Acta Oto-Laryngologica, 2013; 133: 916–923

informa
healthcare

ORIGINAL ARTICLE

Clinical significance of pathological eye movements in diagnosing posterior fossa stroke

CHIA-HUNG SU[1] & YI-HO YOUNG[2]

[1]*Department of Otolaryngology, Catholic Cardinal Tien Hospital, Fu-Jen Catholic University, Taipei and* [2]*Department of Otolaryngology, National Taiwan University Hospital, Taipei, Taiwan*

Abstract

Conclusion: Close observation of pathological eye movements such as disconjugate eye movements, multi-directional gaze nystagmus, and persistent unilateral gaze nystagmus may facilitate the effort of clinicians to arrange magnetic resonance imaging (MRI) study, because physical examinations may overlook the posterior fossa lesions. *Objective:* This paper reviews our experience of patients with posterior fossa stroke via observation of pathological eye movements over the past 10 years. *Methods:* Seventy patients with posterior fossa stroke manifested as acute vertiginous attack were admitted. All patients underwent examination of eye movements, MRI, and a battery of audiovestibular function tests. *Results:* Of the 70 patients, 22 (31%) demonstrated pathological eye movements including persistent (>24 h) unilateral gaze nystagmus in 12 patients, and multi-directional gaze nystagmus in 10 patients. Conjugate eyes movements were identified in 18 patients, and disconjugate eye movements were shown in 4 patients including medial longitudinal fasciculus syndrome in 1, paramedian pontine reticular formation syndrome in 1, and one and a half syndrome in 2. The vestibular test battery revealed abnormal responses for >85% of the patients in each test. MRI demonstrated infarction or hemorrhage involving the brainstem in 12 patients, cerebellum in 8 patients, and both in 2 patients.

Keywords: *Conjugate eye movement, disconjugate, medial longitudinal fasciculus, paramedian pontine reticular formation, one-and-a-half syndrome*

Introduction

Physiological eye movements comprise conjugate and disconjugate types. The conjugate eye movements consist of pursuit, saccade, vestibulo-ocular reflex (VOR), and optokinetic eye movement, whereas disconjugate eye movements indicate vergence. When the vestibular system is insulted, vestibular nuclei transfer the neuronal impulses via the medial longitudinal fasciculus (MLF) and paramedian pontine reticular formation (PPRF) to the bilateral nuclei of the cranial nerves (CNs) III, IV, and VI, causing pathological eye movements [1]. For example, spontaneous nystagmus beating toward the opposite (healthy) side is noted when one labyrinth is damaged, since asymmetric inputs from bilateral labyrinths cause asymmetric firings of bilateral vestibular nuclei [2]. When the lesion encroaches on the posterior cranial fossa, i.e. when there is cerebellar hemorrhage, uni- or multi-directional gaze nystagmus may be shown, mainly due to failure of central compensation at the acute episode [3]. Thus, disinhibition of the cerebellum on the vestibular nuclei may increase the VOR gain [4,5]. Clinically, patients with posterior fossa stroke manifesting as acute vertiginous attack may display various pathological eye movements, providing a clue for the topographic diagnosis of the posterior fossa lesion [6]. This paper thus reviews our experience of patients with posterior fossa stroke via observation of pathological eye movements over the past 10 years.

Correspondence: Yi-Ho Young, Department of Otolaryngology, National Taiwan University Hospital, 1 Chang-te St, Taipei, Taiwan.
Tel: +886 2 23123456 ext. 65221. Fax: +886 2 2394 6674. E-mail: youngyh@ntu.edu.tw

(Received 30 January 2013; accepted 4 March 2013)

ISSN 0001-6489 print/ISSN 1651-2251 online © 2013 Informa Healthcare
DOI: 10.3109/00016489.2013.783716

Table I. Clinical information for 22 patients with posterior fossa stroke.

