## **Reversal of Vision Metamorphopsia**

### Clinical and Anatomical Characteristics

Yaron River, MD; Tamir Ben Hur, MD, PhD; Israel Steiner, MD

**Background:** Metamorphopsia is a visual illusion that distorts the size, shape, or inclination of objects. Reversal of vision metamorphopsia (RVM) is a rare transient form of metamorphopsia described as an upside-down, 180° rotation of the visual field in the coronal plane. The pathophysiological characteristics of RVM remain unclear.

**Design:** Patients with RVM had a complete neurologic examination during or shortly after an episode of metamorphopsia, with particular emphasis on gaze disorders, visual fields, visually guided hand movements, and perceptual or cognitive deficits. Workup included imaging studies, visual field examinations, and brainstem auditory and visual evoked response.

**Setting:** Department of Neurology, Hadassah University Hospital, Hebrew University-Hadassah Medical School, Jerusalem, Israel.

**Patients:** Six consecutive patients were evaluated from 1991 to 1996.

**Results:** Five patients had parieto-occipital brain insult sparing the primary visual cortex, and 3 also had evidence of a concomitant brainstem or cerebellar syndrome. One patient had pure brainstem syndrome underlying the RVM. Three patients had complete RVM as well as oblique RVM of less than 180°.

**Conclusions:** These cases imply a possible anatomical localization of the central integrator of visual extrapersonal orientation. Our observations suggest that a separate central mechanism of visual orientation might exist in each cerebral hemisphere and that occipital and parietal lesions that spare the optic radiations may account for the oblique and complete RVM. We postulate that failure to perceive space in an allocentric coordinate frame, particularly in the coronal roll plane, is potentially the critical event underlying RVM.

Arch Neurol. 1998;55:1362-1368

characterized by an optic illusion that alters the size, shape, or angulation of objects. It is a rare manifestation of an acute central nervous system insult, mainly to the visual or vestibular systems.1 Reversal of vision metamorphopsia (RVM) is a transient form of metamorphopsia described as an upside-down, 180° alteration of the visual field in the coronal plane.<sup>2</sup> The first case of RVM was reported by Winslow<sup>3</sup> in 1868 as a transient phenomenon of hysteria. The transient nature of this phenomenon was clearly evident in this early report as well as in subsequent cases. Only rarely was it observed as a stable phenomenon lasting days or weeks.<sup>4</sup> Klopp<sup>5</sup> reviewed the German literature and added 3 of his RVM cases. He was able to find 13 patients with this disorder: 2 had nonorganic or psychogenic disturbance, 5 had cortical (mainly parietal) lesions, 4 had brainstem lesions, and 2 had cerebellar lesions. The causes of RVM are diverse, including head trauma, tumors, and stroke. Klopp<sup>5</sup> suggested that RVM is not caused by an insult to the optic pathways but rather by a central nervous system disturbance of the vestibular system affecting the perception of the body in space or the perception of the subjective visual vertical.

# For editorial comment see page 1285

The most thorough review of RVM is by Solms et al,<sup>6</sup> who analyzed 21 cases. They conclude that RVM is a transient, sudden, and paroxysmal phenomenon. It is perceived as an actual torsion of the visual array with 180° rotation in the coronal plane, usually in a clockwise direction. Tilting of the visual field less then 180° (for example, 90°) in the coronal

From the Department of Neurology, Hadassah University Hospital, Hebrew University-Hadassah Medical School, Jerusalem, Israel.

> ARCH NEUROL/VOL 55, OCT 1998 1362

ETAMORPHOPSIA is

plane or RVM of the sagittal and horizontal planes is associated with common coronal RVM in some of the cases reviewed by Solms et al. Vertigo and malaise are common, but distorted egocentric orientation (ie, the patient experiences standing on his head or feels that his body is tilted away from the vertical<sup>7,8</sup>) is rare.<sup>6</sup>

In the last 5 years, we have observed 6 patients with RVM. Our evaluation of these cases has contributed to our understanding of this disorder.

