

# Evaluation of Vestibular Function in Diagnosis of Vestibular Schwannomas

Xue-qing ZHANG<sup>1, 2, 3, 4, 5</sup>, Tai-sheng CHEN<sup>1, 2, 3, 4, 5</sup>, Wei WANG<sup>1, 2, 3, 4, 5#</sup>

<sup>1</sup>Department of Otorhinolaryngology Head and Neck Surgery, Tianjin First Central Hospital, Tianjin 300192, China

<sup>2</sup>Institute of Otolaryngology of Tianjin, Tianjin 300192, China

<sup>3</sup>Key Laboratory of Auditory Speech and Balance Medicine, Tianjin 300192, China

<sup>4</sup>Key Clinical Discipline of Tianjin (Otolaryngology), Tianjin 300192, China

<sup>5</sup>Otolaryngology Clinical Quality Control Centre, Tianjin 300192, China

© Huazhong University of Science and Technology 2021

**[Abstract]** Vestibular schwannomas (VS) are benign tumors of the vestibular nerve. The common first symptoms are hearing loss and tinnitus, followed by imbalance, vertigo, and facial nerve involvement. The subjective symptoms of VS patients are not consistent with the severity of vestibular lesions and the results of vestibular tests, which often interfere with clinicians' diagnoses. Thus, the main screening and diagnostic methods for VS are audiometry and magnetic resonance imaging (MRI), ignoring the evaluation of vestibular function at the source of pathological lesions. With the development and improvement of vestibular evaluation technology and its wide application in the clinic, modern vestibular examination technology can reflect the severity and frequency of vestibular lesions and compensation from multiple perspectives, providing an objective basis for the diagnosis and treatment of vestibular diseases. In this report, we review the results and characteristics of vestibular tests in VS patients and further clarify the clinical value of vestibular function assessment in the diagnosis and treatment of VS.

**Key words:** vestibular schwannomas; vestibular function test; vestibular compensation; vertigo

Vestibular schwannoma (VS) is a benign neoplasm arising from the Schwann cells of the vestibular (8th cranial) nerve<sup>[1]</sup>. The slow-growing (1–2 mm per year) tumors account for approximately 6% of all brain tumors and occupy up to 80%–90% of the cerebellopontine angle (CPA) region<sup>[2–4]</sup>. The overall incidence of VS is 1.4 per 100 000 per year and remains relatively stable<sup>[5]</sup>.

The clinical manifestations of VS are varied. Auditory symptoms such as unilateral sensorineural hearing loss (94%) and tinnitus (83%) are the common first symptoms at diagnosis<sup>[6, 7]</sup>. The frequency, severity, and progression of the vestibular symptoms such as vertigo, dizziness, and postural instability vary widely (17%–75% of the patients), but they are likely underreported<sup>[8, 9]</sup>.

The main screening and diagnostic methods for VS are audiometry and magnetic resonance imaging (MRI), but the evaluation of vestibular function at the source of pathological lesions has gained little attention. As the gold standard of VS diagnosis, MRI cannot be widely used in the screening of patients with vestibular symptoms owing to the high costs<sup>[10]</sup>. As a result, VS

may be misdiagnosed as peripheral diseases such as sudden deafness with vertigo, vestibular neuritis (VN), vestibulopathy, and Meniere's disease (MD).

With improvements in vestibular-function detection technology and vestibular pathology cognition, current vestibular function tests are more focused on the diagnosis of VS. Given that the onset of VS is usually insidious and gradual, the severity of patients' subjective symptoms is often inconsistent with objective functional lesions. Modern vestibular function tests have been able to reflect the severity, frequency, and compensation from multiple angles, providing a factual basis for the diagnosis and treatment of vestibular diseases, which have important clinical and diagnostic significance.