Case no.	Sex	Age (years)	Side	Site	Diagnosis
1	M	61	Bilateral	BS	BS infarction
2	M	81	Left	CL	CL hemorrhage
3	M	56	Left	CL	CL hemorrhage
4	M	59	Left	CL	CL hemorrhage
5	M	76	Right	BS	BS infarction
6	M	28	Left	BS + CL	PICA syndrome
7	M	78	Bilateral	CL	CL infarction
8	F	64	Left	BS	AICA syndrome
9	F	44	Left	BS	BS infarction
10	M	69	Right	CL	CL infarction
11	F	60	Bilateral	BS	BS infarction
12	F	23	Bilateral	BS	BS infarction
13	F	54	Bilateral	BS	BS infarction
14	F	36	Left	BS	PICA syndrome
15	F	80	Right	BS	PICA syndrome
16	M	36	Right	CL	CL infarction
17	M	66	Left	CL	CL infarction
18	F	68	Left	CL	CL hemorrhage
19	M	70	Right	BS	MLF syndrome
20	M	51	Right	BS + CL	PPRF syndrome
21	F	50	Left	BS	OAAH syndrome
22	F	85	Left	BS	OAAH syndrome

AICA, anterior inferior cerebellar artery; BS, brainstem; CL, cerebellum; MLF, medial longitudinal fasciculus; OAAH, one and a half; PICA, posterior inferior cerebellar artery; PPRF, paramedian pontine reticular formation.

Material and methods

Patients

From January 2001 to December 2010, 17122 patients with vertigo/dizziness were consecutively encountered at our vertigo clinic. Of them, 70 patients with acute vertiginous attack were admitted and diagnosed as having stroke at the posterior cranial fossa by magnetic resonance imaging (MRI). Twenty-two patients (31%) showing pathological eye movements were enrolled in this study (Table I). Twelve were men and 10 were women, with ages ranging from 23 to 85 years (mean 59 years). Fifteen patients (68%) had systemic diseases such as hypertension, diabetes mellitus, or coronary artery disease.

Initially, the eye movements were observed via videonystagmography. Once pathological eye movements were detected, MRI was subsequently performed by Magneton plus 1.51 (Siemens, Erlangen, Germany) on a superconducting 1.5 Tesla MR system. When the patients were cooperative, a battery of audiovestibular function tests was conducted including audiometry, caloric test with electronystagmographic (ENG) examination, cervical vestibular-evoked myogenic potential (cVEMP) tests, and ocular VEMP (oVEMP) tests. Exclusion criteria consisted of conscious disturbance, head injury, previous ear or brain surgery, and uncooperative patients.

This study was approved by the institutional review board, and each subject signed the informed consent to participate.

ENG examination

ENG examination (OK-5 type, Nagashima, Tokyo, Japan) consisted of recording the gaze nystagmus first, followed by positional, positioning, pursuit, saccade, optokinetic nystagmus (OKN), and bithermal caloric tests. The mean slow phase velocity (SPV) of caloric nystagmus in normal controls at our laboratory was $31 \pm 7^\circ/s$ (mean ± SD), and we defined those above $45^\circ/s$ as hyperactive response. In contrast, canal paresis was defined when the SPV was $<17^\circ/s$, or a greater than 25% difference between maximum SPV measurements for each ear, when compared with the sum of SPVs from each ear. If the caloric test failed

to elicit a response, the subject underwent ice-water (0°C, 10 ml) caloric testing to further confirm the caloric areflexia.

The visual suppression test was performed during caloric testing except in those without caloric responses. When caloric nystagmus reached maximum responses, the light was turned on and the subject's eyes were fixed on a target ahead for 10 s. Thereafter, the light was turned off and recording was continued until the end of caloric nystagmus. The visual suppression index means the difference of SPV between the dark and light conditions divided by SPV in darkness. At our laboratory, the norm for the visual suppression index is 72 ± 6%, and loss of visual suppression is defined when the visual suppression index is <60% [7].

oVEMP test

The subject was in a sitting position. Two active electrodes were placed around 1 cm below the center of the two lower eyelids. The other two reference electrodes were positioned about 1–2 cm below the active ones, and one ground electrode was placed on the sternum. During recording (Smart EP 3.90, Intelligent Hearing Systems, Miami, FL, USA), the subject was instructed to look upward at a small fixed target >2 m from the eyes. The electromyographic (EMG) signals were amplified and bandpass filtered between 1 and 1000 Hz. The stimulation rate was 5/s. The duration of analysis of each response was 50 ms, and 30 responses were averaged for each run.

