain the generation of ocular counter-roll. We proposed the “difference of inertial forces” theory to explain the mechanism of vestibulo-ocular reflex.

Keywords: vestibulo-ocular reflex, inertial force, healthy human, angular acceleration, semicircular canal, otolith organ

1. Introduction

The vestibulo-ocular reflex, which originates upon the bending of the cupula in the semicircular canal (SCC), helps maintain the gaze. There are three SCCs on each side since we live in a three-dimensional space. SCCs are present to sense angular acceleration in all directions of 360 degrees, in a coordinated manner. Therefore, it is highly unlikely that SCCs do not respond to certain angular accelerations.

When the head is rotated in the dark, jerky nystagmus with slow and quick phases occurs. Clinically, rotational test can be used to analyze this response. However, due to the limitations of the devices, only lateral SCC can be stimulated. Recently, the head impulse test has been developed to analyze the functions of the vertical SCCs [1]. This test is based on Ewald's first law which states that when a SCC is stimulated, the eyeball rotates in a plane parallel to the SCC. Horizontal rotation induces horizontal nystagmus. Caloric test can also stimulate the lateral SCC on one side and can produce horizontal nystagmus. In benign paroxysmal positional vertigo, only one posterior SCC is stimulated. A three-dimensional analysis of nystagmus caused by this disease showed that this nystagmus is vertical/torsional [2,3] and the eyeball moves in a plane approximately parallel to the lesioned SCC. Animal studies have also confirmed this observation [4]. These studies suggest that Ewald's first law is correct.

The mechanism of the vestibulo-ocular reflex is closely related to the law of inertia (Newton's first law). Since a stationary object has the property of being in that state, when the head rotates, the endolymph in the SCC experiences an inertial force and moves in a direction opposite to that of acceleration. The role of the SCC is to sense the magnitude and direction of angular acceleration based on the movement of the endolymph.

Horizontal rotation stimulates the lateral SCC. Rotation to the right causes horizontal eye movement to the left (slow phase), since the cupula moves leftward. This reaction is purposive. In pitch (vertical) rotation, vertical nystagmus occurs because both vertical SCCs are stimulated simultaneously. Roll rotation produces torsional nystagmus, known as ocular counter-roll. The prevailing theory states that this reaction is derived from otolith organs (otolith organs theory), and a few studies have applied this as a functional test for otolith organs [5].

However, Ichijo et al. reported that nystagmus does not originate from the otolith organs [6-8]. When the head rotates, the endolymph in the SCC is always subjected to an inertial force, therefore, it is possible that the ocular counter-roll is derived from the SCC (SCC theory). To verify the hypothesis that ocular counter-roll may originate from SCCs, we conducted one physical and three human experiments. This study aimed to validate whether ocular counter-roll can be explained by the SCC theory.

2. Materials and Methods

Experiment 1

Two springs with plastic balls were fixed to a swivel chair and were rotated suddenly. One spring had a turning radius of 0.1 m while the other had a turning radius of 0.25 m. The two springs were identical and the two plastic balls had equal mass.

Experiments 2, 3 and 4 involved four healthy subjects (a 42-year-old male, a 60-year-old female, a 65-year-old male and a 66-year-old male).

Experiment 2

The subject was seated and the entire upper body was bent forward 60 degrees with the neck fixed, and the person was asked to return to the sitting position. The exercise was repeated three times. The frequency was approximately 0.33 Hz.

Experiment 3

The subject was seated, the head was twisted 45 degrees to the right and the entire upper body was bent forward 60 degrees with the neck fixed, and the person was asked to return to the sitting position. The exercise was repeated three times. The frequency was approximately 0.33 Hz.

Experiment 4

In the sitting position, the participant was asked to tilt his (her) upper body 60 degrees to the left and then return to the sitting position. The exercise was repeated three times. The frequency was approximately 0.33 Hz.

Eye movements in the dark were observed and recorded using an infrared camera.

3. Results

Movies show the results of all experiments [9].