## 1 VS AND DIAGNOSIS

VS has three stages of growth—active growth, static, and retreat pattern—and this growth pattern includes progressive growth, stable growth, and fluctuating growth<sup>[11]</sup>. Clinical characteristics vary with progression and mainly depend on tumor size, anatomic location, growth rate, direction of development, blood supply, and cystic changes<sup>[6, 12, 13]</sup>. However, the natural evolution of VS is complex, and the growth pattern

Xue-qing ZHANG, E-mail: zhangxq1993@yeah.net

#Corresponding author, E-mail: wwei1106@hotmail.com

and rate vary across patients at different stages. As the tumor grows, it gradually compresses the surrounding structures, including the auditory nerve, facial nerve, trigeminal nerve, abducens nerve, cerebellum, and brainstem, eventually resulting in corresponding symptoms<sup>[14]</sup>. Although VS mostly originates from the vestibular nerve, which is the first to be affected, there are subtle or no vestibular symptoms in patients with VS. In contrast, auditory symptoms such as hearing loss and tinnitus are the common first symptoms presenting at the time of diagnosis. Hence, the screening and diagnosis of VS mainly rely on clinical symptoms, audiometry, and MRI<sup>[7]</sup>, whereas the evaluation of the vestibular function at the source of pathological lesions has not attracted enough attention. With the development and improvement of modern vestibular function tests, various tests have gradually played a role in the clinical diagnosis of VS patients<sup>[15]</sup>.

## 2 CHARACTERISTICS OF VESTIBULAR NERVOUS SYSTEM LESIONS IN VS

There are two types of vestibular lesions—central and peripheral; of these, peripheral lesions are located in the nucleus of the vestibular nerve and its below<sup>[7]</sup>. Therefore, VS belongs to the category of peripheral vestibular lesions. Vestibular lesions associated with VS have the following characteristics:

(1) Most tumors occur in the peripheral part of the vestibular nervous system and present as vestibular dysfunction in various vestibular tests. At present, it is unclear whether VS is a central or peripheral disease. VS can be divided into internal auditory canal, cistern, brainstem, and cranial hypertension stages. When VS is in the brainstem and cranial hypertension stage, the patients present symptoms of central diseases, which becomes central clinical disease and shows features of central examination results, such as Bruns nystagmus, metagenesis nystagmus in the gaze test, and type III waves in the smooth pursuit test<sup>[13, 16]</sup>.

(2) VS presents with an insidious progression of vestibular dysfunction and is generally asymptomatic because of the timely establishment of vestibular compensation<sup>[7]</sup>. The results of vestibular function tests are contradictory to the clinical symptoms of patients. For instance, in a previous report, one patient had no typical symptoms of vertigo. Still, there was a significant decrease in the gain of video head impulse test (vHIT)<sup>[17]</sup> and unilateral weakness in the caloric test<sup>[18]</sup> at the affected semicircular canal.

(3) There is an excess of static tension in the vestibular system because of chronic stimulation of the vestibular nerve by the tumor, presenting spontaneous nystagmus (SN) towards the affected side, difficulty in standing with closed eyes, or tipping towards the healthy side<sup>[16]</sup>.

(4) The VS on the affected vestibular nerve blocks the transmission of information from the peripheral to the central vestibular system, resulting in vestibular dysfunction characterized by multi- or full-frequency lesions, suggesting the possibility of vestibular neuropathy<sup>[17, 18]</sup>. Caloric test and vHIT were used to detect low- and high-frequency vestibular lesions, respectively, and the sensitivity of the two tests to VS was 72%<sup>[19]</sup> and 80%<sup>[17]</sup> respectively, suggesting that vHIT could be used as a screening tool for VS, owing to its convenience.

## 3 VESTIBULAR FUNCTION TESTS IN VS

The peripheral vestibular lesion can be either reversible or irreversible, wherein the irreversible lesion can be compensated through vestibular rehabilitation training to alleviate and eliminate clinical symptoms. The development of vestibular examination technology has gradually realized the multiple assessments of vestibular function, including exploring the vestibular lesion and evaluating compensatory status<sup>[20]</sup>. The unilateral weakness (UW) in caloric test and the gain and asymmetry in vHIT and vestibular autorotation test (VAT) can be used to explore vestibular lesions, whereas SN, subjective visual vertical/horizontal (SVV/SVH), and sensory organization test (SOT) can assess vestibular compensatory state in many aspects. The lesion sites of different vestibular peripheral disorders can be in the vestibular or terminal nerve. The vestibular terminal lesion is frequency selectively, whereas the vestibular nerve lesion is full- or multi-frequency, and VS is one form of vestibular nerve lesion. The development and improvement of vestibular detection techniques with different frequencies provide effective support for evaluating frequency characteristics of peripheral vestibular disorders<sup>[21]</sup>. The caloric test reflects vestibular function at ultralow frequencies (0.025 Hz), whereas the vHIT and VAT evaluate high frequencies (2–6 Hz). Additionally, the SVV/SVH and SOT can reveal vestibular compensation. vHIT and vestibular evoked myogenic potentials (VEMPs) can identify the superior/inferior vestibular origin of VS and monitor tumor progression. Therefore, the modern vestibular function tests can determine the laterality, affected frequencies, and nerve of origin of the lesion, providing a basis for VS screening, diagnosis, differential diagnosis, and surgical positioning<sup>[15]</sup>.