The operator held the vibrator by hand and supported most of its weight such that the axis of the connected bakelite cap perpendicularly delivered a repeatable tap with little pressure on the subject's skull at the Fz site (the midline forehead at the hairline). If oVEMPs were not elicited, alternatively, tapping at the ipsilateral mastoid site (2 cm behind the opening of the external ear canal) was subsequently performed. The input signal was a half cycle 500 Hz sine wave, with the initial peak driving voltage about 8V, equivalent to 128 dB force level.

The initial negative-positive biphasic waveform comprised peaks nI and pI. Consecutive runs were performed to confirm the reproducibility of peaks nI and pI, and oVEMPs were deemed to be present. At our laboratory, the norm for the latency of peak nI was 11.4 ± 0.8 ms. Those with an nI latency exceeding 13.0 ms were defined as having a delayed response. The norm for the asymmetry ratio was 0.12 ± 0.14, and those with an asymmetry ratio >40% were interpreted as abnormal.

cVEMP test

Each subject was in a supine position. Two active electrodes were placed on the upper half of the sternocleidomastoid (SCM) muscles, one reference electrode was positioned on the suprasternal notch, and a ground electrode was situated on the forehead. The other settings were the same as in the oVEMP test, except that the vibrator delivered a repeatable tap on the subject's head at inion. To measure background muscle activity, subjects were given feedback of the level of EMG activity in their SCM muscles during data collection and were required to keep a background muscle activity of at least >50 μV. The subjects elevated their heads during testing. A total of 50 responses were averaged and recorded bilaterally.

The first positive and second negative polarities of biphasic waveform were termed waves p13 and n23, respectively. At our laboratory, the norm for the latency of p13 was 14.4 ± 1.3 ms, and those with asymmetry ratio >33% were defined as abnormal [8].

Results

Clinical manifestations consisted of rotatory vertigo and ataxia lasting >24 h in all 22 patients (100%), followed by the symptoms of nausea/vomiting (64%), diplopia (45%), headache (36%), hearing loss (36%), and tinnitus (27%). Ten patients (45%) exhibited neurological signs such as CNs VI, VII, IX, X paresis, dysarthria, or lack of coordination.

Observation of eye movements

All 22 patients displayed pathological eye movements including unilateral gaze nystagmus in 11 patients, upbeat nystagmus in 1 patient, bilateral gaze nystagmus in 8 patients, and three-directional gaze nystagmus in 2 patients (Table II). All nystagmus lasted more than 24 h. Eighteen patients exhibited conjugate eye movements, while four patients exhibited disconjugate eye movements (case nos 19–22). For the latter group of four patients, MLF syndrome was noted in one, PPRF syndrome in one, and one and a half (OAAH) syndrome in two (Table I).

Figure 1 shows a 70-year-old male with right MLF syndrome (case no. 19). Examination of eye movements revealed that the rightward, upward, forward, and downward gazes were intact, but the leftward gaze had limited adduction on the right eye, accompanied by monocular nystagmus on the abducting (left) eye beating to the left side, a condition termed internuclear ophthalmoplegia (INO). The convergence function was preserved. MRI demonstrated multiple hemorrhagic foci on the

Table II. Vestibular test battery in patients with posterior fossa stroke.

Case no.	Nystagmus	ET	OKN	Caloric (R/L)	VS (R/L)
1	Unilateral	abn	abn	n/n	n/aug
2	Unilateral	abn	n	cp/–	cnt
3	Unilateral	abn	abn	cp/–	aug/cnt
4	Unilateral	abn	abn	n/cp	aug/–
5	Unilateral	abn	abn	–/n	cnt/–
6	Bilateral	abn	abn	n/–	–/cnt
7	Unilateral	abn	abn	cp/cp	–/aug
8	Unilateral	abn	n	n/cp	–/–
9	3-direction	abn	abn	n/cp	n/aug
10	Unilateral	abn	abn	n/n	–/–
11	Bilateral	abn	abn	n/cp	–/cnt
12	3-direction	abn	abn	n/n	n/–
13	Unilateral	n	n	n/cp	–/–
14	Bilateral	abn	abn	cp/cp	n/aug
15	Bilateral	abn	abn	–/n	cnt/–
16	Upbeat	abn	abn	–/n	cnt
17	Bilateral	abn	abn	cp/–	–/cnt
18	Unilateral	abn	abn	hyper/hyper	–/–
19	Bilateral	abn	abn	–/–	cnt
20	Bilateral	abn	abn	n/–	cnt
21	Bilateral	abn	abn	–/–	cnt
22	Unilateral	abn	abn	n/cp	cnt
Abnormal rates		95%	86%	86%	100% (16/16)

–, absent; abn, abnormal; aug, augmented; cnt, cannot test; cp, canal paresis; ET, eye tracking; hyper, hyperfunction; n, normal; OKN, optokinetic nystagmus; VS, visual suppression.

right pontomedullary junction (Figure 2), which is compatible with the delayed responses in the cVEMP test in this patient.

