Journal of Neurology (2019) 266:1852–1858
https://doi.org/10.1007/s00415-019-09342-9

ORIGINAL COMMUNICATION

Vestibular symptoms in acute hemispheric strokes

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Received: 21 February 2019 / Revised: 22 April 2019 / Accepted: 24 April 2019 / Published online: 30 April 2019

Abstract
A prospective study focused on whether vestibular symptoms are seen in acute hemispheric strokes, and if so, the frequency and lateralization of causative lesions on MRI. Among 668 patients with hemispheric infarction, we prospectively included those with chief complaints of acute vestibular symptoms, such as vertigo/dizziness, nausea/vomiting and gait instability, in the "VS" group. We also retrospectively reviewed MRI of all stroke patients, and included cases with the findings of parieto-insular vestibular cortex (PIVC) or temporo-periSylvian vestibular cortex (TPSVC) lesion by diffusion-weighted MRI, in the "PIVC" group. Eight patients were found to belong to the VS group, and six other patients to the PIVC group. In the VS group, six patients had the responsible lesion on the right hemisphere, in the middle cerebral artery (MCA) territory except one case and two on the left MCA territory, particularly in the insula, retro-insular region, superior/middle temporal gyrus, angular gyrus, supra-marginal gyrus, putamen and hippocampus/para-hippocampal gyrus. In contrast, none of the six other patients of the PIVC group had vestibular symptoms. One of them had a lesion in the right hemisphere and five in the left hemisphere. Four lesions were located in the insular area and two within the temporal lobe. In conclusion, cerebral hemispheric infarction limited to the PIVC or TPSVC does not necessarily cause vertigo. However, unilateral hemispheric infarctions, restricted to the areas belonging to the vestibular cortical network may cause vestibular symptoms. The lesions responsible for vestibular symptoms are located more often in the right hemisphere.

Keywords Hemispheric infarction · Vestibular syndrome · Vestibular cortex · Vestibular cortical network

Introduction

The vestibular cortex has been identified in various areas in monkeys and humans. The parieto-insular vestibular cortex (PIVC) is the area, centered on the posterior insular cortex, described by Grüsser et al. [1] and plays a central role in the cortical vestibular system [2]. In the human cerebrum, the vestibular cortical area has been confirmed to be a region responding to caloric, galvanic and saccular-otolith stimulations [3–5], which includes the insular gyrus, intraparietal sulcus, inferior parietal lobule, superior temporal gyrus (STG), middle temporal gyrus (MTG), precentral gyrus, middle frontal gyrus, hippocampus, cingulate gyrus, putamen, and thalamus. Furthermore, Kahane et al. identified the area corresponding to the monkey's PIVC by cortical electrical stimulation and named it the temporo-peri-sylvian vestibular cortex (TPSVC), which is mainly located in the temporal neocortex and the parietal operculum [6].

In the central nervous system, the lesions that usually cause vestibular symptoms, such as rotatory vertigo/dizziness, nausea/vomiting and unsteady gait with illusory self-motion, are located in the brainstem. On the other hand, a few studies are known on whether supratentorial hemispheric lesions cause vestibular syndrome. Ten cases of acute vertigo syndrome due to strokes in the middle cerebral territory so far have been reported and all cases were reviewed [7].

Therefore, we conducted a prospective study to answer the following questions. (1) Where are the lesions responsible for acute vestibular syndrome in the supratentorial cerebral hemisphere? (2) How frequently could lesions in the vestibular cortex such as PIVC or TPSVC cause vestibular syndrome? (3) Is there any lateralization of the causative lesion?