#### **REPORT OF CASES**

#### CASE 1

A 60-year-old patient was admitted to the emergency department reporting that he suddenly heard a "loud voice" immediately followed by dizziness and weakness in his right hand. His medical history included hypertension, hypercholesterolemia, type 2 diabetes mellitus, and ischemic heart disease. On examination, the patient's blood pressure was 160/100 mm Hg and his pulse was 60/min and regular. Findings from a general physical examination were normal. Neurologic examination revealed a conscious, fully alert, and mentally intact patient with right-sided dysmetria, gait ataxia with a tendency to fall to the right, and a horizontal rotatory nystagmus on right lateral gaze. A vertical nystagmus was observed on gazing upward. Eight hours after the appearance of dizziness and weakness in the right hand, the patient experienced a visual disturbance. The objects in his right visual field appeared upside down. People were seen walking on their heads, the windows close to the ceiling were within reach of his hands, and a cup stood "upside down" on the shelf but the "tea did not spill out" (all the objects including the shelf were upside down). In his left visual field, the patient perceived objects as tilted in a counterclockwise direction (45°-90°, Figure 1). The visual symptoms lasted for 3 hours and then remitted spontaneously. After remission, results from a visual field examination were normal. Findings from a brain computed tomographic scan, an electroencephalogram, and a cerebrospinal fluid examination were within normal limits. Bilateral visual evoked potential recordings showed normal configuration and latency of the P100 wave. Brainstem auditory response was normal on the left side and pathologic on the right, with a first to fifth interwave latency of 6.1 milliseconds (reference range,  $\leq$ 4.6 milliseconds). Results from a perimetric visual field evaluation were normal. Magnetic resonance imaging performed 6 days after the episode demonstrated infarcts of both occipital lobes (Figure 2) occupying an area anterior to the striate cortex in the periphery of Brodmann areas 17 and 18. The infarct on the left was larger. In addition, right cerebellar and middle cerebellar peduncle infarcts were present (Figure 3).

#### CASE 2

A 70-year-old patient with hypertension and diabetes had an episode of true vertigo 1 month before admission for repeated episodes of vertigo and instability of



**Figure 1.** The asymmetrical letters R in the right visual field and L in the left visual field are used to demonstrate the reversal of vision phenomenon. A, Reversal of vision in patient 1. Note the counterclockwise tilting of the left visual field and complete 180° reversal of vision of the right visual field. B, Reversal of vision in patient 4. Complete reversal of vision was interrupted by oblique clockwise vision metamorphopsia. C, Complete reversal of vision in the sagittal plane. E, Complete reversal of vision in the horizontal plane.

gait. Findings from a neurologic examination showed left cerebellar syndrome, left horizontal nystagmus, and a transient absence of the right optokinetic nystagmus. The patient's visual fields were normal. Two hours after the vertiginous attack, he had a complete RVM that lasted for approximately half an hour (Figure 1, C). The RVM momentarily disappeared when the patient saw the flame of a match or his hand moving in front of him. A computed tomographic scan demonstrated bilateral occipital stroke occupying Brodmann area 18 (**Figure 4**).

#### CASE 3

A 79-year-old patient with hypertension was admitted because of acute vertigo. On examination, bilateral cerebellar and pyramidal tract signs were observed but the patient was neurologically stable. Results from a computed tomographic scan were normal. A few hours after admission, the acute episode of vertigo disappeared but the patient developed a sensation of body levitation followed by a counterclockwise rotation of his visual fields. Within 10 minutes, he experienced a complete RVM. He saw people walking on their heads, and the



**Figure 2.** Axial *T*<sub>2</sub>-weighted magnetic resonance imaging scan (repetition time, 2200 milliseconds; echo time, 80 milliseconds) through the occipital regions of patient 1 demonstrating fresh infarcts of both occipital lobes, Brodmann areas 17 and 18, which spare the occipital poles (arrows). The assignment of the lesions was based on the study by Damasio and Damasio.<sup>27</sup>

floor next to his bed appeared to be over his head. To the patient's embarrassment, he made the wrong hand movements when he tried to cover himself with the blanket or to pick up a cup of tea. On examination shortly after the episode of metamorphopsia had disappeared, gaze apraxia with preserved oculocephalic reflexes to all directions was noted. In addition, the patient had optic ataxia without simultanagnosia. When presented with an object in his right upper visual field, he directed his hands to his lower left hemispace. The patient correctly identified the chirality of hands presented to him. His condition stabilized, and results from a repeated neurologic examination the next day were normal aside from bilateral pyramidal tract signs and a mild gait ataxia. The clinical course and the neurologic findings suggested a vertebrobasilar stroke and a transient ischemic attack of the posterior circulation affecting the parietal lobes.

#### CASE 4

A 56-year-old woman had 4 years of recurrent episodes of true vertigo, near syncope, speech disturbances, and inability to control her right hand. These episodes lasted for approximately an hour and sometimes were followed by severe occipital pain or hemicrania. Several episodes were accompanied by a sensation of "heaviness" in her face and dysphagia. During 1 of the episodes, severe vertigo with weakness in the right hand and an inclination of the body to the right



**Figure 3.** Axial *T*<sub>2</sub>-weighted magnetic resonance imaging scan (repetition time, 2200 milliseconds; echo time, 80 milliseconds) through the superior cerebellar regions of patient 1 demonstrating fresh infarct of the right superior cerebellar region and middle cerebellar peduncle. The dark hypodense regions in the right cerebellar and left occipital infarcts represent acute hemorrhage.

