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ORIGINAL COMMUNICATION

Clinical and imaging features of the room tilt illusion

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Abstract Room tilt illusion (RTI) is a transient disorder of the environmental visuo-spatial perception consisting of paroxysmal tilts of the visual scene. It is attributed to an erroneous cortical mismatch of the visual and vestibular three-dimensional coordinate maps. Thirteen subjects were included in this retrospective case series. Clinical presentation was 180° rotation of the visual scene following the coronal plane in seven patients. The most common cause for RTI in our series was posterior circulation ischaemia (five cases). Cases of endolymphatic sac tumour, critical illness neuropathy, acute traumatic myelopathy and multiple system atrophy causing RTI are reported for the first time. No case of supratentorial focal lesion was found. In order to describe the clinical and imaging features of RTI, 135 cases previously reported in the literature were reviewed along with our series. There was a male predominance (60.2 %). Mean age was 51.2 ± 20.3 years. The most common location of the injury was the central nervous system (CNS) (61.4 %). Supratentorial and infratentorial structures accounted for the same frequency of lesions. The most common aetiology was cerebral ischaemia (infarction or transient ischaemic episode; 27.7 %). These patients were significantly older and their lesions commonly involved posterior fossa structures when compared to patients with non-vascular disorders. In summary, RTI is a manifestation of several CNS and vestibular disorders, and rarely of peripheral nervous system disorders, triggered by disruption of vestibular and sensory perception or integration. Cerebral ischaemic disorders are the most common aetiology for this rare syndrome.

Keywords Room tilt illusion · Stroke · Visual tilt illusion · Magnetic resonance imaging · Computed tomography · Vestibular system · Subjective visual vertical

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Introduction

Room tilt illusion (RTI), also known as 'visual tilt', is a transient disorder of environmental visuo-spatial perception consisting of paroxysmal tilting of the visual scene off the true vertical, without any alteration in objects' colour, shape or size [1, 2]. It is a symptom attributed to a transient erroneous cortical mismatch of the visual and vestibular three-dimensional coordinate maps. 'Visual inversion', 'inverted vision', 'upside-down vision' or 'inverted metamorphopsia' are all terms used in the literature referring to 180° rotations in the coronal plane, but RTI may occur in other planes [3]. Images usually rotate following the coronal roll plane or the sagittal pitch plane (Fig. 1), so the mentioned terms do not include the spectrum of manifestations of the RTI phenomenon that may occur in a plane other than the coronal and/or in 90° steps [4–7]. RTI must be differentiated from other visual disorders of verticality perception, such as subjective visual vertical (SVV) tilts. Unlike RTI, SVV tilt is a sign of stable otolithic pathway dysfunction with environmental torsion of a few degrees in the coronal plane lasting days to weeks, in contrast with the