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Patients and methods

Patients

We prospectively included 930 consecutive cases with acute cerebral infarction, admitted to the Asanogawa General Hospital between April 2010 and March 2017. The cases with bilateral hemispheric, cerebellar, and brainstem lesions on diffusion-weighted MRI (DWI) in the acute stage were excluded. Finally, a total of 668 cases were included in this study. Among them, we chose those patients with chief symptoms of the vestibular syndrome, defined as "their symptoms consist of acute rotatory vertigo/dizziness with nausea or vomiting and unsteady gait with illusory self-motion, which were designated as the "VS" group". We also retrospectively reviewed and searched for other patients with findings of DWI lesions in the PIVC/TPSVC areas, irrespective of their symptoms and classified them as the PIVC group. We studied their age, sex, lesion side and the anatomical localization of the lesions on the MRIs for statistical analysis. We also retrospectively reviewed the medical record of the patients belonging to the PIVC group to confirm the chief complaints on admission. The PIVC group was further classified into cases with DWI lesions restricted to the insular area, the temporal lobe area, and the parietal operculum area. The insular area includes the insular gyrus, the subcortical white matter of the insular cortex, and the retro-insular region (Ri). The insular cortex was further divided into an anterior and a posterior part by the insular central sulcus. We defined the subcortical white matter of the insular cortex as an area between the extension line of the anterior limiting sulcus, the posterior limiting sulcus, and the superior limiting sulcus, with the basal ganglia surface as the inner edge. The temporal cortex included only the superior temporal gyrus and middle temporal gyrus, and these were divided into an anterior and a posterior part by an extension of the central sulcus. The parietal operculum area included the cortex of the parietal operculum and its subcortical white matter.

Examinations

The patients of the VS group were interviewed and a detailed history was taken on admission, in particular asking about vestibular sensations such as rotatory vertigo/dizziness with nausea/vomiting, imbalanced sensation of illusory self-motion/ataxia, and diplopia or oscillopsia. After this, all patients underwent a standard neurological examination, including neuro-ophthalmological and neuro-otological tests to assess spontaneous gaze nystagmus, smooth pursuit, optokinetic nystagmus, and horizontal and vertical saccades. The patients of the PIVC group were seen in the emergency room and three patients had atrial fibrillation, one had aortic valve replacement, one on chronic hemodialysis and one had two episodes of previous stroke. These patients were transferred to our service within a day after MRI and were soon thereafter fully evaluated with detailed history taking and neurological examinations to confirm the absence of vestibular signs and symptoms.

Radiological study

Brain MRI was performed in all cases. We defined acute cerebral infarction as a high-signal intensity area on the horizontal DWI, with a slice thickness of 5 mm of brain MRI. MRI was obtained routinely on a 3.0-T and, very rarely, on a 1.5-T MRI unit (GE Signa EXCITE HD 1.5 T/3.0 T, USA). The image diagnosis was performed by radiologists and neurologists using DWI with 3D reconstructed images. An independent Chi-squared test (Statcel 3 software, OMS Publishing INC, Japan) was used between the VS group and the control group for the comparison of hemispheric dominance. The significance threshold was set at 0.05.

Results

Eight patients (1.2%) belonged to the VS group (Table 1, Fig. 1), and six other patients (0.9%) to the PIVC group (Table 2, Fig. 2).

VS group

The age of the eight patients ranged from 52 to 82 years. Six patients were men, and two were women. There was a tendency towards right hemisphere dominance (six patients in the right and two in the left hemisphere). Rotatory vertigo was seen in seven patients, and one had a head-motion dizziness with nausea and unsteady gait (Table 1). None of them had nystagmus in the primary and lateral gaze but one patient (case 4) had gaze-induced nystagmus to the right. No other neurological findings were detected except for postural abnormality of unsteadiness upon standing or walking. The responsible hemispheric lesions were analyzed using the DWI with 3D reconstructed images and were found in the insula, Ri (retro-insular), STG (sup. temporal gyrus)/MTG (middle temporal gyrus), angular gyrus (AG), supra-marginal gyrus, hippocampus/para-hippocampal gyrus and putamen (Fig. 1). In Fig. 3, we plotted the responsible lesions 10 mm and 25 mm above the ACPC plane using normal MRI T2-weighted images. The lesions of case 5 (superior temporal gyrus/middle temporal gyrus) and case 8 (angular gyrus)

