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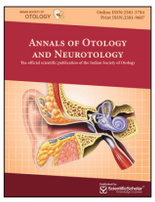

*Case Report*

# Hyperventilation Induced Nystagmus: An Unusual Sign of Unruptured Intracranial Aneurysm

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## ABSTRACT

Intracranial aneurysms (IA) often remain asymptomatic until rupture, with vertigo as a potential overlooked symptom. This report highlights the role of videonystagmography (VNG) in detecting eye movement abnormalities, leading to the diagnosis of an unruptured IA. A 62-year-old male presented with a three-year history of vertigo, unilateral tinnitus, and progressive hearing loss, alongside a past diagnosis of trigeminal neuralgia. Neuro-otological evaluation revealed hyperventilation-induced nystagmus (HVIN) on VNG and abnormal sway on Unterberger testing with craniocorpography (CCG). Subsequent MRI identified a basilar artery aneurysm, successfully treated with coiling. The patient's condition improved significantly over three months. This case emphasizes the importance of early detection through neuro-otological assessment, particularly VNG, in diagnosing and managing unruptured aneurysms.

**Keywords**: Hyperventilation induced nystagmus, Intracranial aneurysms, Vertigo, Videonystagmography

## INTRODUCTION

Intracranial aneurysms (IA) are weak dilatations of major bifurcating cerebral arteries, commonly referred to as saccular aneurysms.[1] Earlier, these were diagnosed at the time of rupture when they presented as subarachnoid haemorrhage. However, with the advent of computed tomography (CT) and magnetic resonance imaging and angiography (MRI and MRA), the incidental diagnosis of unruptured intracranial aneurysm (UIA) has increased.[2] The prevalence of UIAs is estimated by neuroimaging and post-mortem studies in 3.6–6% of the general population.[3] Most of the UIAs are asymptomatic or present with non-specific symptoms like headaches or isolated cranial nerve palsies, thus making the diagnosis difficult.[4] An underrated and overlooked symptom is vertigo. While there is literature suggesting the importance of vertigo in diagnosing UIAs, there is still very little understanding of the pathophysiology behind it, and therefore, it is often overlooked.[2,5,6]

Videonystagmography (VNG) is a useful test to evaluate central and vestibular disorders. Hyperventilation-induced nystagmus (HVIN) induces neurophysiological changes that can unmask latent central, vestibular, and retrocochlear lesions. On the basis of nystagmus patterns detected on VNG, the need for further radiological imaging to diagnose central lesions can be made.[7]

In this case report, we would like to present the use of hyperventilation-induced nystagmus as a sign to warrant further evaluation and potentially pick up life-threatening UIAs. Early diagnosis and timely management could prevent rupture and subsequent consequences like stroke or subarachnoid haemorrhage, which can be fatal.

## CASE REPORT

A 62-year-old male came to Vertigo and Ear Clinic, Jaipur, with complaints of vertigo and tinnitus for 3 years.

The patient's main complaint was recurrent unsteadiness and dizziness lasting less than 30 seconds. It was often associated with disorientation and lightheadedness for the last 3 years. There were no triggers which aggravated the symptoms and no associated headaches. He felt well in between the spells. He had tinnitus in the left ear for 3 years, which was fluctuating and high-pitched. He also complained of progressive hearing loss in the left ear.

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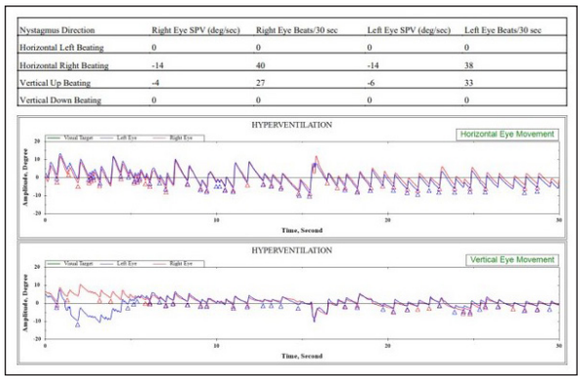



**Figure 1:** VNG findings of hyperventilation.

The patient had co-existing type 2 diabetes mellitus, hypertension, and coronary artery disease, for which he underwent coronary artery bypass grafting in 2013. He was also being treated for trigeminal neuralgia with carbamazepine and gabapentin.

The otoscopic examination was normal. There was gradually sloping sensorineural loss in the left ear and minimal hearing loss in the right ear on pure tone audiometry, with a Speech Discrimination Score of 85% in the left ear and 90% in the right ear.