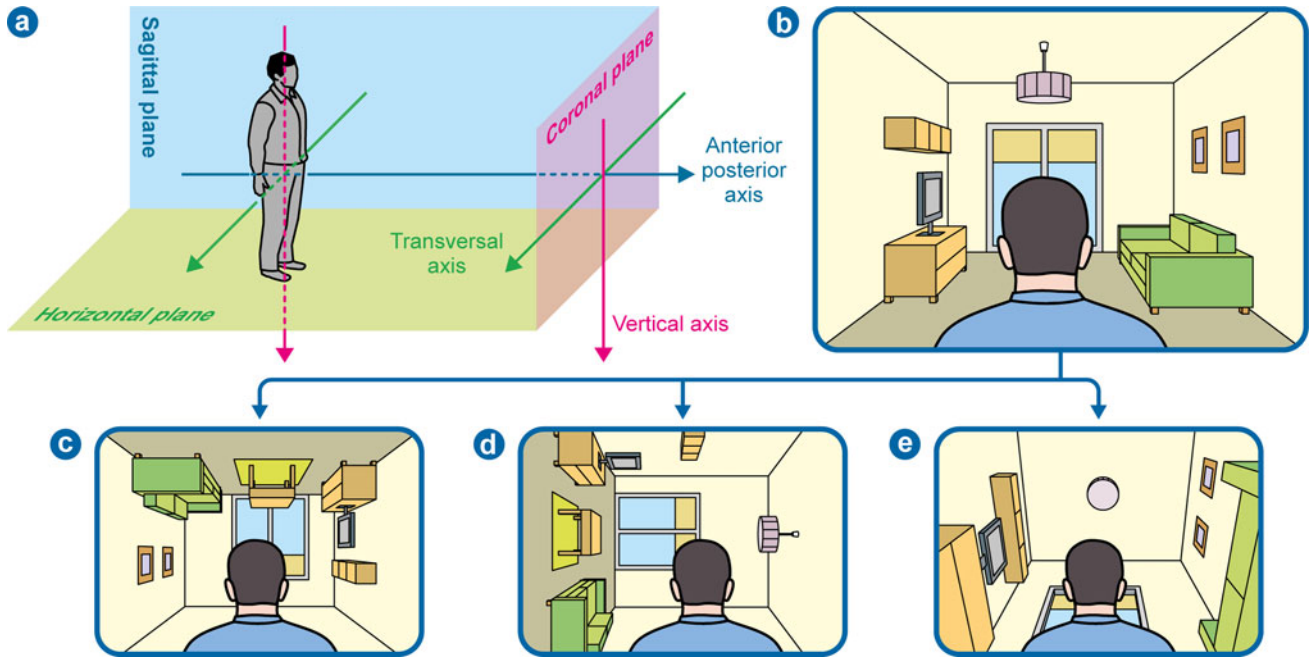

Fig. 1 **a** Three-dimensional representation of axes and planes of the space in relation to a subject; **b** representation of a domestic scene from a subject's view; **c** view of the scene during an RTI of 180° rotation following the coronal plane; **d** view of the scene during a 90° rotation following the coronal plane; **e** view of the scene during a 90° rotation following the sagittal plane

brief, transient, sometimes recurrent, 90–180° tilting of RTI [8, 9].

Bishop first described RTI in 1,805 in a patient thought to suffer from hysteria [10]. Since then, most descriptions of this symptom have tried to provide an anatomical and functional explanation for it. Case reports and short case series have been published, but they only allow a partial view of this phenomenon. Ischaemic, haemorrhagic, demyelinating, infectious, autoimmune, epileptic, traumatic, toxic, neoplastic, neurodegenerative disorders and migraine have been reported to cause RTI because of central nervous system (CNS) or peripheral vestibular system (VS) lesions [3, 5, 11–18]. Development of structural neuroimaging techniques has allowed precise localisation of the injuries in these patients, and a wide spectrum of lesion locations causing RTI, including supratentorial, infratentorial and peripheral vestibular structures, is now recognised. Even peripheral nervous system (PNS) disorders have been reported to cause RTI [19]. A better understanding of the physiology of graviceptive pathways and integrative areas of the brain in the last 2 decades has allowed hypothesising an acute, transient visual and vestibular information mismatch as the better explanation for these paroxysmal phenomena and why such varied lesions involving different structures and pathways may cause the same symptom [1, 20].

As a rare subjective symptom, clinicians need to know RTI's clinical presentation in order to identify possible life-threatening underlying conditions. The aim of this article is to describe the characteristics of RTI in 13 patients who attended our department and to incorporate them into the previously described cases in order to analyse, all together, their clinical and imaging features.

Patients and methods

We retrospectively reviewed the cases of RTI patients attending our department during the 2005–2011 period. The revision was performed using personal records from the authors and medical notes. Patients with at least one episode of transient, paroxysmal visual illusion consisting of rotation of the visual environment, in any direction and of any duration or amount, were included. The records were analysed for sex, age, RTI features (rotation plane, degrees of rotation, duration, number of episodes), associated symptoms and signs, neuroimaging studies, other ancillary tests, presumed location of the injury, final diagnosis and short-term outcome.

Epidemiologic, clinical and imaging features of previously reported cases of RTI were recorded from the literature. An extensive literature search was performed in February 2011 using PubMed, with the following terms: 'Room tilt illusion', 'environmental tilt illusion', 'visual tilt illusion', 'visual inversion', 'inverted vision', 'inverted metamorphopsia', 'upside down vision', 'upside down reversal of vision', 'reversal of vision metamorphopsia' and 'visual environmental rotation'. Additional cases were collected from the references of the articles reporting on

RTI. The same clinical criterion described above for the identification of RTI was used to select the cases for the review. Descriptions of patients with other isolated visual or integrative disorders (e.g. SVV tilt, visual allesthesia, rotated drawing) not associated to the paroxysmal phenomena of RTI were excluded. Data regarding the following variables were collected: year of publication, sex, age, features of RTI (plane, degrees of rotation, duration, number of episodes, accompanying signs), location of the injury, aetiology and neuroimaging tests performed.

Data collection and statistical analysis were performed by means of SPSS 14.0 (SPSS Inc., Chicago, IL, USA). Frequencies are described in total number and percentage. Mean and standard deviation are used for description of continuous variables. All the comparisons were two-tailed. Differences in proportions were tested by χ^2 test. All other comparisons between groups were made with Student's unpaired t test. p values <0.05 were considered to be statistically significant.

Results

Ten male and three female patients aged between 35 and 82 years old were included. Clinical features are detailed in Table 1. The illusion was described as an 'upside-down' reversal of vision (180° rotation in the coronal plane) in seven patients. A 90° rotation in the coronal plane occurred in three patients, two in clockwise and another one in counter-clockwise direction. Three more patients had a 90° forward rotation in the sagittal plane. Five subjects had one isolated episode of RTI and eight patients had two or more episodes. Only one patient (case 7) suffered from recurrent RTI for 1 year. The rest of patients had RTI episodes in an acute-subacute manner. The duration of the phenomenon was highly variable, ranging from seconds to hours. RTI episodes were triggered by changes in head position in two patients, up to the point that the episodes ran exclusively when rolling over on the right side in case 6 and when lying down in a supine position in case 13.