Table 1 The clinical data of the VS group

	Age	Sex	Lesion side	Handedness	Chief complaint and clinical findings	Anatomical localization of lesions on MRI
Case 1	75	M	R	L	vertigo, nausea no nystagmus	Subcortical white matter of parietal operculum
Case 2	53	M	R	R	vertigo, nausea head-motion dizziness no nystagmus	Subcortical white matter of Ri and the insula
Case 3	63	M	R	R	unsteadiness on standing and walking, nausea no nystagmus	Subcortical white matter of the insula
Case 4	62	M	R	R	vertigo, vomiting gaze nystagmus to right > left	Supra-marginal gyrus
Case 5	71	M	R	R	vertigo, vomiting unsteadiness on walking no nystagmus	STG/MTG
Case 6	52	F	L	R	vertigo, nausea no nystagmus	Putamen
Case 7	82	M	R	R	vertigo, nausea unsteadiness on walking no nystagmus	Hippocampal tail, para-hippocampal gyrus
Case 8	74	F	L	R	positional dizziness, nausea unsteady gait no nystagmus	Angular gyrus, parieto-occipital sulcus

STG superior temporal gyrus, *MTG* middle temporal gyrus

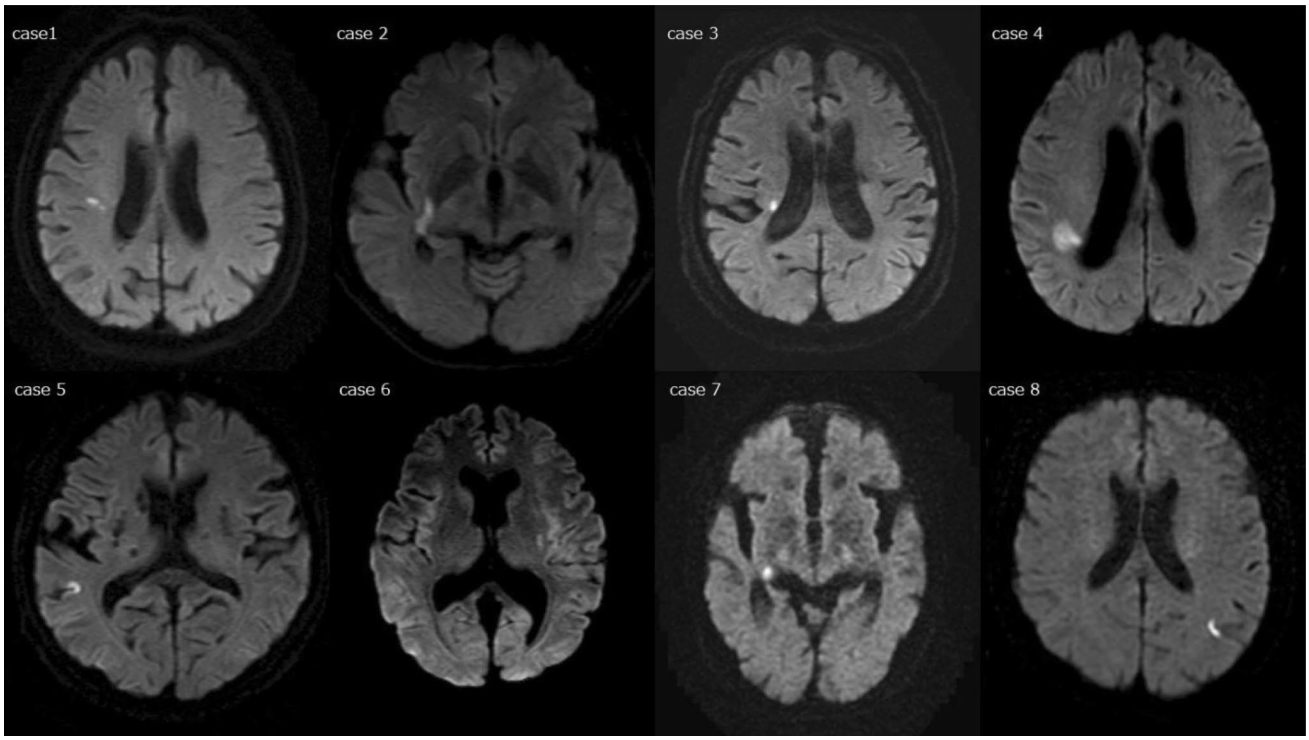

Fig. 1 Each MRI lesion of the VS group with vestibular symptoms

were restricted to the cortex. To compare these lesions to the previously reported ten MRI areas reviewed by Dieterich et al. [7], the causative lesions of six of our patients (cases 1, 2, 3, 4, 6 and 7) were overlapped fairly well to those reported lesions. But our two patients (case 5 and 8) had a lesion over the temporo-parietal cortex, the former over the superior

Table 2 Clinical data of the PIVC group

	Age	Sex	Lesion side	Handedness	Chief complaints and clinical findings	Anatomical localization of lesions on MRI
No. 1	83	F	L	R	dysphasia, right hemiparesis	Insular cortex (long gyrus)
No. 2	85	M	L	R	dysphasia, right facial weakness	The cortex of Ri and insula (long gyrus)
No. 3	62	M	L	R	dysphasia, right hemiparesis	Insular cortex (short gyri III and long gyrus)
No. 4	66	F	L	R	motor aphasia, right hemi-spatial neglect	Insular cortex (long gyrus) Subcortical white matter of insula
No. 5	28	M	R	R	dysarthria, left hemiparesis	Anterior part of STG
No. 6	83	F	L	R	dysphasia, right hemiparesis	Posterior part of STG/MTG Subcortical white matter of the temporal lobe

STG superior temporal gyrus, *MTG* middle temporal gyrus, *Ri* retro-insular region