### 3.1 Caloric Test

Vertigo is generally not the primary symptom of VS, but it is one of the clinical features of the vestibular lesion with increased VS. Caloric test is a routine itemized vestibular function test and should not be neglected in VS diagnosis<sup>[15]</sup>. The horizontal semicircular canal stimulated unilaterally by the caloric test is innervated by the superior vestibular nerve. So

that there may be a significant unilateral weakness in caloric response when VS originates from the superior part of the vestibular nerve. Borgmann *et al*<sup>[22]</sup> tested 111 patients with VS preoperatively. They defined pathologic caloric response as an indicator of the involvement of superior vestibular nerve schwannomas (SVN) and the normal finding as a sign of inferior vestibular schwannomas (IVN). Of 111 patients, 90 (81%) with pathologic results in the preoperative caloric test could predict nerve origin. This study suggested that caloric test can help to predict the origin of nerve in VS patients and could be used as an indirect predictor of hearing preservation because patients with SVN have less postoperative hearing loss than IVN. On the contrary, Ushio *et al*<sup>[23]</sup> found that there was no significant difference in abnormal caloric response between SVN and IVN patients, indicating that there was no clear correlation between caloric response and the origin of nerve in VS. Of note, the caloric test was not performed in different tumor size groups in their study; therefore, the effect of tumor volume on nerve compression could not be excluded. The frequent pathologic results of the caloric test may be due to large tumors that may compress both the superior and inferior vestibular nerves.

A study of 629 patients with VS by Tringali *et al*<sup>[18]</sup> showed that there was a good correlation between UW in caloric test and tumor size before surgery. The VS patients with UW <20% had smaller tumor size and higher postoperative hearing preservation rate, whereas postoperative facial palsy was more frequently observed in the group of VS patients with UW >70%. Their study suggested that a normal caloric response can be a good prognostic factor for postoperative hearing and facial function preservation. In a prospective study involving the assessment of 38 patients with unilateral VS, Wagner *et al*<sup>[24]</sup> compared groups with VS <20 mm and VS ≥20 mm, and found that the latter group had more severe vestibular dysfunction (median UW: 36%). Ushio *et al*<sup>[23]</sup> also found that the mean tumor size in VS patients who showed abnormal caloric responses was larger than that in those who had normal responses. In contrast, Teggi *et al*<sup>[25]</sup> suggested that it was intracanalicular length and diameter of the tumor, not total tumor volume, that influenced the vestibular function. In summary, the available data indicates that UW of the caloric test is correlated with tumor size—which can provide a basis for objective diagnosis of VS—and can be used as a predictive factor for postoperative hearing preservation<sup>[26]</sup>.

### 3.2 Video Head Impulse Test

Both caloric test and vHIT serve to evaluate the function of horizontal vestibulo-ocular reflex (VOR). The former cannot evaluate anterior and posterior semicircular canal function, whereas the latter can assess six semicircular canal functions separately.