Cases 1, 2, 3 and 4 presented RTI episodes secondary to acute posterior circulation infarctions involving different posterior fossa structures. They presented with a single episode of the visual illusion during the hyperacute phase of the stroke, which was confirmed by magnetic resonance imaging (MRI) in every case (Fig. 2). Case 5 was a 65-year-old man who suffered a fall from 3 m height. A cervical computed tomography (CT) scan demonstrated a C1 Jefferson's fracture. Two days after admission, he presented several episodes of 180° environmental rotation illusion lasting for several minutes. A brain MRI was normal. A digital subtraction angiography suggested the presence of a dissection of the intracranial segment of the right vertebral artery (Fig. 3). Intravenous heparin was then started and RTI episodes disappeared within 24 h.

Patients 6, 7 and 8 were diagnosed with VS disorders. Case 6 was a patient that presented with several episodes of RTI that lasted for seconds and appeared only when lying on his right side during an acute right vestibular syndrome consistent with vestibular neuritis. Case 7 was a 62-year-old man with a previous diagnosis of Meniere's disease who complained of recurrent episodes of upside-down vision of variable duration (from minutes to hours) accompanying vertigo spells. Patient 8 had a first-ever episode of right posterior canal benign paroxysmal positional vertigo (BPPV). She reported suffering an RTI during Epley's repositioning manoeuvre. The illusion lasted longer than the evoked nystagmus, and it was only relieved when she closed her eyes for several seconds. She had never experienced such a visual phenomenon, and it did not appear again during the manoeuvre.

Patient 9 had a 7-year-history of right deafness without any medical assessment. He attended the emergency room because of an acute vestibular syndrome and three consecutive episodes of RTI of 180° in the coronal plane. MRI demonstrated a right ponto-cerebellar angle-occupying tumour with ipsilateral cerebellar hemisphere compression and oedema (Fig. 3). Biopsy proved it to be a low-grade endolymphatic sac tumour. It was surgically removed and later radiated with good further evolution.

Patient 10 was a 60-year-old man with multiple bone adenocarcinoma metastases of unknown origin who underwent surgery for a pathologic femur fracture. He experienced several RTI episodes lasting for 10 min during the immediate postoperative period, in close relationship to IV morphine initiation. The episodes disappeared after morphine pump discontinuation, but this was reintroduced again a few days later because of poor pain control. RTI episodes returned for some days until the pain was relieved and morphine therapy could be stopped. A cranial CT scan and an electroencephalogram (EEG) showed no abnormalities. An MRI was contraindicated because he had a pacemaker. The RTI was attributed to opioid intoxication.

Patient 11 had a past medical history of poliomyelitis and right-sided deafness after a surgical intervention for cholesteatoma removal 6 years before. She was admitted to the intensive care unit (ICU) with respiratory failure because of bilateral pneumonia and developed a severe critical illness neuropathy. She experienced three episodes of 90° RTI following the sagittal plane when she was transferred to the conventional medical ward. A brain MRI and an inner ear CT scan showed no abnormalities apart from previous right ear surgery changes. The EEG was normal. Nerve conduction studies and an electromyogram confirmed the presence of an acute severe sensory-motor axonal polyneuropathy.

Table 1 Features of RTI cases attended at our institution

Case	Sex	Age	Number of episodes	Room tilt illusion features				Imaging	Diagnosis	Location	Associated symptoms and signs, and observations
				Duration	Plane	Degrees	Direction				
1	M	46	1	5 min	C	180	–	MRI	Infarction	Cerebellum (right SCA)	Vertigo, nausea, vomiting, truncal ataxia, gaze-evoked nystagmus (R > L)
											Normal MRA
2	M	58	1	30 s	S	90	Fw	MRI	Infarction	Lateral medulla (right PICA)	Dysphonia, ataxia, right cerebellar syndrome, gaze-evoked nystagmus (R > L), right vestibular syndrome, left pinprick and thermal hypoesthesia
											Traumatic vertebral artery dissection (MRA)
3	M	59	1	5 min	C	90	Cw	MRI	Infarction	Cerebellum (right SCA)	Vertigo, nausea, vomiting, headache, gaze-evoked nystagmus, right cerebellar syndrome, truncal ataxia
											Previous atherosclerotic posterior circulation TIAs
											Mild Charcot-Marie-Tooth disease type 1A
											Mild bilateral familial presbyacusis
											Normal MRA
4	M	35	1	1 h	C	180	–	MRI	Infarction	Lateral medulla (right PICA)	Truncal ataxia, gaze-evoked nystagmus
											PICA obstruction (MRA)
											Cryptogenic stroke
5	M	62	7	2–3 min	C	180	–	MRI	Recurrent TIA?	Vertebrobasilar territory	Traumatic right vertebral artery dissection
6	M	73	"Several"	Seconds	C	90	Ccw	CT	Vestibular neurits	VIII cn (right)	HT nystagmus towards the left, RTI only when lying over the right side
											Improvement with vestibular suppressants
7	M	62	Countless	2–3 h	C	180	–	CT	Meniere's disease	Inner ear (left)	RTI accompanying vertigo spells
											No improvement with vestibular suppressants
8	F	67	1	2 min	C	90	Cw	No	Posterior canal BPPV	Inner ear (right)	Torsional nystagmus, RTI during Epley manoeuvre
9	M	60	3	5–7 min	C	180	–	MRI	Endolymphatic sac tumour	Cerebellum + VIII cn (right)	HT nystagmus towards the left, right deafness
10	M	64	"Several"	10 min	C	180	–	CT	Opioid intoxication	Diffuse	Metastatic adenocarcinoma of unknown origin
11	F	66	3	3, 8, 60 min	S	90	Fw	MRI	Critical illness neuropathy	Peripheral nerve	Operated right-sided cholesteatoma
12	F	82	3	2–3 min	C	180	–	MRI	Haemorrhagic contusion	Cervical spinal cord	Severe presbyacusis
13	M	66	Countless	5 min	S	90	Fw	MRI	MSA	Cerebellum	RTI only when lying in supine
											Mild alcoholic neuropathy