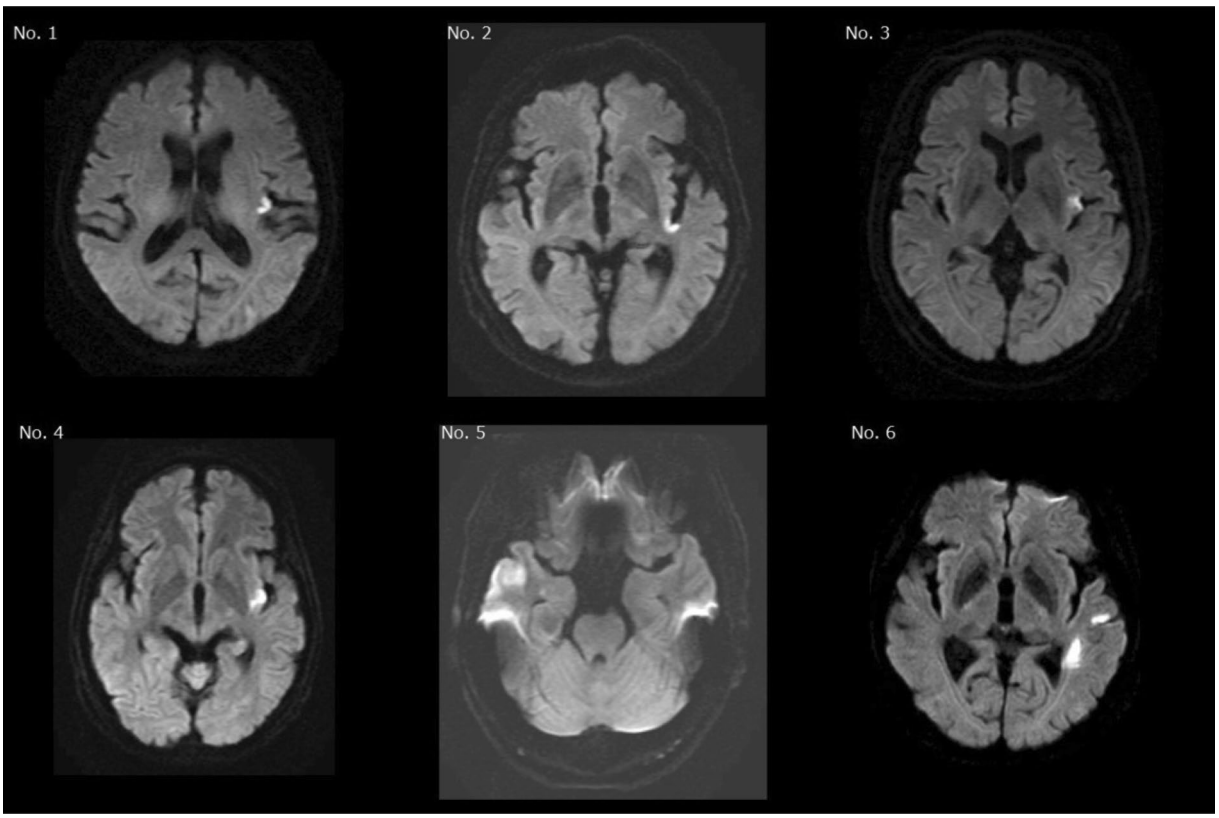

Fig. 2 Each MRI lesion of the PIVC group without vestibular symptoms

and middle temporal gyrus and the latter over the parieto-occipital sulcus. These lesions could be over the separate parieto-temporal vestibular cortex.

PIVC group

The age of these six patients ranged from 28 to 85 years. There were three men and three women in this group. Three patients had atrial fibrillation and one had an aortic valve replacement. Their chief complaints on admission were speech disturbance and mild limb weakness in all six patients. On examination, five patients with a MRI lesion