vHIT can effectively evaluate the origin of VS through VOR and detect the severity of the affected semicircular canal and monitor the progression of VS. West *et al*<sup>[17]</sup> examined 59 patients with unilateral VS and found that the sensitivity was 80% for vHIT, and the vHIT asymmetry ratio did not correlate positively with tumor size. However, the asymmetry ratio of medium-sized tumors was significantly higher than that of small tumors, and the occurrence of saccades was related to larger tumors, while the smaller tumors have significantly fewer saccades. Bloching *et al*<sup>[27]</sup> found a relationship between low tumor grade and caloric test, but not vHIT. The available data suggested that vHIT is unsuitable for predicting tumor size accurately. However, the vHIT may be sensitive to the progression of an individual tumor so that it is possible to investigate waiting-and-scan patients in a longitudinal setting repeatedly. Conpanzo *et al*<sup>[28]</sup> preoperatively evaluated 31 sporadic VS patients with vHIT (gain of VOR, overt and covert saccades on each semicircular canal), and the results showed that vHIT could correctly identify the nerve of origin in 89.7% cases. They concluded that the pattern of semicircular canal dysfunction on vHIT had a localizing value to identify the nerve of origin in VS. The vHIT and caloric test detected the VOR function at different frequencies (for vHIT, 2–5 Hz<sup>[29]</sup>; for caloric test, 0.003 Hz<sup>[30]</sup>). The vestibular function test at different frequencies provides a deeper insight into the VOR function. According to available studies, both vHIT and caloric test should be considered complementary; mainly, vHIT could be used as a screening tool for VS given its convenience.

### 3.3 Vestibular Autorotation Test

VAT is a high-frequency and broadband VOR detection technique with a detection frequency band of 2.0–6.0 Hz, which is close to the natural activity frequency of the human body<sup>[31]</sup>. This technique offers a simple and easy method to detect the function of the horizontal and vertical semicircular canals through the subjects fixated at a stable target, whereas moving the head horizontally from side to side and vertically up to down according to auditory signals with increasing frequencies. The evaluation criteria of VAT include gain, phase, and asymmetry. The gain, defined as the ratio of eye-movement velocity to head-movement velocity, is the intensity index of VOR. The phase represents the temporal relationship between the input of head movement information and the output of eye movement information, i.e., the response speed of the vestibular system to the stimulus. Asymmetry is a symmetric index of left and right eye-movement velocity in VOR, reflecting the difference of the strength and weakness of horizontal semicircular canal function. Compared with the gain, the phase is relatively stable because it is not affected by vestibular compensation<sup>[32]</sup>. Previous studies have shown that VAT has an essential value in

diagnosing VN<sup>[33]</sup>. Therefore, the value of VAT in the diagnosis of VS deserves attention because VS is also one type of vestibular nerve lesion.

### 3.4 Vestibular Evoked Myogenic Potentials

In recent years, the role of vestibular evoked myogenic potentials (VEMPs) in the evaluation of VS patients has gained increasing attention. The cervical VEMP (cVEMP)<sup>[34]</sup> and ocular VEMP (oVEMPs)<sup>[35]</sup>, stimulated by air-conducted sound (ACS) and bone-conducted vibration (BCV), reflect the function of the saccule and utricle, respectively, and predict the nerve origin of VS. Previous studies have shown that VEMPs have a significant clinical value in the diagnosis of VS, and may even be the only sign of unilateral VS<sup>[36]</sup>. Iwasaki *et al*<sup>[37]</sup> thought that BCV oVEMP could reflect the function of the SVN, whereas some studies have also shown that VEMPs cannot be used to predict the nerve of origin in VS. In a study of 130 VS patients histologically diagnosed by surgery, there was no significant difference in VEMPs between patients with SVN and IVN<sup>[38]</sup>. Ushio *et al*<sup>[23]</sup> were also unable to find a clear correlation between VEMP results and the origin of nerve of the tumor. They suggested that large VS affects functions of both the SVN and the IVN, regardless of the origin of nerve because of the limited space of the internal acoustic canal.

Concerning tumor size, some authors described that the amplitude of ACS cVEMP was decreased or absent in up to 80% of VS patients<sup>[39]</sup>. The amplitude decreases in association with an increase in tumor size. Larger tumors and those located more medially are more commonly associated with cVEMP abnormalities<sup>[12, 40]</sup>. However, comparing the volume of tumor in the internal acoustic canal, Ushio *et al*<sup>[23]</sup> could not observe any difference between patients with abnormal and normal VEMPs responses. In summary, although there is currently no consensus regarding the use of VEMPs in detecting VS, they have played a role in VS diagnosis.