BPPV benign paroxysmal positional vertigo, *C* coronal, *Cw* clockwise, *Ccw* counter clockwise, *CT* computed tomography, *F* female, *Fw* forward, *GE* gaze evoked, *HT* horizontal-torsional, *L* left, *M* male, *MRA* magnetic resonance angiography, *MRI* magnetic resonance imaging, *MSA* multiple system atrophy, *PICA* posterior inferior cerebellar artery, *R* right, *S* sagittal, *SCA* superior cerebellar artery, *TIA* transient ischaemic attack, *VIII cn* vestibulo-cochlear nerve

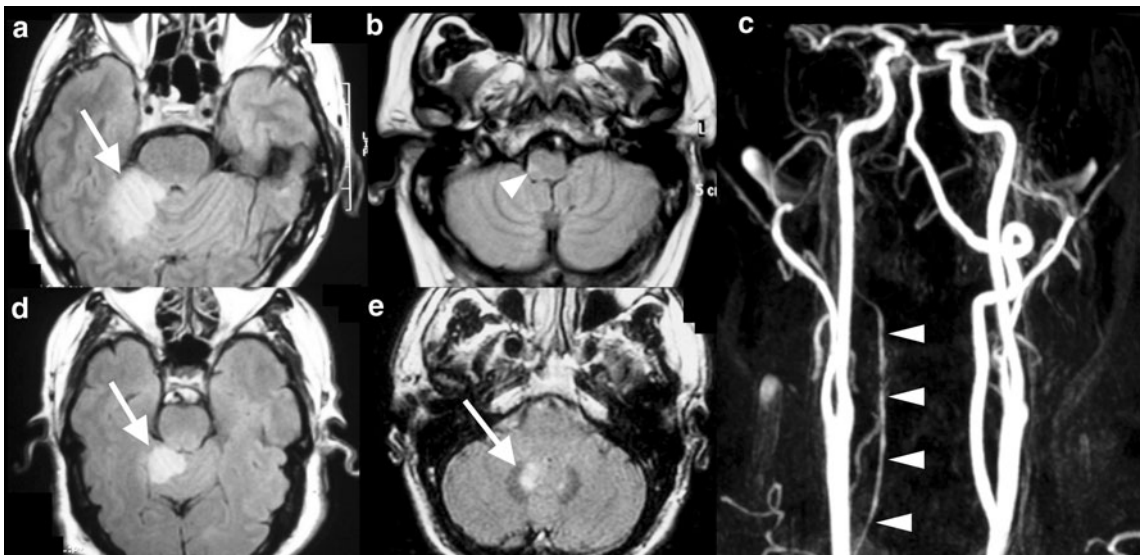

Fig. 2 Brain MRI of patients with neurovascular disorders causing RTI: **a** case 1, axial FLAIR, right SCA infarction (*arrow*); **b** case 2, axial FLAIR, right lateral medullary infarction (*arrowhead*); **c** case 2, anterior-posterior view of MRA, irregular narrowing of the whole course of the right vertebral artery (*arrowheads*); **d** case 3, axial FLAIR, right SCA infarction (*arrow*); **e** case 4, axial DWI, right PICA infarction (*arrow*). *DWI* diffusion-weighted imaging, *FLAIR* fluid-attenuation inversion-recovery, *MRA* magnetic resonance angiography, *MRI* magnetic resonance imaging, *PICA* posterior-inferior cerebellar artery, *SCA* superior cerebellar artery

