Nose blowing can increase middle ear and cerebrospinal fluid pressure. We report a case of biphasic nystagmus induced by nose blowing. The patient showed biphasic, right-beating, and left-beating nystagmus, and complained of spinning vertigo after blowing her nose. The nose-pinched and glottic Valsalva maneuver and the Toynbee maneuver suggested that biphasic nystagmus was induced by increased middle ear pressure. Diseases inducing biphasic nystagmus were ruled out. A left tympanotomy resolved the patient’s vertigo and biphasic nystagmus, whereas right tympanotomy did not alleviate them. When middle ear pressure changes are thought to be the cause of vertigo and nystagmus, a right or left tympanotomy may be considered a possible option for diagnosis and treatment, even if the cause and the affected side of the disease are unknown.

Herein, we report a case showing biphasic nystagmus induced by nose blowing.

Case report

A 55-year-old woman, who complained of spinning vertigo that persisted for 1–2 min after nose blowing, followed by lightheadedness for 1–2 min, for 1 year, provided written informed consent to participate in this study. The project conforms to the code of ethics of the World Medical Association, and was conducted in accordance with the guidelines of the Declaration of Helsinki.

She had visited several other clinics but was referred to our clinic because of the unknown cause of vertigo. There was no vertigo or dizziness other than that at nose blowing, and there were no concomitant cranial nerve symptoms, including cochlear symptoms. There were no episodes of suspected perilymphatic fistula, head trauma, or long-term bed rest. No relevant history was noted.

On examination, the bilateral tympanic membranes were intact (Figure 1(a)), but markedly inflated by nose blowing (Figure 1(b)). The right tympanic membrane inflated immediately upon nose blowing and...
deflated immediately after the end of the nose blowing (Figure 1(c)). Conversely, the left tympanic membrane inflated for 1–2 s after nose blowing and remained inflated for several seconds after the end of nose blowing (Figure 1(c)). Pure tone audiometry and tympanometry were intact, with no difference between the left and right sides. Eustachian tube function was evaluated by acoustic method. Band noise of
5,250–9,310 Hz was introduced through the nasal cavity and sound was recorded from a microphone in the external auditory canal. When the Eustachian tube is opened during swallowing, the microphone in the external auditory canal records the sound pressure change along with the pharyngeal noise. If the Eustachian tube opens with swallowing and closes within 1 s, the Eustachian tube function is considered normal. Eustachian tube function was intact, with no difference between the left and right sides.

On electronystagmography, no spontaneous nystagmus, including periodic alternating nystagmus, was observed. There were no abnormalities in eye tracking, saccades, and optokinetic nystagmus tests. No head positional or positioning nystagmus was observed. No periodic alternating nystagmus (PAN) were observed in the ENG records. The caloric test results were normal, with no difference between the left and right sides. A right-beating nystagmus was observed approximately 22 s after nose blowing (Figure 2(a)), and the patient became aware of spinning vertigo 25 s after nose blowing. Once the nystagmus disappeared (Figure 2(b)), a left-beating nystagmus appeared approximately 45 s after nose blowing (Figure 2(c)), and the direction of rotation of the spinning vertigo was reversed. The nystagmus disappeared approximately 95 s after nose blowing (Figure 2(d)), and the patient was no longer aware of vertigo. Lightheadedness, however, persisted for approximately 30 s. Spinning vertigo and biphasic nystagmus caused by nose blowing were observed in both the sitting and supine positions, and were reproducible upon re-examination.

The nose-pinched Valsalva maneuver, which increased middle ear pressure by pinching the nose and holding the breath, revealed left-beating monophasic nystagmus 30 s after stimulation, and neither the glottic Valsalva maneuver, which increased CSF pressure by trying to close the glottis and holding the breath, nor the Toynbee maneuver, which decreased middle ear pressure by swallowing with the nose pinched, elicited either vertigo or nystagmus. Neither the Tullio phenomenon nor fistula signs were observed. No hyperventilation-induced nystagmus was observed. The short tone burst-evoked cervical and ocular vestibular evoked myogenic potentials (cVEMP and oVEMP) showed normal responses on both sides, but the right threshold was slightly decreased (Figure 3). Temporal bone computed tomography (CT) showed no abnormalities like superior canal dehiscence [5]. Endolymphatic hydrops were not apparent on either side of the inner ear gadolinium-enhanced magnetic resonance imaging (MRI) [13]. Brain MRI also showed no abnormalities.

Although treatments such as round window enforcement were considered, we decided to employ tympanotomy in our outpatient clinic to prevent middle ear pressure changes because the tympanic membrane was inflated due to nose blowing. Initially, phase 1 nystagmus was considered irritative, that is, the right side was considered to be affected because phase 1 nystagmus was right-beating. Additionally,
the thresholds for the right cVEMP and oVEMP were slightly decreased, suggesting a third mobile window or endolymphatic hydrops of the right ear. However, neither the symptoms nor nystagmus was alleviated 2 weeks after right tympanotomy.

Phase 2 nystagmus was considered irritative, that is, the left was considered to be the affected side because phase 2 nystagmus was left-beating and had larger amplitudes and a longer duration. Furthermore, phase 2 nystagmus was similar in terms of latency and direction to the nystagmus induced by the Valsalva maneuver, suggesting that the left side was affected. After left tympanotomy, the patient’s symptoms and nystagmus due to nose blowing disappeared completely. Upon follow-up, after the placement of a left ventilation tube, she displayed no symptoms on nose blowing.