### 3.5 Evaluation of Balance Function in VS

Vestibular, visual, and somatosensory information is required to maintain balance, and the computer dynamic posturography (CDP) can provide an objective evaluation of the postural stability<sup>[41]</sup>. Postural stability in VS patients deteriorates with a gradual decrease of vestibular input due to tumor growth, even though VS patients do not experience severe vertigo. VS patients with vertigo, dizziness, and/or imbalance symptoms scored lower in CDP testing than those without symptoms, and even patients with small tumors had lower scores than normal subjects<sup>[42, 43]</sup>. The central compensation may mask the slow progression of peripheral vestibular dysfunction caused by slow-growing tumors, whereas the progressive deterioration of vestibular function eventually leads to postural instability that affects the patients' quality of life.

Ribeyre *et al*<sup>[43]</sup> found a correlation between postural stability and tumor size, postural swaying increased when the tumor was small, and this would improve with the increase of tumor size until the tumor compressing the brainstem. Nam *et al*<sup>[9]</sup> demonstrated that postural instability was prevalent in patients with VS. There were no significant differences in parameters of SOT between the acute onset and insidious onset groups, but increased tumor size and canal weakness were noted in the insidious onset group. Yin *et al*<sup>[44]</sup> examined 22 patients with small VS (<20 mm) and found that these patients may have a normal gait. Still, their vestibular deficit could be detected by proper gait analysis especially with visual deprivation. In conclusion, CDP can objectively evaluate the balance function of VS patients and dynamically monitor the status of the vestibular lesion in VS patients.

### 3.6 Subjective Visual Vertical/Horizontal Values

The subjective visual vertical (SVV) and subjective visual horizontal (SVH) values evaluate the function of the otolithic pathways, which belong to the evaluation technology of VOR, reflecting the static tension balance of the bilateral otolithic system<sup>[45]</sup>. Zhao *et al*<sup>[46]</sup> showed that SVV can not only evaluate the range of unilateral peripheral vestibular dysfunction but also assess the static compensatory state of the otolithic system. However, comparing the abnormal deviation of SVV in 47 VS patients, Cada *et al*<sup>[47]</sup> did not observe a significant difference before and after operation (on the postoperative week 3 and month 3). Similarly, Thomeer *et al*<sup>[48]</sup> evaluated the vestibular compensations of 48 VS patients at different stages after surgery and suggested that SVV was not a good prognostic factor because of its lack of sensitivity in evaluating the postoperative compensation in VS patients. These results are in agreement with those reported by Furman *et al*<sup>[49]</sup>. In conclusion, SVV/SVH is currently not a practical compensatory assessment for VS, but it can be used to assess otolithic function in VS patients at the early stage of the disease.

With the development and improvement of modern vestibular examination technology, the weight of vestibular function tests in VS diagnosis will be gradually increased.

## 4 DISCUSSION

VS are benign neoplasms arising from the Schwann cells of the vestibular nerve, with the vestibular nerve being the earliest involved. But based on its growth characteristics, hearing loss and tinnitus are the primary symptoms, followed by imbalance, vertigo and facial nerve involvement, and even vestibular symptoms are absent. The separation of internal vestibular lesions and external symptoms in some patients with VS seriously interferes with the clinician's perspective,

and even leads to missed diagnosis, delayed diagnosis and misdiagnosis. Hence, the intrinsic pathological characteristics and the corresponding changes of clinical symptoms of VS should be given adequate concern.

VS is a kind of slow-growing tumor that has significantly different clinical manifestations at different stages. VS often presents as an acute vestibular syndrome (AVS) at the active stage, similar to VN, with significant vestibular symptoms such as vertigo and balance disorders. Given the slow growth of the tumor, vestibular compensation is established over time, and the typical vertigo symptoms of UVD are absent, which can be inconsistent with the results of vestibular function tests. The diagnosis of VS is directly related to prognosis<sup>[20, 50, 51]</sup>. VS presents with the insidious progression of vestibular dysfunction and is generally asymptomatic. The vestibular function tests can reveal the intrinsic vestibular lesion and avoid misdiagnosis and missed diagnosis.