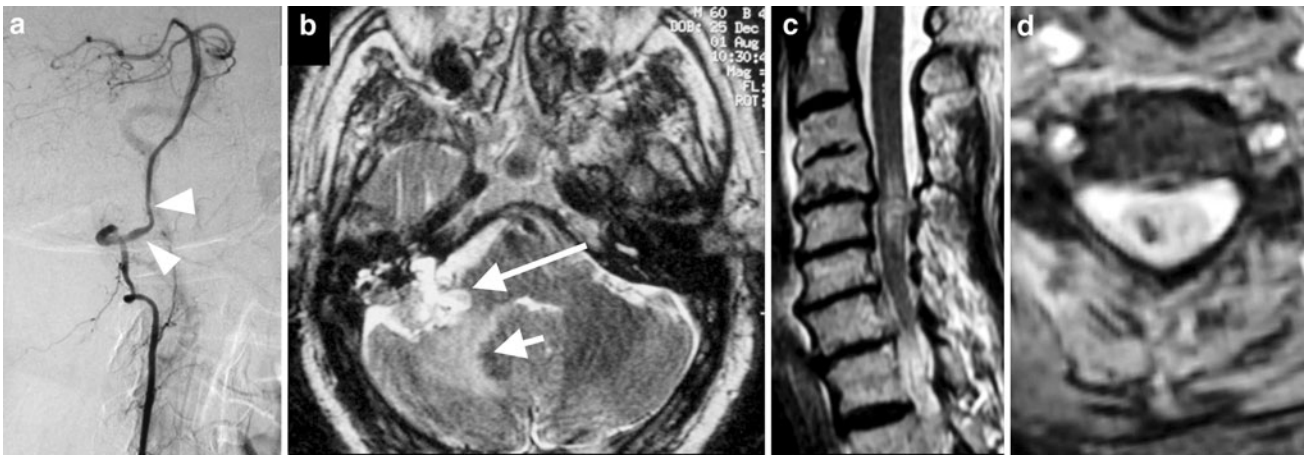

Fig. 3 Neuroimaging of patients with RTI caused by other aetiologies than brain infarction. **a** Case 5, digital subtraction angiogram, anterior-posterior view, irregular narrowing of the intracranial course of right vertebral artery (*arrowheads*); **b** case 9, axial T2-weighted brain MRI, ponto-cerebellar angle irregular mass imprinting cerebellum (*arrow*) and causing right hemisphere oedema (*short arrow*); **c** case 12, sagittal T2-weighted cervical MRI, traumatic C5 myelopathy with cord oedema; **d** case 12, axial gradient-echo weighted MRI, C5 level, punctiform focus of central intramedullary bleeding. *MRI* magnetic resonance imaging

Patient 12 was an 82-year-old woman with severe bilateral presbyacusis admitted with reduced mobility following a fall. She presented an asymmetric quadriparesis, right vibration and position hypoesthesia, and a left deficit to pinprick and thermal examination with a C7–C8 sensory level. A cervical MRI showed a posterior-central haemorrhagic contusion at the C5 level, which was managed conservatively (Fig. 3). She had three episodes of RTI in the ICU during the first 24 h after the fall.

Patient 13 was a 66-year-old man with the antecedent of mild alcoholic axonal neuropathy who was admitted with recurrent orthostatic syncopes. He complained of two or three 90° sagittal RTI episodes every night during 2 months triggered by lying supine in bed. He had symptoms consistent with autonomic failure, and presented cerebellar signs and a symmetric hypokinetic-rigid syndrome on examination. Eye movements appeared to be full, and no spontaneous or positional nystagmus existed.

A brain MRI was normal. A video-nystagmography demonstrated hypometric horizontal and vertical saccades, complete loss of optokinetic nystagmus and broken pursuit. The EEG was normal. [^{123}I] FP-CIT SPECT (DaT-SCAN^{TM}) demonstrated a bilateral reduction of striatal dopamine transporter. A diagnosis of multiple system atrophy (MSA) was made. Dysmetria and parkinsonism slowly progressed during the following months, and gaze-evoked nystagmus and progressive truncal ataxia became evident. Treatment with benzodiazepines to reduce the frequency of RTI episodes was unsuccessful, but they spontaneously disappeared 2 months after the initial evaluation.

Overall, MRI demonstrated parenchymal brain lesions involving the posterior fossa in five patients (cases 1, 2, 3, 4 and 9), and two more patients showed other visible injuries outside of the brain (cases 5 and 12). EEG was performed on those patients without apparent neurovascular or vestibular causes for the RTI phenomenon (cases 10, 11, 12 and 13), showing absence of epileptiform discharges in every case. The most common final diagnosis was vertebrobasilar territory infarction (four cases). Every patient had some kind of NS or VS disorder when investigated: six cases were diagnosed to be secondary to brainstem or cerebellar disorders, one case was considered to have a toxic mechanism with probable diffuse CNS involvement, three cases appeared in relation with VS disorders, and two cases were thought to be triggered by acute peripheral sensory deafferentation (cases 11 and 12). A definite diagnosis could not be established in patient 5 because of the existence of several potential mechanisms of injury despite the absence of a structural parenchymal lesion in MRI. Recurrent transient vertebrobasilar ischaemia secondary to traumatic vertebral artery dissection was the most plausible explanation for the symptoms of this patient.

