

Motion Sickness: Its Pathophysiology and Treatment

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Abstract: The pathogenesis of motion sickness includes both inner-ear stimulation by body movement, especially a Coriolis-type stimulus, and optokinetic stimulation due to the shift of the surrounding visual fields. According to Kornhuber, Sakata and others, the vestibular cerebellum also participates in an important way. We conducted this study to elucidate the influence of the vestibular cerebellum on the development of motion sickness. We initially focused attention on the visual suppression test of Takemori et al. as a test for vestibular cerebellar function. We reported a modification of this test, described as postrotatory nystagmus. We employed this test as a rotatory visual suppression test using milder stimulus for patients complaining of motion sickness. The pathogenesis and treatment of motion sickness are also discussed.

Key Words: motion sickness; pathophysiology; treatment

Motion sickness is thought to be an imbalance of the autonomic nervous system in response to a repeated motion stimulus during vehicular travel [1,2]. In ancient times, humans moved on their legs alone; no motion sickness existed because of an adequate adaptation of the body to inner-ear and visual stimuli. When canoes were made, humans first suffered from motion sickness (Fig. 1).

Vivid descriptions of seasickness are found in ancient Greek writings. Various methods of transportation were developed subsequently, along with the remarkable progress of civilization. At each stage, humans suffered from the consequences of their own civilization. The modern era is about to enter space, and the well-known problem of space sickness has drawn a great deal of attention.

The pathogenesis of motion sickness includes both inner-ear stimulation by body movement, especially a Coriolis-type stimulus, and optokinetic stimulation due to the shift of the surrounding visual fields. According to Kornhuber, Sakata and others [3–5], the vestibular cerebellum also participates in an important way.

We conducted this study to elucidate the influence of the vestibular cerebellum on the development of motion sickness (Fig. 2). We initially focused attention on the visual suppression test of Takemori and Cohen [6] as a test for vestibular cerebellar function. We reported a modification of this test, described as postrotatory nystagmus [7,8]. We employed this test as a rotatory visual suppression test (VST) using a milder stimulus for patients complaining of motion sickness.

METHOD

With eyes closed in a dark room, a patient was seated on an electrically controlled rotating chair. This chair was rotated with an angular acceleration of $0.5/\text{sec}^2$. After a constant angular velocity of $90/\text{sec}$ was reached, the rotation continued for 1 minute at that velocity and then was stopped suddenly. During this period, the test subject's eyes were opened in the dark room. After 5 seconds, the room was lighted for 5 seconds, and the subject's gaze was fixed on a target 50 cm to the front. The light was again extinguished, although the test subject's eyes remained open in the dark. As shown in the upper part of Figure 3, the proportion of visual suppression may be expressed as $(A - b)/A \times 100\%$, where the approximate curve for the slow-phase velocity of the postrotatory nystagmus is expressed as A , and the curve of gaze fixation at the target is expressed as b .

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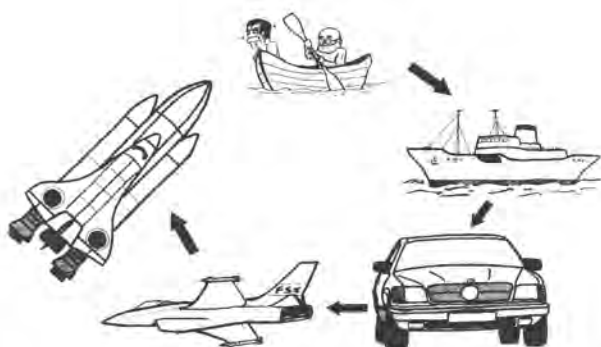


Figure 1. Development of motion sickness in concert with the development of vehicular travel.

According to Teramoto [3], the mean value for rotatoric VST in normal subjects was 61 ± 145 . Representative cases in our study will be presented.