Zhou *et al*<sup>[52]</sup> reported that UVD, headache, and ataxia were positively correlated with tumor size. By estimating tumor size and the development of VS according to patients' symptoms and the results of vestibular tests, clinicians can more effectively preserve the facial nerve and hearing function during surgery. Additionally, vestibular function in VS patients can provide clinical evidence for the differential diagnosis of VS<sup>[53]</sup>. Some researchers considered that the possibility of VS should be excluded when patients have tinnitus, hearing loss, vertigo, and abnormal unilateral cochlea and vestibular function<sup>[6]</sup>. VS is screened by using audiologic tests and MRI, whereas tests of vestibular function are typically omitted. In addition to a comprehensive and detailed history, audiology and MRI, the clinical value of vestibular function evaluation should also be considered in the diagnosis of VS. Modern vestibular examination technology can reflect the severity and frequency of vestibular lesions, and compensation from multiple perspectives, timely and effectively reveal the hidden vestibular lesions and evaluate the vestibular compensation status. It provides a more comprehensive and powerful support for the objective and accurate evaluation of VS, and will play an important role in the clinical diagnosis, treatment, rehabilitation and research of VS in the future.

#### Conflict of Interest Statement

All authors declare no conflicts of interest.

#### REFERENCES

- 1 St Martin MB, Hirsch BE. Imaging of hearing loss. *Otolaryngol Clin North Am*, 2008,41(1):157-178
- 2 Lin D, Hegarty JL, Fischbein NJ, *et al*. The prevalence of "incidental" acoustic neuroma. *Arch Otolaryngol Head Neck Surg*, 2005,131(3):241-244
- 3 Morcos JJ. Vestibular schwannomas. *J Neurosurg*, 2013, 118(3):550-553
- 4 Propp JM, McCarthy BJ, Davis FG, *et al*. Descriptive epidemiology of vestibular schwannomas. *Neuro Oncol*, 2006,8(1):1-11
- 5 Pandrangi VC, Han AY, Alonso JE, *et al*. An Update on Epidemiology and Management Trends of Vestibular Schwannomas. *Otol Neurotol*, 2020,41(3):411-417
- 6 Kentala E, Pyykko I. Clinical picture of vestibular schwannoma. *Auris Nasus Larynx*, 2001,28(1):15-22
- 7 Goldbrunner R, Weller M, Regis J, *et al*. EANO guideline on the diagnosis and treatment of vestibular schwannoma. *Neuro Oncol*, 2020,22(1):31-45
- 8 Andersen JF, Nilsen KS, Vassbotn FS, *et al*. Predictors of vertigo in patients with untreated vestibular schwannoma. *Otol Neurotol*, 2015,36(4):647-652
- 9 Nam GS, Jung CM, Kim JH, *et al*. Relationship of Vertigo and Postural Instability in Patients With Vestibular Schwannoma. *Clin Exp Otorhinolaryngol*, 2018,11(2):102-108
- 10 Welling DB, Glasscock ME, Woods CI, *et al*. Acoustic neuroma: a cost-effective approach. *Otolaryngol Head Neck Surg*, 1990,103(3):364-370
- 11 Charabi S. Acoustic neuroma/vestibular schwannoma *in vivo* and *in vitro* growth models. A clinical and experimental study. *Acta Otolaryngol Suppl*, 1997,530: 1-27
- 12 Day AS, Wang CT, Chen CN, *et al*. Correlating the cochleovestibular deficits with tumor size of acoustic neuroma. *Acta Otolaryngol*, 2008,128(7):756-760
- 13 Gouveris H, Akkafa S, Lippold R, *et al*. Influence of nerve of origin and tumor size of vestibular schwannoma on dynamic posturography findings. *Acta Otolaryngol*, 2006,126(12):1281-1285
- 14 Hoffmann CP, Seigle B, Frere J, *et al*. Dynamical analysis of balance in vestibular schwannoma patients. *Gait Posture*, 2017,54:236-241
- 15 von Kirschbaum C, Gurkov R. Audiovestibular Function Deficits in Vestibular Schwannoma. *Biomed Res Int*, 2016,2016:4980562
- 16 Stipkovits EM, Van Dijk JE, Graamans K. Electronystagmographic changes in patients with unilateral vestibular schwannomas in relation to tumor progression and central compensation. *Eur Arch Otorhinolaryngol*, 1999,256(4):173-176
- 17 West N, Sass H, Klokker M, *et al*. Video Head Impulse Test Results in Patients With a Vestibular Schwannoma-Sensitivity and Correlation With Other Vestibular System Function Tests, Hearing Acuity, and Tumor Size. *Otol Neurotol*, 2020,41(5):e623-e629
- 18 Tringali S, Charpiot A, Ould MB, *et al*. Characteristics of 629 vestibular schwannomas according to preoperative caloric responses. *Otol Neurotol*, 2010,31(3):467-472.
- 19 Blodow A, Helbig R, Wichmann N, *et al*. The video head impulse test: first clinical experiences. *HNO*, 2013,61(4):327-334.
- 20 Hebb ALO, Erjavec N, Morris DP, *et al*. Quality of life related to symptomatic outcomes in patients with vestibular schwannomas: A Canadian Centre perspective. *Am J Otolaryngol*, 2019,40(2):236-246
- 21 Kim HJ, Park SH, Kim JS, *et al*. Bilaterally Abnormal Head Impulse Tests Indicate a Large Cerebellopontine Angle Tumor. *J Clin Neurol*, 2016,12(1):65-74