Experiment 1

Immediately after the movement, the two balls moved tangentially, in a direction opposite to that of rotation. The ball on the outside spring moved more compared to the ball attached to the inside spring.

Experiment 2

In all subjects, nystagmus was vertical. The direction was downbeat during downward movement and upbeat during upward movement.

Experiment 3

In all subjects, nystagmus was vertical/torsional. The direction was downbeat (torsional component was leftward) during downward movement and upbeat (torsional component was rightward) during upward movement.

Experiment 4

In all subjects, nystagmus was torsional. The direction was leftward during downward movement and rightward during upward movement.

4. Discussion

From the results of Experiment 1, it can be concluded that the farther the object from the center of rotation, greater its acceleration and stronger the inertial force it experiences. The direction of the inertial force is tangential.

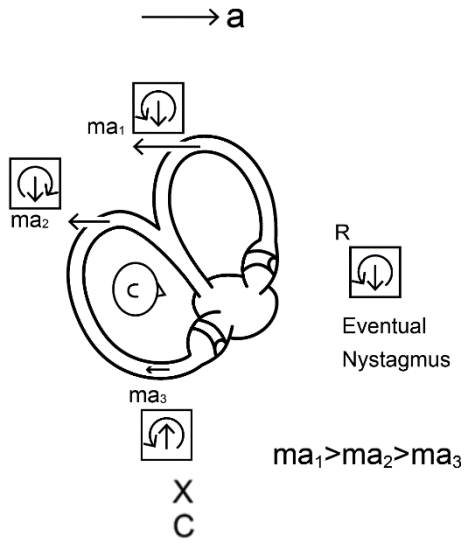

Figure 1.

Illustration depicting the "difference of inertial forces" theory. The figure represents 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. 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 (hip joint).

R = Right.

The results of Experiment 2 are purposive and natural. An overview of the movement of the inner ear is shown in Figure 1. Amazingly, the center of rotation is not the center of SCC. The center of rotation in Experiments 2, 3 and 4 is the hip joint. That is to say, the inner ear is separated from the center of rotation by approximately 0.7 m. Since the inner ear has an eccentric rotation, endolymphatic flow centered on the center of the SCC does not occur. Based on the results of Experiment 1, we propose the "difference of inertial forces" theory to explain the mechanism by which SCC senses angular acceleration (Figure 1). Below is the explanation of our proposed theory.

Assume that only the right inner ear is stimulated. Focus on one molecule of water at the upper pole of the right anterior SCC. In the case of forward bending in Experiment 2, 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 acceleration 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.

From the results of Experiment 1, 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.

In Experiment 2, the left inner ear is stimulated equally, symmetrical phenomenon occurs by the same mechanism. Since torsional components are canceled out, vertical nystagmus eventually occurs. Thus, the observed phenomenon can be explained by the "difference of inertial forces" theory.

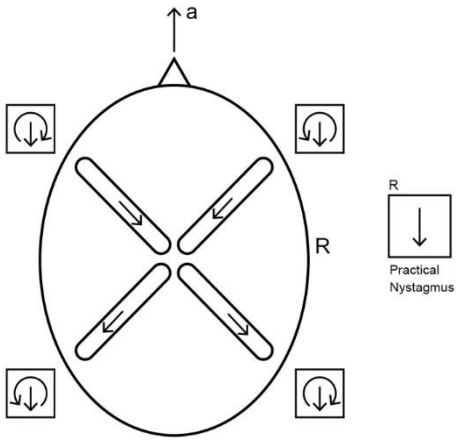

Figure 2.

The direction of the forces experienced by the endolymph in the upper pole of SCCs during forward bending in Experiment 2. The squares represent the direction of theoretical nystagmus.

a = Acceleration of the head.

R = Right.

Figure 2 shows the direction of movement of the endolymph in four SCCs and the theoretical nystagmus during forward bending in Experiment 2.