Literature review

One hundred thirty-five previously published cases of RTI were included in this review. After the first description by Bishop in 1805, a century passed until RTI was again reported [10, 21]. During the first 90 years of the twentieth century, few papers reporting RTI appeared in the literature [21–40]. Early reports, most of them by German authors, were reviewed by Solms et al. [41] in 1988 in the only literature review on this topic. Cases of RTI have been increasingly published during the last 2 decades (73 cases during the 1991–2010 period) [42–59]. Before 1991, 25 of the 62 published cases were reported by Justo Gonzalo [15], a Spanish neuroscientist who deeply studied perceptual disorders as sequels of traumatic brain injury in soldiers from the Spanish civil war. He profusely analysed the cases of 25 patients with parieto-occipital lesions and RTI.

Table 2 Location of lesions causing RTI (previously described and own cases)

Anatomical structure	Injuries	
	Number	%
Central nervous system	97	61.4
Posterior fossa	48	30.4
"Vertebrobasilar territory"	15	9.5
Cerebellum	14	8.9
Medulla	10	6.3
Pons	5	3.2
Mesencephalon	1	0.6
"Brainstem"	3	1.9
Supratentorial	48	30.4
Parieto-occipital	29	18.4
Parietal lobe	7	4.4
Occipital lobe	3	1.9
Frontal lobe	3	1.9
Temporal lobe	3	1.9
Thalamus	3	1.9
Spinal cord	1	0.6
Vestibular system	31	19.6
VIII cranial nerve	11	7.0
Inner ear	20	12.7
Peripheral nervous system	4	2.5
Diffuse/Multiple	6	3.8
Unknown/Not specified	20	12.7
Total[a]	158	100.0

[a] As some patients had lesions of more than one structure, the total number of lesions is higher than number of subjects

After including our own 13 cases, a total of 148 patients suffering RTI have been reported in the literature. Gender was male in 60.2 % and female in 39.8 % ($n = 98$). Mean age was 51.2 ± 20.3 years (range 8–85 years; $n = 96$). RTI was caused by single focal injuries to the NS or the VS in 117 patients (79.1 %); by multifocal injuries in 9 (6.1 %); by diffuse CNS injuries in 2 (1.4 %); and by unknown or not specified cause of the injury in 20 cases (13.5 %). Location of the injuries causing RTI was varied (Table 2). Injuries were located involving posterior fossa structures and supratentorial structures (mainly the parieto-occipital junction) in the same number of cases (48 injuries each). VS injuries were reported in 31 patients (19.6 %). Only four patients have been reported to suffer from PNS disorders causing RTI: three previously described cases of Guillain-Barré syndrome and patient no. 11 of the present study [19].

The aetiology of RTI was attributed to CNS disorders in 98 patients (66.2 %), to VS disorders in 29 (19.6 %), to PNS disorders in 4 (2.7 %) and had unknown causes in 16 cases (10.8 %) (Table 3). Among CNS disorders, the cause

Table 3 Aetiology of RTI (previously described and own cases)

Disorder	Number	%
Central nervous system disorders	98	66.2
Focal disorders	84	56.8
Infarction	32	21.6
Traumatic brain injury	27	18.2
Transient ischaemic attack	9	6.1
Epilepsy	5	3.4
Intracranial tumour	5	3.4
Haematoma	3	2.0
Surgical brain injury	2	1.4
Brain abscess	1	0.7
Multifocal/Diffuse disorders	14	9.5
Migraine	5	3.4
Multiple sclerosis	3	2.0
Neurocysticercosis	1	0.7
Opioid intoxication	1	0.7
Multiple system atrophy	1	0.7
Parkinson's disease	1	0.7
Susac's syndrome	1	0.7
Encephalitis	1	0.7
Vestibular disorders	29	19.6
Menière's disease	13	8.8
Vestibular neuritis	6	4.1
Benign paroxysmal positional vertigo	4	2.7
VIII cranial nerve section	3	2.0
Ramsay-Hunt syndrome	1	0.7
Perilymphatic fistula	1	0.7
Laberintectomy	1	0.7
Peripheral nervous system disorders	4	2.7
Guillain-Barré syndrome	3	2.0
Critical illness neuropathy	1	0.7
Unknown mechanism	16	10.8
After mild cranial trauma	1	0.7
Other/not specified	15	10.1
Hysteria	1	0.7
Total	148	100.0

of RTI was diagnosed to be vascular in origin (brain infarction, transient ischaemic attack or intracerebral haemorrhage) in 44 cases (44.9 % of all CNS disorders). Patients suffering RTI of vascular origin were significantly older than patients with RTI of other causes (63.6 ± 12.3 years vs. 46.5 ± 21.4 years; $n = 82$; $t = 4.569$; $p < 0.0001$), and their injuries were more commonly located in posterior fossa structures (97.2 % of patients with a vascular cause vs. 9.5 % among patients with non-vascular cause; $\chi^2 = 79.079$; $df = 2$; $n = 110$; $p < 0.0001$). Posterior circulation infarctions aggregated involving the paramedian pons, superior cerebellar artery and posterior-inferior cerebellar artery territories (Fig. 4). No differences existed in sex, duration or number of RTI episodes between vascular and non-vascular aetiologies.