Figure 3 shows an 85-year-old female with left OAAH syndrome (case no. 22). Examination of eye movements revealed intact upward, forward, and downward gazes. However, gaze palsy was noted when gaze leftward (one), associated with INO (a half) when gaze rightward. MRI demonstrated multiple foci of recent infarct on the left pons (Figure 4). Both oVEMP and cVEMP tests showed absent responses in this patient. She was then transferred to the neurological ward where conservative treatment without surgical intervention was given. Three months later, she was free from vertiginous episodes, and the pathological eye movements were resolved and had returned to normal eye movements.

Audiovestibular function testing

Nineteen patients underwent audiometry and 13 patients (68%) showed sensorineural hearing loss, probably due to aging, since 11 of these 13 patients were aged >60 years old.

ENG examination showed abnormal eye tracking test in 21 patients (95%), abnormal OKN test in 19 patients (86%), and abnormal caloric test in 19 patients (86%) including hyperfunction in 1 patient, canal paresis in 8 patients, and caloric areflexia in 10 patients. Visual suppression test during caloric testing was performed in 16 patients, and all (100%) showed abnormal results including loss of visual suppression in 12 patients, and augmentation of caloric nystagmus in light in 6 patients. Two patients (case nos 4 and 7) displayed loss of visual suppression and augmentation of caloric nystagmus on each ear (Table II).

Seven patients underwent cVEMP testing and all showed abnormal responses including absent cVEMPs in three patients (case nos 11, 12, and 22) and delayed cVEMPs in four patients (case nos 9, 13, 14, and 19). Three patients underwent oVEMP testing and absent oVEMPs were noted in all cases (nos 17, 18, and 22). Thus, each test of the vestibular test battery had more than 85% abnormalities.

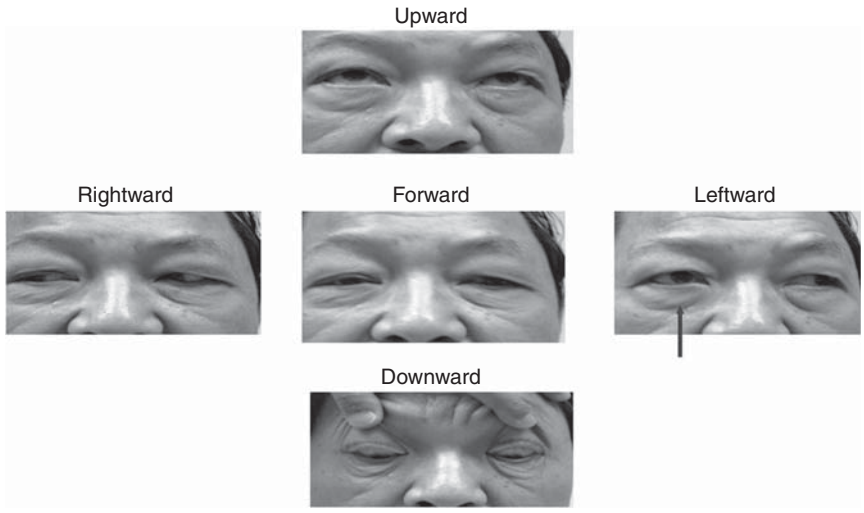

Figure 1. A 70-year-old male with medial longitudinal fasciculus (MLF) syndrome, right (case no. 19). The rightward, upward, forward, and downward gazes are intact, but the leftward gaze has limited adduction on the right eye (arrow), accompanied by monocular nystagmus on the abducting (left) eye beating to the left side. The convergence function is preserved.