The results of Experiment 3 are purposive and natural. When the head is rotated 45 degrees to the right, only the left anterior SCC and the right posterior SCC are stimulated (Figure 3). During forward bending, an inertial force is generated backward in the left anterior SCC (same direction as in Experiment 2) and downbeat nystagmus occurs (torsional component is leftward). At the upper pole of the right posterior SCC, an inertial force is generated posteriorly (same direction as in Experiment 2), and downbeat nystagmus occurs (torsional component is leftward). As a result, downbeat nystagmus occurs (torsional component is leftward). Thus, theoretical nystagmus and practical nystagmus were the same.

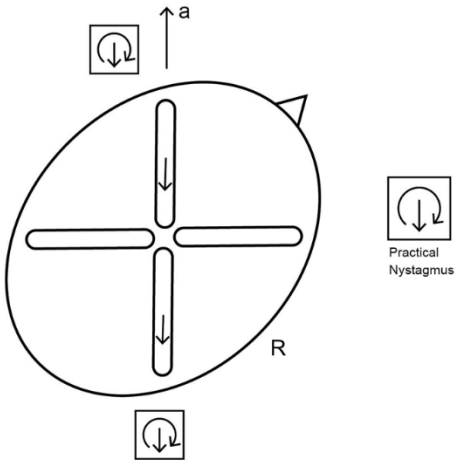

Figure 3.

The direction of the forces experienced by the endolymph in the upper pole of SCCs during forward bending in Experiment 3. The squares represent the direction of theoretical nystagmus.

a = Acceleration of the head.

R = Right.

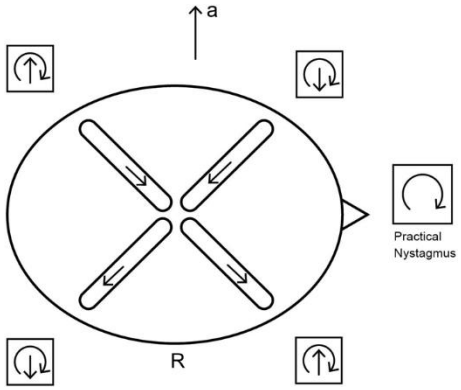

Figure 4.

The direction of the forces experienced by the endolymph in the upper pole of SCCs during roll rotation to the left in Experiment 4. The squares represent the direction of theoretical nystagmus.

a = Acceleration of the head.

R = Right.

The results of Experiment 4 are purposive and natural. During roll rotation, SCCs are stimulated by the same mechanism (Figure 4). Physically, it is highly unlikely that only this movement does not stimulate SCCs. For left anterior SCC, the stimulus is similar to that in Experiment 3. In the endolymph of the upper pole of the left anterior SCC, backward inertial force is produced (same direction as in Experiment 2). As a result, a nerve impulse is generated that causes downbeat nystagmus (torsional component is leftward). In the endolymph of the upper pole of the left posterior SCC, it is obvious that the molecule of water receives an inertial force and relatively moves. The direction of movement is opposite to that induced in Experiment 2. As a result, a nerve impulse is generated that causes upbeat nystagmus (torsional component is leftward). In the endolymph of the right anterior SCC, forward inertial force is produced (direction is opposite to that induced in Experiment 2). As a result, a nerve impulse is generated that causes upbeat nystagmus (torsional component is leftward). In the endolymph of the upper pole of the right posterior SCC, backward inertial force is produced (same direction as in Experiment 2). As a result, a nerve impulse is generated that causes downbeat nystagmus (torsional component is leftward). The vertical components are canceled out, resulting in leftward torsional nystagmus.

As described above, assuming that four SCCs are stimulated by roll rotation, theoretical nystagmus corresponds to practical nystagmus. From the above physical considerations, the mechanism of ocular counter-roll can be explained by the SCC theory.

5. Conclusion

SCC theory can explain the generation of ocular counter-roll. We proposed the “difference of inertial forces” theory to explain the mechanism of vestibulo-ocular reflex.

Acknowledgments

We thank Dr Kazunori Futai for preparing the figures.

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