RTI occurred in the coronal plane in 102 patients (87.2 %; mean rotation of $155.5 \pm 43.1^\circ$; 75 of them had 180° rotations, 21 had 90° rotations, and 6 suffered rotations of 30°, 45°, 150° or 160°) and in the sagittal plane in 15 cases (12.8 %; all of them had tilts of 90°) ($n = 117$). Forty-two patients experienced a single RTI episode (47.7 %), 32 had 2–10 episodes (36.4 %), and 14 (15.9 %) had more than 10 episodes ($n = 88$). The duration of the episodes was not precisely defined in the majority of the manuscripts, so it was classified in the range of seconds (26 cases; 28.6 %), minutes (49; 53.8 %), hours (12; 13.2 %) or days (4; 4.4 %) ($n = 91$). No statistical relationship existed between the location of the injuries and planes of scene rotation ($\chi^2 = 2.754$; $df = 2$; $n = 83$; $p = 0.25$), duration of RTI ($\chi^2 = 8.846$; $df = 6$; $n = 72$; $p = 0.18$) and number of episodes ($\chi^2 = 6.121$; $df = 4$; $n = 67$; $p = 0.19$).

A total of 49 patients underwent MRI and 20 underwent CT, all of them from 1988 to the present time. Patients who underwent a neuroimaging test were more commonly diagnosed to have an infratentorial lesion (79.5 % with neuroimaging vs. 27.9 % without neuroimaging; $\chi^2 = 21.816$; $n = 82$; $p < 0.00001$), and so a vascular aetiology for the RTI (49.2 % with neuroimaging vs. 18.6 % without neuroimaging; $\chi^2 = 14.057$; $df = 1$; $n = 133$; $p < 0.001$).

The above-reported 13 patients were older than those previously reported in the literature (61.5 ± 21.0 years vs. 49.5 ± 11.5 years; $t = 3.046$; $n = 96$; $p < 0.01$). No statistical differences existed between our own and previously reported cases in the frequency of rotation in each plane ($\chi^2 = 1.376$; $df = 1$; $n = 117$; $p = 0.24$), number of episodes ($\chi^2 = 0.679$; $df = 2$; $n = 88$; $p = 0.71$), duration of the illusions ($\chi^2 = 3.437$; $df = 3$; $n = 91$; $p = 0.33$) and frequency of vascular aetiology ($\chi^2 = 0.439$; $df = 1$; $n = 133$; $p = 0.51$). A statistical difference existed in the location of the injuries between both groups (the present series does not include any patient with RTI caused by a supratentorial lesion), but this significance disappeared when compared only to previously reported patients who underwent a CT scan or an MRI ($\chi^2 = 2.197$; $df = 2$; $n = 50$; $p = 0.33$).

Discussion

Spatial orientation mainly depends on visual-vestibular integration on a three-dimensional coordinate system [1]. Two simultaneous verticality perceptions cannot exist, so a mechanism that integrates the information provided by

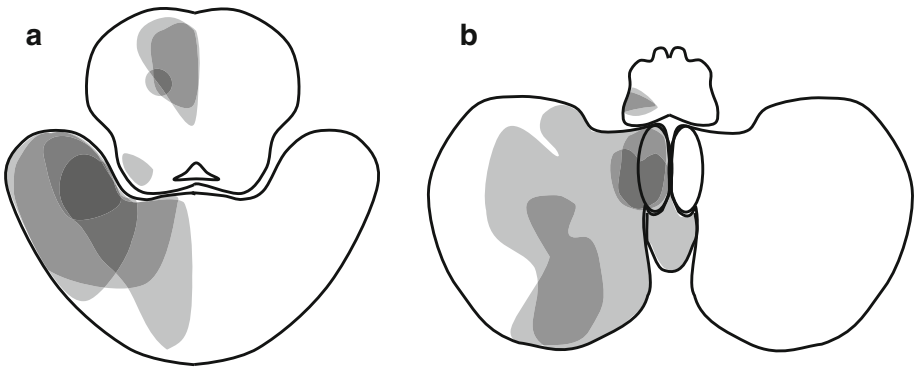

Fig. 4 Lesion mapping showing the location of 12 vertebrobasilar territory infarctions with available MRIs (from cases 1, 2, 3 and 4, and references 6, 7, 11, 46, 51, 55, 56 and 58). All the lesions were located on the right side for illustrative purposes. **a** Section through the pons; six infarctions involving the paramedian pons and the SCA territory; **b** section through the medulla oblongata; six infarctions involving the lateral medulla and the medial portion of the cerebellar hemisphere in the PICA territory. *MRI* magnetic resonance imaging, *PICA* posterior-inferior cerebellar artery, *SCA* superior cerebellar artery

these systems must exist. The perception of verticality is mediated by inputs from the vestibular, somatosensory and visual systems, indicating verticality in an independent but congruent manner in healthy subjects [9]. These signals converge and are processed in the vestibular nuclei, and are then projected to the cortex via ventral lateral and ventral posterior nuclei of the thalamus [60]. The right hemisphere plays a predominant role in the perception of the postural vertical and a critical role for the elaboration of an internal model of verticality [20]. When a conflict between the inputs appears, one of them should be selected and used as the perceived verticality [1].