MRI studies

MRI demonstrated infarction or hemorrhage involving the brainstem in 12 patients, cerebellum in 8 patients, and both in 2 patients. The right side was affected in 6 patients, the left side in 11 patients, and both sides in 5 patients. Table I summarizes the diagnoses from the imaging study.

Treatment and outcome

Four patients underwent surgical intervention and the remaining 18 patients received conservative treatments. Eleven (50%) of the 22 patients in this series were alive after 2 years, while 7 had passed away and 4 patients were lost to follow-up.

Discussion

The posterior cranial fossa, which receives vascular supply from the vertebrobasilar artery system, houses numerous critical structures (brainstem and cerebellum) involved in the generation, control or regulation of eye movements [6]. Slow blood flow or disturbed circulation in the territory of the vertebrobasilar artery may occur before infarction [9]. Since the vertebrobasilar artery system also supplies the vestibular nuclei and labyrinths, an acute vertiginous episode may occur before the overt derangement of posterior fossa stroke. Thus, stroke at the brainstem and cerebellum may give rise to a broad spectrum of pathological eye movements, providing a clue to delineate the lesion in the posterior fossa.

We first consider midbrain-pontine stroke. The midbrain houses the vertical gaze centers at the level of the mesodiencephalic junction, whereas horizontal gaze generation resides in the lower pontine tegmentum. Consequently, conjugate or disconjugate eye movements are common owing to infarcts or hemorrhages involving mainly, but not exclusively, the medial and lateral pontine tegmentum [1].

The most common disconjugate eye movement is INO, which means limitation or slowing of adduction ipsilateral to the MLF lesion, often accompanied by dissociated contralateral horizontal nystagmus in the abducting eye and preserved convergence, as shown in Figure 1 (case no. 19). Most infarcts causing INO occur at the pontomesencephalic junction or rostral

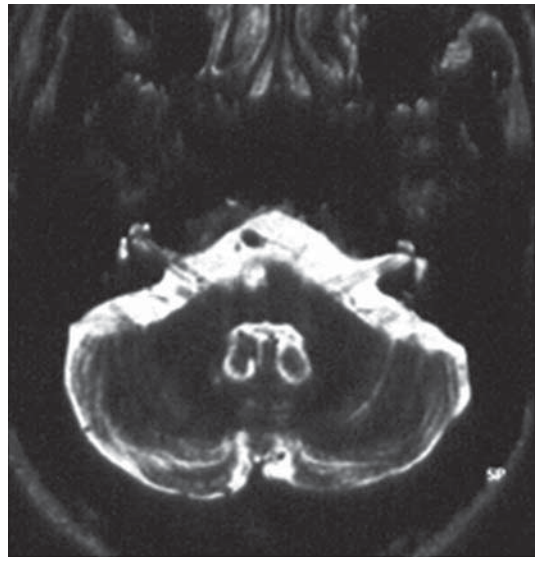

Figure 2. Same patient as in Figure 1. MRI demonstrates multiple hemorrhagic foci on the right pontomedullary junction.

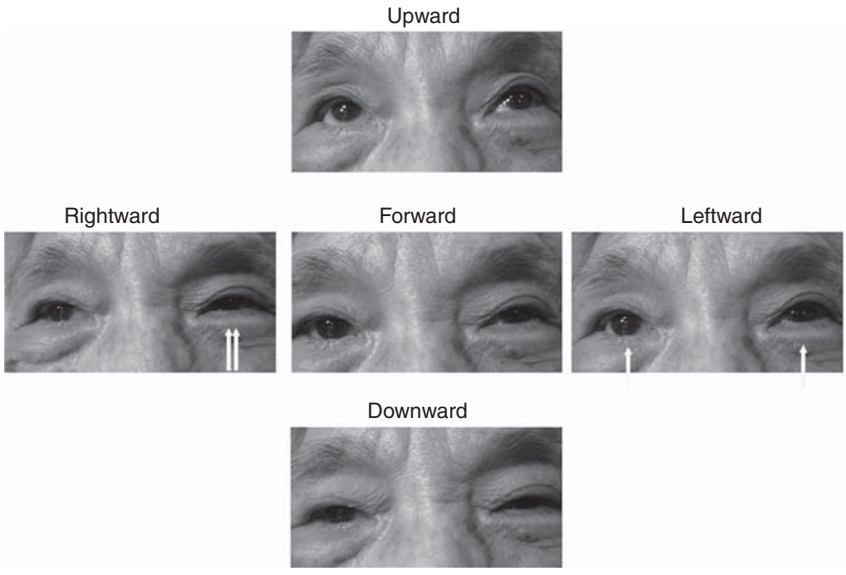

Figure 3. An 85-year-old female with one and a half syndrome (OAAH), left (case no. 22). The upward, forward, and downward gazes are intact. However, gaze palsy is noted when gaze leftward (arrows) associated with internuclear ophthalmoplegia (double arrows) when gaze rightward.

pons level (Figure 2), both of which affect the MLF [10,11].