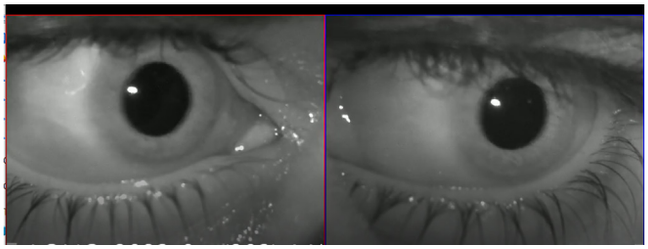
The findings of the vestibular evaluation are mentioned below:

1. Videonystagmography (VNG)
    1. Hyperventilation—right and upbeating nystagmus present

       [Figure 1] [Video 1]

       No spontaneous nystagmus.

       Other oculomotor and vestibular tests were within normal limits.



**Video 1:** Vng - hyperventilation induced nystagmus - right and upbeating nystagmus.

2. Subjective Visual Vertical: Normal
3. Unterberger test (Fukuda): Abnormal Sway: 30 cm [Figure 2]
4. Video Head Impulse Test: Normal

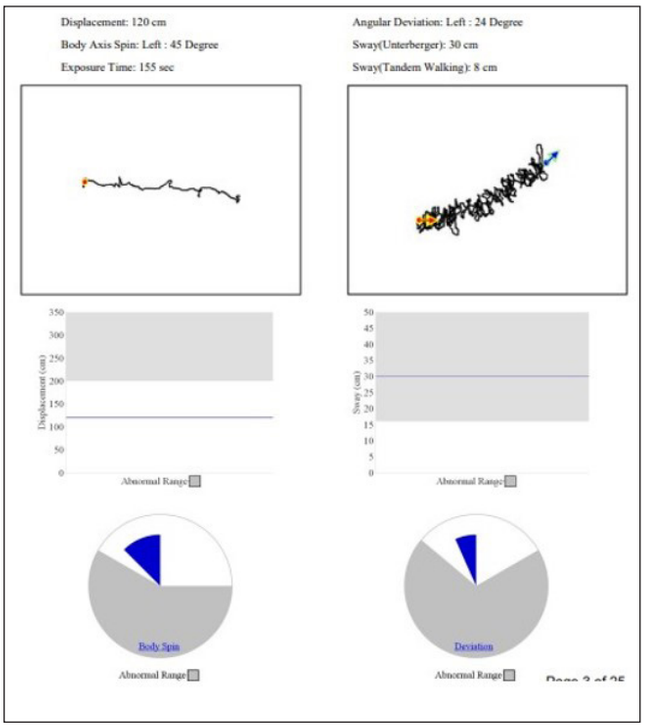



**Figure 2:** Craniocorpography.

The patient was advised Gadolinium contrast-MRI Brain to rule out neurovascular conflict as there was right upbeating nystagmus on hyperventilation, and sway was seen during Unterberger test.

MRI revealed a large contrast-filled outpouching/fusiform lobulated aneurysm of size 31 × 19 × 19 mm arising from the basilar artery involving the confluence of right and left vertebral arteries on the left cerebellopontine angle region, which was causing a significant mass effect over the left half of the pons and displacing it towards the right side. The mass effect over the cranial nerves and extensions was suggestive of aneurysm.

The patient was then advised for a neurosurgical opinion and was planned for coiling of the aneurysm, which was successfully done. The patient was followed up after 3 months of surgery telephonically and reported improvement in symptoms of vertigo and facial pain.

## DISCUSSION

Vertebral-basilar aneurysms comprise 5–10% of all aneurysms.[8,9] The most common saccular aneurysms of the posterior circulation are (1) at the basilar bifurcation, (2) along the basilar trunk, and (3) at the junction of the vertebral and the posterior inferior cerebellar artery. Vertebral-basilar aneurysms are classified as small (<12.5 mm), large (12.5–25

mm), and giant (>2.5 cm). The aneurysms are named for the nearest vessel junction to the origin of the aneurysmal sac, e.g., basilar-labyrinthine artery aneurysm. Basilar artery aneurysms usually remain occult until their rupture. Commonly, there are initial warning leaks and (at times) significant rebleeding.[8,9] The spectrum of symptoms may range from transient ischaemic attacks to sudden brainstem death.[10,11] The manifestations of basilar artery aneurysms are related to ischaemia, caused by mass effect, dissection (rare), or rupture.[12] Classic symptoms include vertigo, tinnitus, facial weakness, facial paraesthesias, dysarthria, limb weakness, drop attacks, headache, etc.[13] Indeed, basilar artery aneurysms may mimic cerebellopontine angle tumours, Meniere's disease, tic douloureux, or pheochromocytoma.[14] Vertebral-artery ischaemia gives rise to dizziness in 87% of cases,[15] visual disturbances in 44%, ataxia (32%), spontaneous nystagmus (27%), and sensorineural hearing loss (22%). Transient ischaemic attacks—similar to those of carotid origin—have also been reported.[16]

### Mechanism of Hyperventilation Induced Nystagmus (HVIN)

Hyperventilation causes "ventilation that exceeds metabolic needs."[17]

Increases serum pH and lowers the concentration of ionised calcium;

It reduces both the cerebral and inner ear circulation.