RTI may be the result of lesions involving the necessary inputs to create a verticality perception, not only the associative cortical areas. The cerebral cortex needs a minimum balance of information coming from vestibular, visual and somatosensory systems to integrate them in a correct way. A sudden complete loss of one of the inputs may cause a disruption of the balanced integrative process. Most patients were examined by neuroimaging techniques in the present series, identifying posterior fossa lesions in 6 out of 13 patients. We could not demonstrate cortical lesions in any of the cases. Vascular aetiology of RTI in our patients, two of them showing vertebral artery dissection, was similar in frequency to the one reported in the literature [47]. The results of the literature review suggest that ischaemic posterior fossa lesions were probably under-recognised before the systematic use of neuroimaging techniques, leading to the erroneous perception of parieto-occipital cortex injuries being the most common cause of RTI. Taking into account that MRI is the most precise imaging test for assessing the integrity of posterior fossa structures, we would recommend performing this study in every patient complaining of RTI. Other imaging modalities should be considered when clinical suspicion of another disorder not involving brainstem or cerebellum exists.

RTI usually consists of 90° or 180° steps as the erroneous result of the attempt to match the pair of axes in acute vestibular tone imbalance, but six patients were reported to suffer visual scene rotations of 30°, 45°, 150° or 160°, not according to this scheme of solving visual and vestibular conflicts with 90° step rotations in a three-dimensional coordinate cortical system [1, 7, 17, 35, 39, 44]. This variation may just be the result of patient's difficulties to explain this confusing symptom, or it may be the expression of a residual ability of the vestibular nuclei or integrative cortex to adapt the inputs together in a proportional way. This would result in a midway perception, non-predominant for both inputs [61].

Some patients could trigger the illusion by means of changing their head position, so changing their inputs from the vestibular system and therefore modifying their processing balance between visual, somatosensory and vestibular information. On the other hand, some patients could relieve the illusion by means of closing their eyes for a moment, suggesting a reorganisation of cortical integration that quickly adapts to new demands from disrupted inputs. Short duration of the phenomena, common recurrence and rapid disappearance of the transient illusions after some minutes or hours suggest that RTI evolves in a dynamic compensation process.

We have described cases of RTI occurring in patients without any acute vestibular or visual disorders, but carrying recently acquired sensory disorders by means of lesion of the PNS or of central sensory pathways into the spinal cord. These cases expand the spectrum of lesions causing RTI and cannot be explained by a visual-vestibular mismatch in isolation. Somatosensory inputs are involved in the perception of subjective haptic (tactile) vertical stimuli derived from proprioception and touch [62]. The two cases of somatosensory deafferentation described in our series (patients 11 and 12) were carriers

of a chronic compensated vestibular dysfunction. It is likely that disruption of somatosensory inputs may not be enough in isolation to confuse the verticality perception system, but may trigger a mismatch of visual and vestibular cues in patients with a previously fragile balance on central integration, leading to an erroneous verticality perception.

We report several cases of RTI caused by not previously described aetiologies. Endolymphatic sac tumours only comprise 4 % of tumoral lesions of the temporal bone, and to our knowledge it has not been previously reported as a cause of RTI [63]. Three cases of Guillain-Barré syndrome with visual hallucinations and illusions including RTI were previously reported, but other types of PNS involvement had never been associated with RTI before [19]. The addition of critical illness neuropathy in a patient with a previous vestibular lesion may explain the visual illusion in patient 11. A spinal cord lesion has never been reported to be associated with RTI. In case 12, an acute spinal cord lesion could act as a trigger for RTI in this patient. RTI has been reported in one patient with Parkinson's disease, but never reported in MSA [18]. Case 13 showed clinical and functional evidence of cerebellar dysfunction in the setting of a newly diagnosed MSA. No other peripheral vestibular or focal cerebral disorder was found in functional or imaging tests, so we assumed the recurrent RTI was a symptom of cerebellar degeneration.

The limitations of this study come from its retrospective design. A few reported cases were probably missed in the literature search, and other cases were not included because they did not meet inclusion criteria regarding RTI information. Unexplained cases may not have been published because of a publication bias.

In conclusion, RTI is an uncommon transient visual symptom probably caused by disruption of vestibular or proprioceptive inputs to the integrative parieto-occipital cortex, causing an imbalance that leads to anomalous verticality perception. It usually presents with an 180° rotation of the visual scene following the coronal plane, although other planes and extents of rotation of the visual surrounding may occur. The most common causes are single focal CNS injuries, mainly posterior circulation stroke. It is important for the clinicians to be aware of this syndrome because it may be the initial manifestation of a potentially severe neurological disorder despite its innocent appearance. A careful history should be taken in order to identify data from current complaints, physical examination and past medical history (mainly associated factors, such as vestibular or PNS disorders) that may give the clue to make an accurate diagnosis. MRI should be performed in every patient to rule out posterior fossa lesions that may not be evident in a cranial CT scan.

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

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