on the left hemisphere had dysphasia/aphasia and one patient with a lesion on the right hemisphere had dysarthria. One patient (no. 4) had a hemi-spatial neglect as well. None had vestibular symptoms on admission. Among this group, two patients had a causative lesion in the temporal lobe (Fig. 2, cases 5 and 6), one was in the anterior superior temporal gyrus and the other in the posterior part of the superior temporal gyrus/middle temporal gyrus within the subcortical white matter.

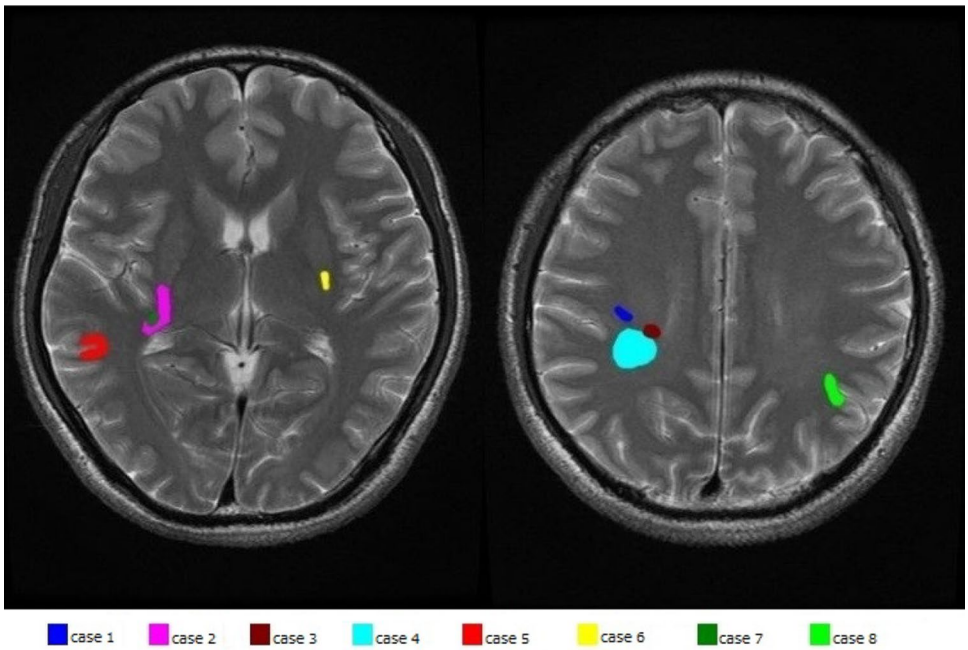

Fig. 3 Superimposed MRI lesions with the VS group in each hemisphere at two levels