Reduces the tissue oxygenation through a left shift of the haemoglobin-oxygen dissociation curve

Lowers both the middle ear pressure [(partial pressure of CO2) $pCO_2$ between the middle ear and blood] and the intracranial pressure.[18–20]

Hyperventilation (HVT) acts through non-vestibular mechanisms on several parts of the vestibular system, and it is able to highlight the neurophysiological effects of both central and peripheral lesions by interfering with the mechanisms of genesis and control of oculomotor responses.

Several mechanisms have been proposed to explain the effects of HVT on the vestibular system[18,21–32]:

1. In acute neuritis, the neuronal excitability increases in partially damaged fibres due to the reduction of $pCO_2$ and of $H^+$ and $Ca^{2+}$ concentration, which leads to transitory up-regulation of the central compensation mechanisms of the vestibular deficit or the activation of threshold channels.
2. In the early stages of acute neuritis and acoustic neuroma, there is a mechanism of the temporary improvement of the conduction along demyelinated fibres causing excitatory patterns and might also cause the inhibition of central-type nystagmus in multiple sclerosis.
3. HVIN in perilymphatic fistulas and Superior Canal Dehiscence Syndrome is explained by the changes in intracranial and perilymphatic pressure.
4. In acute and compensated vestibular neuritis, in acoustic neuromas, and some phases of Menière's disease, the breakdown of central compensatory mechanisms might cause paretic HVIN.
5. Metabolic effects on cerebellar $Ca^{2+}$ channels might cause a central-type HVIN in cerebellar diseases.

A detailed history, clinical examination, and neuro-otological evaluation could pick up the unruptured intracranial aneurysm, and then MRI helped in clinching the diagnosis.

## CONCLUSION

A 62-year-old patient presented with complaints of vertigo with subsequent decreased hearing, pulsatile tinnitus, and facial pain. The neuro-otologic evaluation led to the detection of HVIN and increased sway on the Unterberger test. Suspecting a neurovascular conflict, an MRI brain was advised, which revealed a giant basilar artery aneurysm. HVIN raised the suspicion and allowed successful treatment of a potentially lethal condition.