REPRESENTATIVE CASES

Patient 1

A 50-year-old woman presented with a complaint of motion sickness induced by motor vehicle travel. She had this disorder since childhood, exhibiting especially intense symptoms during bus rides. On the left side of Figure 4, the electronystagmography record of the rotatoric VST is shown. On the right side, the result of caloric VST is shown as a reference. As shown on the upper and lower left of the figure, the values were 98% on counterclockwise rotation, respectively. These values are more pronounced than those obtained with caloric VST in normal subjects. After water infusion into the right ear, the value was 86.5%, with a corresponding value of 86.5% after water infusion into the left ear (lower and upper right of Figure 4, respectively), again

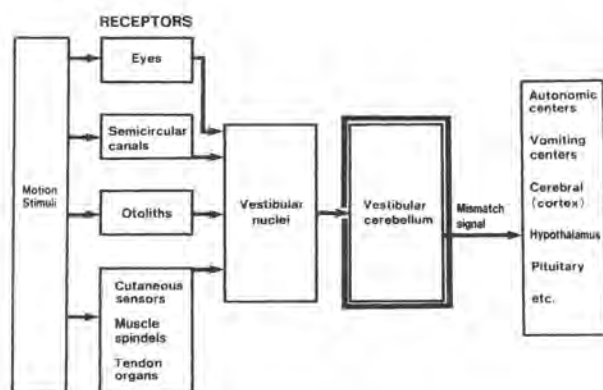


Figure 2. Pathogenesis of motion sickness.

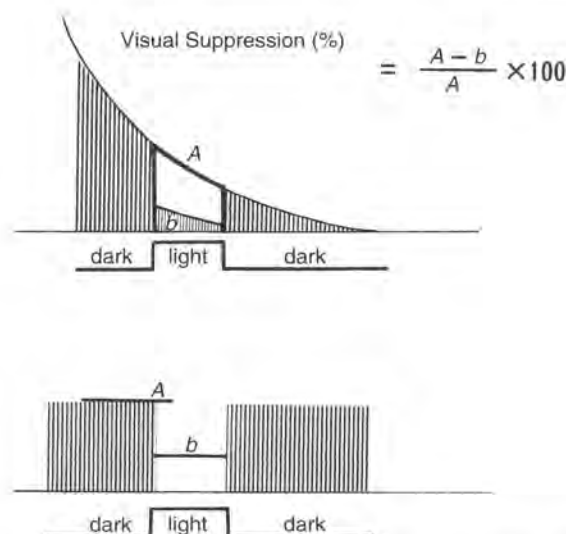


Figure 3. Measurement of visual suppression. The upper diagram shows the recording of the visual suppression test using postrotatory nystagmus, whereas the lower diagram shows the recording using caloric stimulation.

representing more pronounced values than normal. Subsequent values represent only rotatoric VST results.

Patient 2

A 15-year-old girl complained of unpleasant nausea after riding 20 minutes in an automobile, 30 minutes on a bus, or 1 hour on a train. As shown in the upper part of Figure 5, a value of 90.7% was obtained with clockwise rotation and 92.2% with counterclockwise rotation.

Patient 3

A 35-year-old woman complained of motion sickness after riding in an automobile for 20 minutes. She had also suffered from hypotensive episodes since her youth. Rotatory VST studies demonstrated a value of 86.6% with clockwise rotation, as shown in the upper part of Figure 6, and a value of 87.5% with counterclockwise rotation, as shown in the lower part of the figure.

Patient 4

A 52-year-old woman complained of difficulties with automobile travel continuing from childhood. She became nauseated after even 5 minutes of travel. The value of the rotatory VST was 85.7% with clockwise rotation, as shown in the upper part of Figure 7, and 80.9% with counterclockwise rotation, as shown in the lower part of the figure.