The PPRF, located immediately ventral to the CN VI nuclei, is the prenuclear generator of ipsilateral horizontal saccade [12]. Lesions located in the PPRF cause ipsilateral horizontal gaze palsy. Thus, all conjugate eye movements to the ipsi-lesional side are impaired, whereas those to the contra-lesional (healthy) side are intact [13].

The pathological eye movements of OAAH syndrome [14] are characterized by an ipsilesional horizontal gaze palsy (one) and an INO (a half), with abduction of the contralesional eye being the only preserved movement (Figure 3). Horizontal OAAH syndrome is less common than INO in pontine ischemia, indicating pontine tegmentum damage (Figure 4).

We next consider medullary stroke. Lateral medullary (posterior inferior cerebellar artery (PICA), Wallenberg's) syndrome caused by ischemia in the PICA territory has been known for over a century. Facial paresis, dysphagia, and dysarthria are significantly more common in patients with rostrally located lesions than in those with caudally located lesions. Conversely, severe headache and gait ataxia are more common in the caudal group than in the rostral group [15–17].

Either horizontal or torsional nystagmus is common in patients with PICA syndrome, with the direction beating away from the lesion side, which may result from the otolith and semicircular canal imbalance. Of the vestibular test battery, a possible link exists between caloric areflexia and medullary infarction located rostrally, while absent or delayed cVEMPs are linked to medullary infarction located caudally [18].

Cerebellar stroke mostly originates from the superior cerebellar artery (SCA) and PICA, with the frequencies of cerebellar stroke almost equally distributed [19]. Gaze-evoked nystagmus is a common finding in cerebellar infarct, and appears in pathological

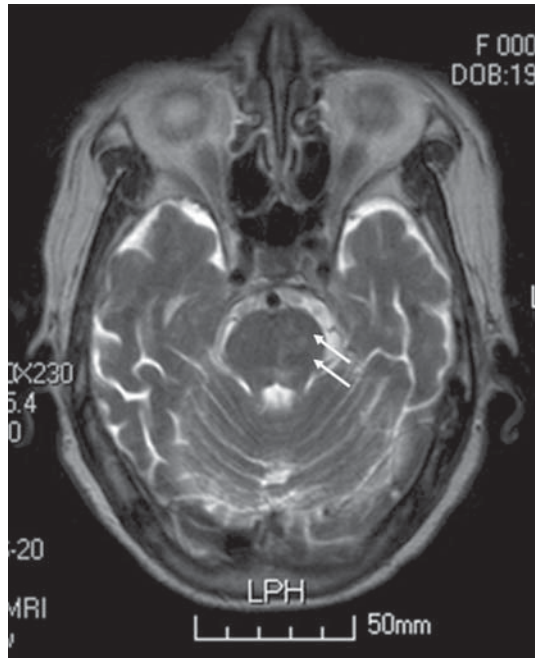

Figure 4. Same patient as in Figure 3. MRI demonstrates multiple foci of recent infarct on the left pons.