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## REFERENCES


1. Hackenberg KA, Hänggi D, Etminan N. Unruptured Intracranial Aneurysms: Contemporary Data and Management. Stroke 2018;49:2268–75.
2. Gkasdaris G, Tabatabaei P, Kourtopoulos H, Birbilis T. Vertigo: Could this Symptom Indicate the Existence of an Unruptured Intracranial Aneurysm? Maedica 2021;16:555.
3. Matsuda M, Watanabe K, Saito A, Matsumura KI, Ichikawa M. Circumstances, Activities, and Events Precipitating Aneurysmal Subarachnoid Hemorrhage. J Stroke Cerebrovasc Dis 2007;16:25-9.
4. Boulouis G, Rodriguez-Régent C, Rasolonjatovo EC, Hassen WB, Trystram D, Edjlali-Goujon M, et al. Unruptured Intracranial Aneurysms: an Updated Review of Current Concepts for Risk Factors, Detection and Management. Rev Neurol (Paris) 2017;173:542–51.
5. Oh D, Lee ES, Shin DS, Lee TK. A Rare, but Dangerous Cause of Vertigo: Central Positional Vertigo Due to a Large Vertebral Artery Aneurysm. Otol Neurotol 2019;40:e170–2.
6. Batjer HH, Stieg PE, Schwartz RB. A Case of Acute Vertigo with Incidental Aneurysms. Neurosurgery 1995;36:827–33
7. Califano L, Melillo MG, Vassallo A, Mazzone S. Hyperventilation-Induced Nystagmus in a Large Series of Vestibular Patients. Acta Otorhinolaryngol Italica 2011;31:17.
8. Drake CG. The Treatment of Aneurysms of the Posterior Circulation. In: Carmel PW, Fleischer A, Geiger L, et al., Editors. Clinical Neurosurgery. Vol. 26. Baltimore: Williams & Wilkins; 1979. p. 96–144.
9. Peerless SJ, Drake CG. Management of Aneurysms of Posterior Circulation. In: Youmans JR, editor. Neurological Surgery. Philadelphia: WB Saunders Co 1982:1715–63.
10. Stewart RM, Samson D, Diehl J, Hinton R, Ditmore QM. Unruptured Cerebral Aneurysms Presenting as Recurrent Transient Neurologic Deficits. Neurology 1980;30:47–51.
11. Hirsh LF, Gonzalez CF. Fusiform Basilar Aneurysm Simulating Transient Ischemic Attacks. Stroke 1979;10:598–601.
12. Martini E. Intracranial Dissecting Aneurysm. Arch Psychiatr Nervenkr 1979;227:17–21.
13. Blau IN, Spillane JA. Vertebro-basilar Insufficiency and the ENT Surgeon, with an Approach to the Giddy Patient. Clin Otolaryngol 1981;6:73–8.
14. Emanuele MA, Dorsch TR, Scarff TB, Lawrence AM. Basilar Artery Aneurysm Simulating Pheochromocytoma. Neurology 1981;31:1560–1.
15. Valvasorri GE. Vertigo in Vertebral-Basilar Iinsufficiency. Rev Laryngol Otol Rhinol 1985;106:215–8.
16. Love IT. Basilar Artery Migraine Presenting as Fluctuating Hearing Loss and Vertigo. ORL J Otorhinolaryngol Relat Spec 1978;86:450–8.
17. Sakellari V, Bronstein AM, Corna S, Hammon CA, Jones S, Wolsley CJ. The Effects of Hyperventilation on Postural Control Mechanisms. Brain 1997;120:1659–73.
18. Davis FA, Becker FO, Michael JA, Sorensen E. Effects of Intravenous Sodium Bicarbonate, Disodium Editate (Na2EDTA) and Hyperventilation on Visual and Oculomotor Signs in Multiple Sclerosis. J Neurol Neurosurg Psychiatry 1970;33:723–32.
19. Ikarashi F, Takahashi S, Yamamoto Y. Carbon Dioxide Exchange via the Mucosa in Healthy Middle Ear. Arch Otolaryngol Head Neck Surg 1999;125:975–8.
20. Ikarashi F, Tsuchiya A. Middle Ear Gas Exchange via the Mucosa: Estimation by Hyperventilation. Acta Otolaryngol 2008;128:9–12.
21. Bance ML, O'Driscoll M, Patel N, Ramsden RT. Vestibular Disease Unmasked by Hyperventilation. Laryngoscope 1998;108:610–4.
22. Walker MF, Zee DS. The Effect of Hyperventilation on Down-Beat Nystagmus in Cerebellar Disorders. Neurology 1999;53:1576–9.
23. Robichaud J, Roches H, Bance ML. Is Hyperventilationinduced Nystagmus More Common in Retrocochlear Vestibular Disease than in End-Organ Vestibular Disease? J Otolaryngol 2002;31:140–3.
24. Minor LB, Halswanter T, Strauman D, Zee DS. Hyperventilation-Induced Nystagmus in Patients with Vestibular Schwannoma. Neurology 1999;53:2158–67.
25. Choi KD, Kim JS, Kim HJ, Koo J-W, Kim JH, Kim C-Y, et al. Hyperventilation-Induced Nystagmus in Peripheral vestibulopathy and cerebellopontine angle tumor. Neurology 2007;69:1050–9.
26. Minor LB, Solomon D, Zinreich JS, Zee DS. Sound- and/or Pressure-Induced Vertigo Due To Bone Dehiscence of the Superior Semicircular Canal. Arch Otolaryngol Head Neck Surg 1998;124:249–58.
27. Minor LB, Carey JP, Cremer PD, Lustig LR, Streubel S-O, Ruckenstein MJ. Dehiscence of Bone Overlying the Superior Canal as a Cause of Apparent Conductive Hearing Loss. Otol Neurotol 2003;24:270–283.
28. Manto MU, Bosset P. A second Mechanism of Increase of Cerebellar Hypermetria in Humans. J Physiol 2003;547:989–94.
29. Matsuo T. Vestibular Neuritis: Serum and CSF Virus Antibody Titer. Auris Nasus Larynx 1986;13:11–34.
30. Matsuo T, Sekitani T, Honio S, Imate Y, Inokuma T. Vestibular Neuronitis: Pathogenesis in the View of Virological Study of CSF. Acta Otolaryngol 1989;468:365–9.
31. Kuwabara S, Misawa S, Mori Mori, Tamura N, Kubota M, Hattori T. Long Term Prognosis of Chronic Inflammatory Demyelinating Polyneuropathy: A Five-Year Follow-Up of 38 Cases. J Neurol Neurosurg Psychiatry 2006;77:66–70.
32. Somjen GG, Allen BW, Balestrino M, Aitken PG. Pathophysiology of pH and $Ca^{2+}$ in Bloodstream and Brain. Can J Physiol Pharmacol 1987;65:1078–85.


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