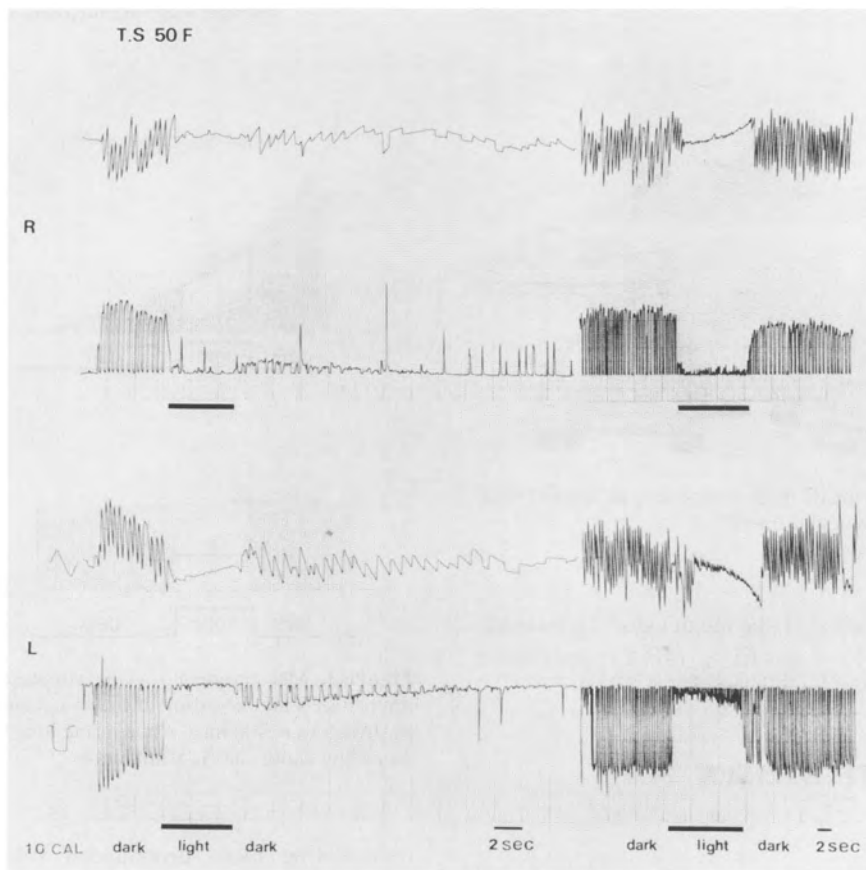


Figure 4. Patient 1 (50-year-old woman).