- 22 Borgmann H, Lenarz T, Lenarz M. Preoperative prediction of vestibular schwannoma's nerve of origin with posturography and electronystagmography. *Acta Otolaryngol*, 2011,131(5):498-503
- 23 Ushio M, Iwasaki S, Chihara Y, *et al.* Is the nerve origin of the vestibular schwannoma correlated with vestibular evoked myogenic potential, caloric test, and auditory brainstem response? *Acta Otolaryngol*, 2009,129(10):1095-1100
- 24 Wagner JN, Glaser M, Wowra B, *et al.* Vestibular function and quality of life in vestibular schwannoma: does size matter? *Front Neurol*, 2011,2:55
- 25 Teggi R, Franzin A, Spatola G, *et al.* Vestibular assessment in patients with vestibular schwannomas: what really matters? *Acta Otorhinolaryngologica Italica Organo Ufficiale Della Società Italiana Di Otorinolaringologia E Chirurgia Cervico Facciale*, 2014, 34(2):123-128
- 26 Stidham KR, Roberson JB. Hearing improvement after middle fossa resection of vestibular schwannoma. *Otology & Neurotology*, 2001,22(6):917-921
- 27 Bloching, Boris M, Bloedow, *et al.* Horizontal VOR function shows frequency dynamics in vestibular schwannoma. *European archives of oto-rhino-laryngology: Official journal of the European Federation of Oto-Rhino-Laryngological Societies (EUFOS)*, 2015
- 28 Constanzo F, Sens P, Teixeira BCA, *et al.* Video Head Impulse Test to Preoperatively Identify the Nerve of Origin of Vestibular Schwannomas. *Oper Neurosurg (Hagerstown)*, 2019,16(3): 319-325
- 29 Jorns-Haderli M, Straumann D, Palla A. Accuracy of the bedside head impulse test in detecting vestibular hypofunction. *J Neurol Neurosurg Psychiatry*, 2007, 78(10):1113-1118
- 30 Halmagyi GM, Curthoys IS, Cremer PD, *et al.* The human horizontal vestibulo-ocular reflex in response to high-acceleration stimulation before and after unilateral vestibular neurectomy. *Exp Brain Res*, 1990,81(3):479-490
- 31 O'Leary DP, Davis LL. High-frequency autorotational testing of the vestibuloocular reflex. *Neurologic Clinics*, 1990,8(2):297-312
- 32 Hirvonen TP, Pyykkö I, Aalto H, *et al.* Vestibulo-ocular reflex function as measured with the head autorotation test. *Acta Otolaryngol*, 1997,117(5):657-662
- 33 Yan T, Zong F, Han X, *et al.* Vestibular Neuritis in Patients Among Different Age Groups: Clinical Features and Outcomes. *J Am Acad Audiol*, 2020,31(9):629-635
- 34 Rosengren SM, Welgampola MS, Colebatch JG. Vestibular evoked myogenic potentials: past, present and future. *Clin Neurophysiol*, 2010,121(5):636-651
- 35 Kantner C, Gurkov R. Characteristics and clinical applications of ocular vestibular evoked myogenic potentials. *Hear Res*, 2012,294(1-2):55-63
- 36 Adamec I, Skoric MK, Handzic J, *et al.* The role of cervical and ocular vestibular-evoked myogenic potentials in the follow-up of vestibular neuritis. *Clin EEG Neurosci*, 2014,45(2):129-136