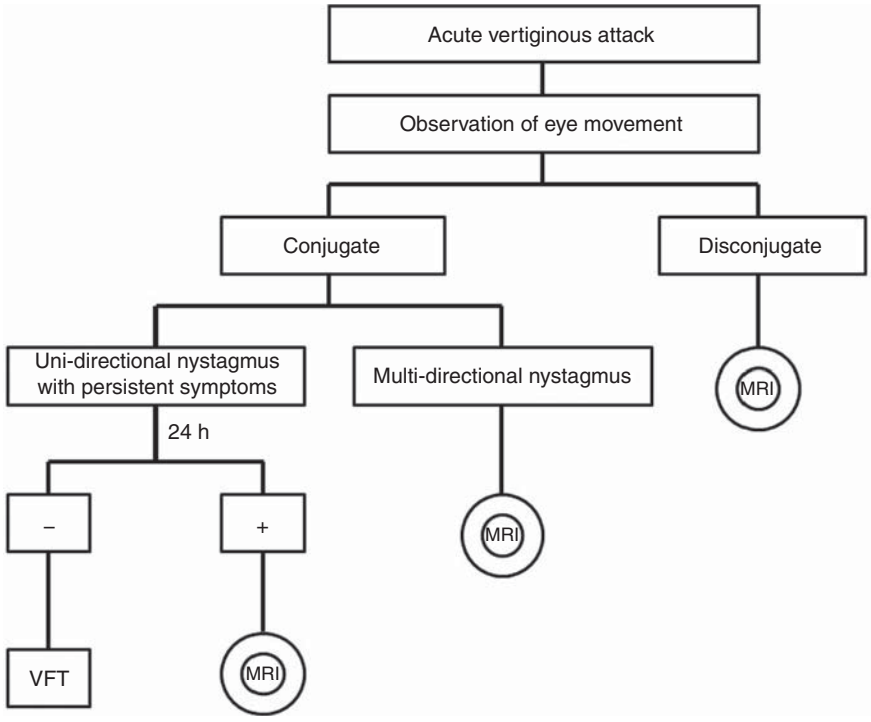

Figure 5. The working protocol for diagnosing posterior fossa stroke in a vertiginous patient. VFT, vestibular function testing.

conditions when an attempt is made to hold the eye in an eccentric position. As the eye slowly drifts towards the primary position, a corrective saccade brings the eye back on target [8]. This implies that an important function of the flocculus is to improve the fidelity of the brainstem neural integrator that normally holds the gaze position. Bilateral gaze nystagmus is related to disturbance of the brainstem neural integrator, which comprises the medial vestibular nucleus, nucleus prepositus hypoglossi, and flocculo-nodular lobe of the cerebellum [2,4,5].

In addition to imaging studies, a battery of vestibular testing helps delineate the cerebellum and/or brainstem lesions. Applying visual fixation during caloric testing can suppress caloric nystagmus by affecting the floccular Purkinje cells of the cerebellum, in turn inhibiting the superior and medial vestibular nuclei. When the visual suppression of caloric nystagmus toward the lesion side is reduced or abolished, it indicates a unilateral flocculus lesion, i.e. cerebellar stroke [20]. In contrast, loss of visual suppression associated with augmentation of the caloric nystagmus in light indicates a brainstem lesion [21]. However, distinguishing cerebellum and brainstem lesions through the visual suppression test can be challenging, since both regions are closely related anatomically.

Alternatively, the recently emerging oVEMP test, like the caloric test, has been used for assessing a crossed VOR [22]. Unlike the caloric test, oVEMPs are very short responses (10 ms). Since suppression of linear VOR appears at 102 ms, the oVEMP test thus can differentiate between cerebellar and brainstem lesions [8]. Abnormal oVEMPs in patients with cerebellar disorder (case nos 17 and 18) may indicate adjacent brainstem involvement, even when MRI fails to delineate brainstem lesion (Table I).

As regards clinical relevance, clinicians must be aware that patients with acute vertiginous attack may be suffering a potentially life-threatening posterior fossa stroke. Observation of pathological eye movements may facilitate the efforts of clinicians to arrange MRI study, because physical examinations, which sometimes cannot be performed in acute vertiginous patients, may overlook the posterior fossa lesions. Figure 5 summarizes the working protocol for diagnosing posterior fossa stroke in a vertiginous patient. If disconjugate eye movement, multi-directional gaze nystagmus, or persistent (>24 h) uni-directional gaze nystagmus associated with symptoms, i.e. vertigo and ataxia, are noted in an acute vertiginous patient, MRI should be performed immediately to achieve timely recognition of the involvement of posterior fossa stroke. For those without pathological eye movements, a battery of vestibular function tests may serve as a supplementary aid to MRI to delineate the lesion site.