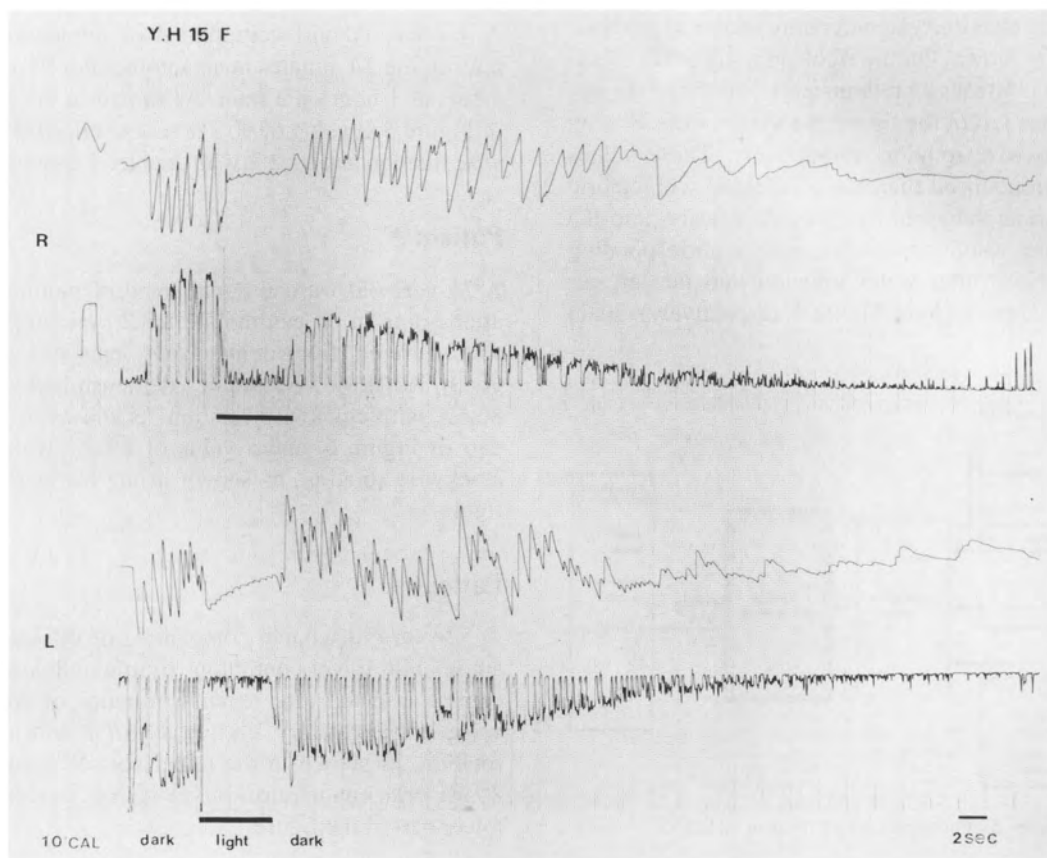


Figure 5. Patient 2 (15-year-old girl).

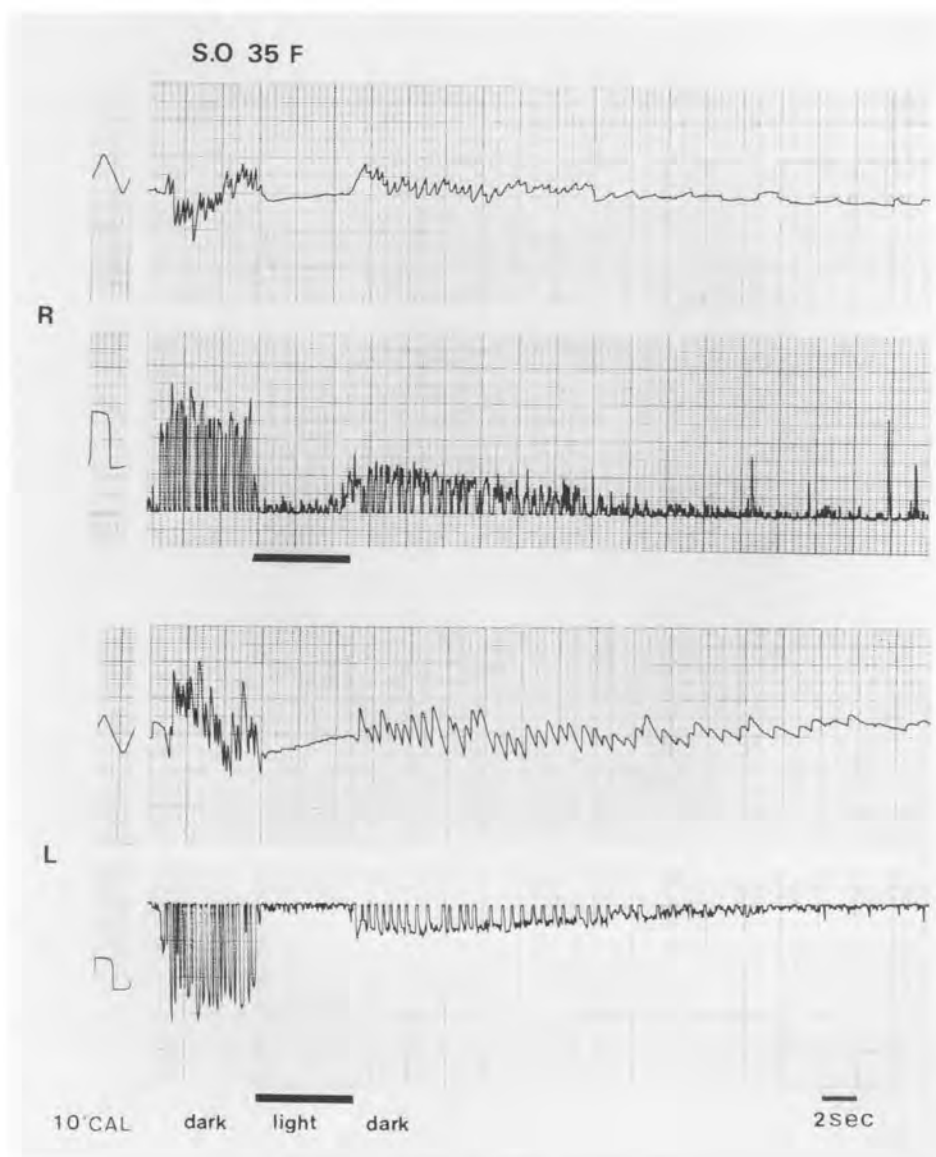


Figure 6. Patient 3 (35-year-old woman).