- 37 Iwasaki S, Murofushi T, Chihara Y, *et al.* Ocular vestibular evoked myogenic potentials to bone-conducted vibration in vestibular schwannomas. *Otol Neurotol*, 2010,31(1):147-152
- 38 Church S, Murofushi T, Chihara Y, *et al.* Endocarditis caused by *Pasteurella caballi* in a horse. *Aust Vet J*, 1998,76(8):528-530
- 39 Ushio M, Matsuzaki M, Takegoshi H, *et al.* Click-and short tone burst-evoked myogenic potentials in cerebellopontine angle tumors. *Acta Otolaryngol Suppl*, 2001,545:133-135
- 40 Hu YF, Cheng PW, Young YH. Comparison of vestibular function between large cerebellopontine angle meningioma and schwannoma. *Acta Otolaryngol*, 2009,129(2):161-165
- 41 Gouveris H, Helling K, Victor A, *et al.* Comparison of electronystagmography results with dynamic posturography findings in patients with vestibular schwannoma. *Acta Otolaryngol*, 2007,127(8):839-842
- 42 Gouveris H, Stripf T, Victor A, *et al.* Dynamic posturography findings predict balance status in vestibular schwannoma patients. *Otology & Neurotology*, 2007,28(3):372-375
- 43 Ribeyre L, Frere J, Gauchard G, *et al.* Preoperative balance control compensation in patients with a vestibular schwannoma: does tumor size matter? *Clin Neurophysiol*, 2015,126(4):787-793
- 44 Yin M, Ishikawa K, Omi E, *et al.* Small vestibular schwannomas can cause gait instability. *Gait Posture*, 2011,34(1):25-28
- 45 Pagarkar W, Bamiou DE, Ridout D, *et al.* Subjective visual vertical and horizontal: effect of the preset angle. *Arch Otolaryngol Head Neck Surg*, 2008,134(4):394-401
- 46 Zhao Y, Chen T, Wang W, *et al.* The application of subjective visual gravity in assessment of vestibular compensation: a pilot study. *Zhonghua Er Bi Yan Hou Tou Jing Wai Ke Za Zhi*, 2016, 51(5):355-360
- 47 Cada Z, Balatkova Z, Cakrt O, *et al.* Predictors of central vestibular compensation after surgery for vestibular schwannomas. *Acta Otorhinolaryngol Ital*, 2019,39(1):46-52
- 48 Thomeer H, Bonnard D, Franco-Vidal V, *et al.* Prognostic Factors of Balance Quality After Transpetrosal Vestibular Schwannoma Microsurgery: An Instrumentally and DHI-based Prospective Cohort Study of 48 Patients. *Otol Neurotol*, 2015,36(5):886-891
- 49 Furman JM, Balaban CD, Pollack IF. Vestibular compensation in a patient with a cerebellar infarction. *Neurology*, 1997,48(4):916-920
- 50 Breivik CN, Nilsen RM, Myrseth E, *et al.* Working disability in Norwegian patients with vestibular schwannoma: vertigo predicts future dependence. *World Neurosurg*, 2013,80(6):e301-e305
- 51 Dayal M, Perez-Andujar A, Chuang C, *et al.* Management of vestibular schwannoma: focus on vertigo. *CNS Oncol*, 2013,2(1):99-104
- 52 Zhou Y, Zhao W, Tian L, *et al.* The prediction of the tumor size of a vestibular schwannoma by clinical performance and vestibular function tests. *J Neurooncol*, 2018,140(3):679-686
- 53 Halliday J, Rutherford SA, McCabe MG, *et al.* An update on the diagnosis and treatment of vestibular schwannoma. *Expert Rev Neurother*, 2018,18(1):29-39