Conclusion

Close observation of pathological eye movements such as disconjugate eye movements, multi-

directional gaze nystagmus, and persistent unilateral gaze nystagmus may facilitate the effort of clinicians to arrange MRI study in an acute vertiginous patient, because physical examinations sometimes overlook the posterior fossa lesions. For those without pathological eye movements, a vestibular test battery can serve as a supplementary aid to MRI to delineate the involvement of the lesion.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

[1] Moncayo J, Bogousslavsky J. Eye movement abnormalities. Front Neurol Neurosci 2012;30:131–6.

[2] Vibert N, de Waele C, Escudero M, Vidal PP. The horizontal vestibulo-ocular reflex in the hemilabyrinthectomized guinea-pig. Exp Brain Res 1993;97:263–73.

[3] Zee DS, Yamazaki A, Butler PH, Gücer G. Effects of ablation of flocculus and paraflocculus on eye movements in primate. J Neurophysiol 1981;46:878–99.

[4] Baloh RW, Jenkins HA, Honrubia V, Yee RD, Lau CG. Visual-vestibular interaction and cerebellar atrophy. Neurology 1979;29:116–19.

[5] Sharpe JA, Goldberg HJ, Lo AW, Herishanu YO. Visual-vestibular interaction in multiple sclerosis. Neurology 1981; 31:427–43.

[6] Moncayo J, Bogousslavsky J. Oculomotor disorders in vertebrobasilar stroke. Expert Rev Ophthalmol 2009;4:259–81.

[7] Su CH, Young YH. Differentiating between cerebellar and brainstem lesions with ocular vestibular-evoked myogenic potential test. Eur Arch Otorhinolaryngol 2011;268:923–30.

[8] Su CH, Young YH. Disorders affecting the fourth ventricle: etiology and clinical correlates. Otol Neurotol 2011;32: 1329–35.

[9] Welsh LW, Welsh JJ, Lewin B. Basilar artery and vertigo. Ann Otol Rhinol Laryngol 2000;109:615–22.

[10] Bolaños I, Lozano D, Cantú C. Internuclear ophthalmoplegia: causes and long-term follow-up in 65 patients. Acta Neurol Scand 2004;110:161–5.

[11] Kim JS. Internuclear ophthalmoplegia as an isolated or predominant symptom of brainstem infarction. Neurology 2004;62:1491–6.

[12] Büttner U, Büttner-Ennever JA. Present concepts of oculomotor organization. Prog Brain Res 2006;151:1–42.

[13] Bronstein AM, Morris J, Du Boulay G, Gresty MA, Rudge P. Abnormalities of horizontal gaze. Clinical, oculographic and magnetic resonance imaging findings. I. Abducens palsy. J Neurol Neurosurg Psychiatry 1990;53:194–9.

[14] Pierrot-Deseilligny C, Chain F, Serdaru M, Gray F, Lhermitte F. The 'one-and-a-half' syndrome. Electro-oculographic analyses of five cases with deductions about the physiological mechanisms of lateral gaze. Brain 1981;104: 665–9.

[15] Dieterich M, Brandt T. Wallenberg's syndrome: lateropulsion, cyclorotation, and subjective visual vertical in thirty-six. patients. Ann Neurol 1992;31:399–408.

[16] Kim JS. Pure lateral medullary infarction: clinical-radiological correlation of 130 acute, consecutive patients. Brain 2003;126:1864–72.

[17] Vuilleumier P, Bogousslavsky J, Regli F. Infarction of the lower brainstem. Clinical, aetiological and MRI-topographical correlations. Brain 1995;118:1013–25.

[18] Tseng CL, Young YH. Topographical correlations of lateral medullary infarction with caloric and vestibular evoked myogenic potential results. Eur Arch Otorhinolaryngol 2010;267: 191–5.

[19] Macdonell RA, Kalnins RM, Donnan GA. Cerebellar infarction: natural history, prognosis, and pathology. Stroke 1987; 18:849–55.

[20] Takemori S, Cohen B. Loss of visual suppression of vestibular nystagmus after flocculus lesions. Brain Res 1974;72: 213–24.

[21] Takemori S, Aiba T, Shizawa R. Visual suppression of caloric nystagmus in brain-stem lesions. Ann N Y Acad Sci 1981; 374:846–54.

[22] Huang CH, Wang SJ, Young YH. Correlation between caloric and ocular vestibular-evoked myogenic potential test results. Acta Otolaryngol 2012;132:160–6.