DISCUSSION

Because the vestibular cerebellum has the function of controlling the vestibuloocular motor system, a relationship with motion sickness has been pointed out long ago. In an experimental dog model, removal of the nodulus and uvula suppressed vomiting and other autonomic nervous system symptoms usually seen in response to vestibular stimulation. A clinical observation confirms this finding: Patients with spinocerebellar degeneration cease to be susceptible to motion sickness. Coriolis stimulation to the inner ear and optokinetic stimulation from shifting scenery have long been considered as the primary causes of motion sickness. However, the degree of inhibition from the vestibular cere-

bellum, especially excessive inhibition, is apparently related intimately to the development of motion sickness.

In our study, we attempted to demonstrate a part of the mechanism of motion sickness using rotatoric VST. As a result, the degree of suppression in patients with motion sickness was found to be more pronounced than that in healthy controls. As Kornhuber and Sakata maintain, the vestibular cerebellum is probably related to the development of motion sickness.

THERAPY

Therapy for motion sickness included oral clonazepam, cinnarizine, and tofisopam three times daily after meals

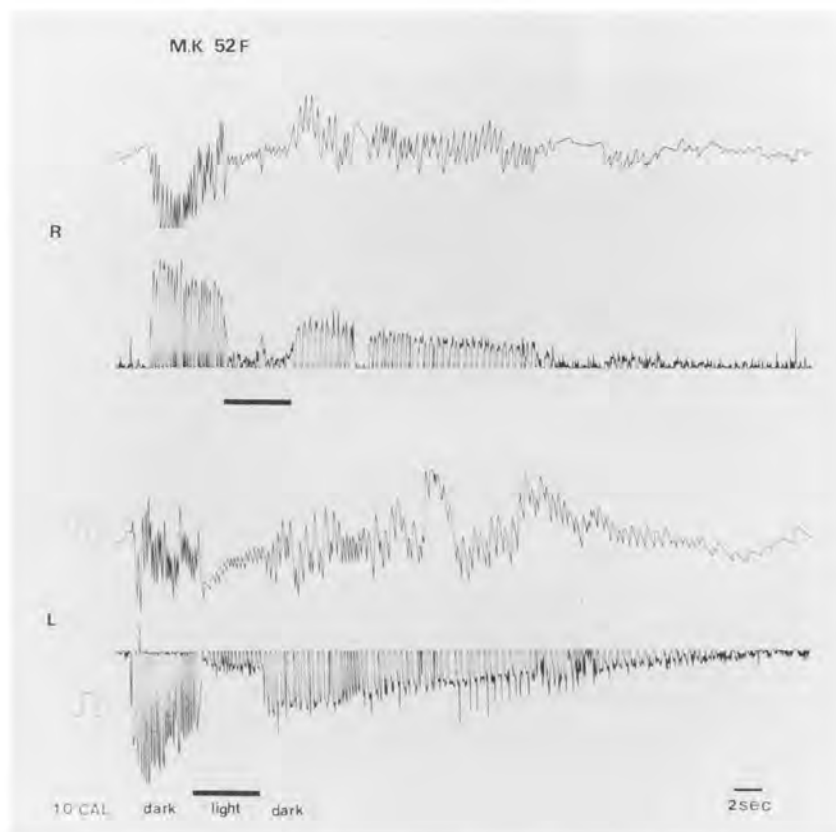


Figure 7. Patient 4 (52-year-old woman).

and imipramine once before sleep for 3 months. As a result, affected patients are often relieved of their motion sickness permanently.

SUMMARY

We performed rotatoric VST as a functional test for the vestibular cerebellum in patients complaining of motion sickness. The results of rotatoric VST appeared to be more pronounced in patients with motion sickness than in normal subjects. Although the stimulus to the inner ear, especially Coriolis and optokinetic stimuli, are important in the generation of motion sickness, the degree of inhibition from the vestibular cerebellum, and in particular excessive inhibition, appears to be intimately related to the development of this condition.

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