**Discussion**

In this case of biphasic nystagmus induced by nose blowing, left tympanotomy resolved the patient’s vertigo and biphasic nystagmus. Several diseases and laboratory findings are known to show biphasic or direction-changing nystagmus. However, diseases or examinations that induce biphasic nystagmus in a short time in the same head and eye position are uncommon. Also, our patient did not fit into any of the aforementioned diseases or laboratory findings.

Nose blowing increases the middle ear and CSF pressure and can cause a perilymphatic fistula via the implosive and explosive routes [14]. If the same amount of pressure is applied to the inner ear simultaneously via the explosive and implosive routes, the pressures should cancel out and no collapse of the round and oval windows should occur. A perilymphatic fistula is likely to develop when the pressure changes from one of the routes predominates. The same should also be true for nose blowing, that is, if a similar amount of pressure is simultaneously applied to the inner ear via the explosive and implosive routes, the pressure should cancel out and have no effect on the inner ear. Vertigo and nystagmus caused by nose blowing would, therefore, occur when the pressure change from one of the routes predominates.

In our patient, the nose-pinched Valsalva maneuver (increased middle ear pressure) induced irritative nystagmus, but neither the glottic Valsalva maneuver (increased CSF pressure) nor the Toynbee maneuver (decreased middle ear pressure) elicited nystagmus. Changes in middle ear pressure cause changes in the inner ear pressure; however, in cat experiments, a part of the change in inner ear pressure is relieved by

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**Figure 3.** Findings in the cervical and ocular vestibular evoked myogenic potentials (cVEMP and oVEMP). Short tone burst-evoked cVEMP and oVEMP show a normal response on both sides, but the right threshold is slightly decreased. Arrow heads indicate p13 in cVEMP. Arrows indicate n10 in oVEMP.
flowing out of the cochlear and vestibular aqueduct (mainly the cochlear) into the spinal space [15]. In our patient, nose blowing might have caused a higher middle ear pressure than CSF pressure, leading to vertigo and nystagmus.

The patient’s bilateral tympanic membranes were markedly inflated by nose blowing. Since left tympanotomy eliminated not only phase 2 nystagmus but also phase 1 nystagmus, both phase 1 and 2 nystagmus could have been due to the left middle ear pressure changes. However, it is not clear what was caused by the left middle ear pressure changes because various examinations were negative for the presence of a third window or endolymphatic hydrops; there may have been a small perilymphatic fistula [4], a small third mobile window, such as superior canal dehiscence [5], or proximity or contact between the stapes foot plate and the endolymphatic membrane [6].

The nose-pinched Valsalva maneuver also elicited left irritative nystagmus, supporting the hypothesis that the increased left middle ear pressure caused the irritative left-beating nystagmus. If only the left side was affected, a left paralytic right-beating nystagmus was induced, followed by left irritative left-beating nystagmus, although the order of the paralytic and irritative nystagmus was unusual.

The cause of phase 1 nystagmus is still unknown, and it may be difficult to explain it collectively with the phase 2 nystagmus induced by the increased pressure in the left middle ear. The phase 1 right-beating nystagmus could have been left paralytic nystagmus induced by negative pressure in the left middle ear because the patient was breathing deeply before blowing the nose. However, no deflation of the tympanic membrane due to deep breathing before nose blowing was apparent during the observation of the tympanic membrane. Furthermore, the right Eustachian tube opened more easily than the left, although the Eustachian tube function was normal on both sides.

Right irritative nystagmus was induced by increased right middle ear pressure via the right Eustachian tube, and then left irritant nystagmus was induced by increased left middle ear pressure via the left Eustachian tube. The right tympanic membrane inflated immediately after nose blowing and deflated quickly, whereas the left tympanic membrane inflated 1–2 s after nose blowing and did not deflate for several seconds. The left tympanic membrane inflated only 1–2 s later than the right tympanic membrane, but the left-beating nystagmus was induced more than 20 s after the right-beating nystagmus. There was a significant difference in these time lags. Additionally, right tympanotomy did not eliminate phase 1 nystagmus, whereas left tympanotomy eliminated both phase 1 and phase 2 nystagmus.

It is possible that the hypermobility of the stapes may have made the inner ear more susceptible to stimulation. Increased left middle ear pressure might cause the stapes to deviate in one direction, and normalization of the middle ear pressure causes the stapes to deviate in the opposite direction, resulting in biphasic nystagmus. However, it is difficult to explain the time lag between the onset of right-beating and left-beating nystagmus.

In this case, although the sequence was atypical, left tympanotomy could have been performed first as the stronger and longer left-beating nystagmus was irritative. In the future, we need to make more careful decisions about the side the tympanotomy should be initiated.

Ultimately, the patient’s symptoms and biphasic nystagmus resolved after left tympanotomy and ventilation tube placement, although the cause of the patient’s biphasic nystagmus could not be confirmed.

**Conclusion**

We reported a case showing biphasic nystagmus induced by nose blowing.

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**Informed consent statement**

Patient provided written informed consent to participate in this study.

**Disclosure statement**

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