Discussion

In our study, the following three points were noted: (1) cerebral hemispheric infarctions that cause vestibular syndrome were localized in the regions recognized as the components of the vestibular cortical network, which has been known to respond to vestibular stimuli [3–6]. (2) Furthermore, these responsible lesions showed hemispheric dominance of the right side. (3) None of patients with radiologically diagnosed PIVC or TPSVC lesions had vestibular sensations such as vertigo/dizziness with nausea/vomiting or gait unsteadiness with illusory self-motion, regardless of the lesion side. Unilateral vestibular cortex lesions do not usually cause rotatory vertigo, nausea, nystagmus or falls [7]. Furthermore none of 112 patients with acute supratentorial strokes caused vertiginous sensations [8]. These reports are supportive of our results.

Responsible DWI lesions in our series were all distributed in the region recognized as the vestibular cortical areas, such as the insular cortex, inferior parietal lobule, parietal operculum, superior temporal gyrus, middle temporal gyrus, precentral cortex, middle frontal gyrus, hippocampus, cingulate gyrus, and putamen. These lesions were rather small in size, and no unique area causing vestibular symptoms was identified. Similarly varied localizations of the hemispheric lesion causing vestibular symptoms have been reported previously [9–16].

Regarding the lateralization of the lesion in the VS group, the responsible lesions were localized in the right hemisphere, within the territory of the vestibular cortical network, in six out of eight patients. This hemispheric dominance has been noted in a paper summarizing previously published cases of acute hemispheric vertigo syndrome [17]. In a study of the human vestibular cortex as identified by caloric stimulation, Fasold et al. reported a strong right hemispheric dominance of the vestibular multisensory cortex areas, regardless of the stimulated side [18]. Furthermore, Brandt et al. hypothesized that the mechanism of visuo-spatial neglect is probably elicited by a vestibular tonus imbalance, based on the analysis of imaging data in patients with neglect, which revealed the overlap of the cortical areas involved with the vestibular cortical areas [19]. Hemi-spatial neglect has been known to result, most commonly, from unilateral injuries to the right cerebral hemisphere. The similarity of the anatomical substrates of human vestibular cortex and spatial neglect has been noted over the years, and spatial neglect has been hypothesized to have a close relationship to a vestibular disorder [20, 21]. Thus, our finding of a right hemisphere dominance of the lesions causing vestibular sensations is not contradictory, but rather confirmative of the previous studies reporting right hemispheric dominance [17–19, 22].

In all cases, acute vestibular symptoms disappeared within 2 days, and this brief duration of the symptoms may be related to the mechanism of "higher vestibular function" recently proposed by Brandt et al. [19]. The central vestibular network to mediate "higher vestibular function" is organized by convergence of multisensory and numerous polysynaptic pathways; therefore, deficits due to a small hemispheric causative lesion can be easily compensated via different modality pathways. So a causative lesion in the cortical multisensory vestibular areas is not necessarily restricted to the cortical structures, as indicated in the

VS group, which included a number of adjacent subcortical white matter small infarcts as the responsible lesion radiologically identified.

In our study, vestibular symptoms did not occur in six PIVC patients with lesions restricted to the insular cortex, which has been thought as "a core vestibular cortex" [2, 23]. But two cases of the VS group had a causative lesion in the subcortical white matter very close to the posterior insular cortex (Fig. 1e, f).

According to the stimulation study of Penfield and Jasper, labyrinthine responses were seen in seven patients out of 108 temporal explorations. They were described as "sinking feeling", "head rest jumping", "dizziness all over", "turning around", "dizziness", "moving around" and "spinning" [24]. Among these seven stimulation sites, six were located over the superior temporal gyrus and one in the inferior parietal operculum. Therefore, their explorations rarely evoked vestibular sensations (6.5%) by temporo-parietal stimulations in patients with temporal lobe epilepsy. A similar stimulation study of the human insular cortex by Mazzola et al. [25] reported that vestibular sensations occurred in 7.6% of the 541 evoked sensations, and were mostly obtained by stimulation of the posterior insula. The evoked vestibular sensations consisted of rotatory sensations in nine cases, and translation sensations in 14 patients, accounting for a total of 23 evoked sensations out of 541 stimulations. A cortical stimulation study, aiming to detect which parts of the cortex can produce vestibular symptoms on stimulation, identified a lateral cortical temporo-parietal area, namely the temporo-periSylvian vestibular cortex (TPSVC), from which all rotatory sensations were elicited in 58.5% of the cases [6]. In their study, only one patient experienced vestibular symptoms during insular stimulation among 44 patients with electrically induced vestibular symptoms. From this evidence, it is clear that the cortical areas responsible for eliciting vestibular symptoms are not necessarily restricted to the posterior insular cortex. Lopez et al. performed a statistical analysis of the localization of the human vestibular cortex, and the results reveal that the main regions activated by caloric and galvanic stimulations are located in the Sylvian fissure, insula, retro-insular cortex, fronto-parietal operculum, superior temporal gyrus, and cingulated cortex [26], which are widely spread over the frontal, temporal and parietal regions. Eickhoff et al., in a study based on fMRI and cytoarchitectonic mapping, hypothesized that the human equivalent of the PIVC is an area they call OP 2, located deep within the Sylvian fissure at the junction of the posterior parietal operculum with the insular/retro-insular region [27]. But the cytoarchitecture of the OP 2 is not as prominent as that of other primary cortices, and they suggested that this OP 2 might not be a strictly unimodal vestibular area. Furthermore, multiple vestibular cortex areas, including the PIVC, have bilateral connectivity from the vestibular nuclei, with three ipsilateral non-crossing and two crossing pathways [17, 20], so that an isolated, unilateral ischemic small lesion of the PIVC might not influence vestibular information processing, as some other intact vestibular cortical areas could take over and assume a compensating role in such processing. Recently, Baier et al. reported no VS or vestibular deficits in ten patients with posterior insular cortex stroke [28], which is in accordance with our results. However, there is another possible explanation regarding why unilateral vestibular cortex lesions do not manifest themselves with vertigo [7]. They propose the concept that the unaffected, opposite hemisphere can suppress vertigo, based on visual–vestibular interactions for motion perception and orientation. To prove or disprove this or our explanations, prospective anatomic–radiologic studies of ischemic MRI lesions in patients with and without vestibular symptoms will have to be performed.

The vestibular system, consisting of the peripheral labyrinths, the nerves, the brainstem/cerebellum, the thalamus, and the vestibular cortical network, subserves a number of perceptual functions, including vestibular symptoms. The vestibular cortex is defined as the multisensory network receiving input from all the above-mentioned areas, from which vestibular signals are coming, as well as signals from somatosensory, visual, and motor areas. The processing of such signals is exceedingly complex, and thus further investigation of these cortical systems may lead to a better understanding of vestibular sensations such as vertigo, dizziness with nausea or illusory self-motion. The recent review of the PIVC indicates not only PIVC but also posterior insular cortex (PIC), a visual-vestibular area, is included as the core of the human vestibular cortex [29].

In conclusion, we confirmed that cerebral hemispheric infarction restricted to the areas belonging to the vestibular cortical network does cause vestibular symptoms, while cerebral infarction limited to the PIVC does not necessarily cause vertigo. From the clinical point of view, the responsible lesions show hemispheric dominance of the right side. The concept of a vestibular cortical network could be applied to explain the presence or absence of vestibular symptoms in patients with hemispheric lesions. A recently established new strategy termed the human brain connectome [30] may provide and improve a map of the vestibular network connections including the white matter in the near future.

Acknowledgements The authors thank Professor Michael G. Halmagyi for his critical reading and suggestions.

Compliance with ethical standards

Conflicts of interest The authors declare that they have no conflict of interest.

Ethical standards The study protocol and patient information and other relevant study documentation were approved by the Hospital's Institutional Review Board. All patients agreed for their medical data to be used for this study without the identity of the subjects.

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