were accompanied by complete RVM that lasted 45 minutes and was interrupted by oblique clockwisevision metamorphopsia (Figure 1, B). A repeated episode of oblique vision occurred a few months later and was associated with tilting of vision in the sagittal plane of less than 90°. The patient was admitted for an examination, the results of which revealed blood pressure of 120/80 mm Hg and a regular pulse of 88/min. Findings from a general physical and neurologic examination were normal apart from mild dysmetria on the right upper and lower extremities and upward vertical nystagmus. Results from a laboratory investigation revealed hypothyroidism and hyperlipidemia. The patient's cerebrospinal fluid was normal without oligoclonal bands. There was no serologic evidence of a collagen vascular disease. Findings from an electroencephalogram and audiometry were normal. A brainstem evoked response study showed bilateral prolongation of the first to fifth interwave latency (>6 milliseconds; reference range, <5 milliseconds). T<sub>2</sub>weighted magnetic resonance imaging demonstrated 4 small hyperintense foci in the white matter above and below the tentorium, which were compatible with the patient's age. Since the patient had experienced severe headaches during some of the episodes, a tentative diagnosis of basilar migraine was made and treatment with propranolol hydrochloride was started at 160 mg/d. A dramatic improvement in both the severity and frequency of the episodes ensued. Also of note was the considerable reduction in some of the



Figure 4. Axial computed tomographic scan without contrast demonstrating bilateral hypodense occipital regions occupying Brodmann area 18.<sup>27</sup>

acute attacks following treatment with sumatriptan succinate.

#### CASE 5

A 54-year-old patient was admitted because of recurrent seizures. His medical history included severe ischemic heart disease and angina pectoris, diabetes mellitus with diabetic retinopathy and neuropathy, and peripheral vascular disease. A right hemispheric cerebrovascular accident had caused a left hemihypoesthesia 10 years before his present admission. Two weeks before admission, the patient had episodes of confusion, delusions, and visual hallucinosis, with the experience that somebody was nearby when he was alone. In addition, the patient experienced recurrent twitching of the left side of his face. On the day of his admission, the patient experienced multiple adverse seizures with deviation of the eyes and head to the left and tonic convulsions of the left hand. He reported seeing a stereotypic visual aura: a dark tunnel ending with a bright flash of light. On 3 occasions, the patient had complete RVM postictally, which lasted for approximately 5 to 10 minutes, during which he was fully oriented and not confused (Figure 1, C). Findings from a neurologic examination revealed mild left hemihypoesthesia and hemiparesis, hypometric saccades to the left, disturbed eye pursuit movements to the right, disturbed left optokinetic nystagmus, and transient left hemianopia. A few hours later, the patient's ocular and visual disturbance disappeared, but left hemiparesis and hemihypoesthesia persisted. Results from a computed tomographic scan with contrast material demonstrated right parietal enhancement.

Results from an electroencephalogram performed a few minutes after the disappearance of metamorphopsia showed slow theta and delta background activity with focal epileptiform activity in the right parietal and posterior temporal regions. The patient was treated with continuous intravenous diazepam and the seizures ceased within 48 hours.

#### CASE 6

A 75-year-old patient who was a heavy smoker with chronic obstructive lung disease and ischemic heart disease was involved in a car crash. He sustained polytrauma, including brain concussion and laceration of the scalp. Results from a computed tomographic scan showed a left temporo-occipital linear skull fracture without parenchymal brain injury. The morning after the crash, the patient woke up and saw people around him walking on their heads, his entire visual field turned completely upside down. This phenomenon lasted for approximately half an hour and recurred 48 hours later for a short period. Findings from a neurologic examination after the second episode of metamorphopsia were normal. Neither ocular motility abnormality nor cerebellar dysfunction were observed.

#### COMMENT

Thirty cases of RVM have been reported in the literature.<sup>3-21</sup> A critical analysis of these cases and our own observations allow us to draw certain conclusions regarding the clinical and pathophysiological aspects of this phenomenon.

Reversal of vision was a transient phenomenon in all of our patients. It involved the rotation of the entire visual array without distorting other features of the extrapersonal space. Body sense disturbances with inclination or levitation of the body occurred in 3 patients (cases 1, 3, and 4), but was relatively mild when metamorphopsia occurred. In addition, none of the patients experienced a complete reversal of their bodies in space (ie, no patients had nonvisual perceptions of walking on their heads). Only 5 patients (16%) described in the literature had dramatic alterations in body sense, such as body tilt, a sense of complete body reversal, or head tilt.5,7,8,13,20 All except Halpern's<sup>7</sup> patient had an insult to the brainstem. These findings suggest that RVM is primarily a visual illusion of the extrapersonal space, and that the alteration of the body scheme is the result of a dysfunction of vestibular centers in the brainstem. Alternatively, body scheme disorders could be epiphenomenona. This conclusion is supported by the early experiments of Stratton,<sup>22</sup> who used goggles that reinverted the retinal image. The visual scene was inverted in the coronal (upside down) and horizontal (right to left) planes. Consequently, one's sense of body parts and whole body position was acutely altered.

Transient recovery from metamorphopsia occurred in 1 of our patients (case 2) when he moved his hand in front of him or in response to a familiar visual object with a certain orientation. This is an important observa-

Patient No./ Age, y/Sex	Cause	Duration of RVM (No. of Episodes)	Signs and Symptoms	Associated Disease	Ocular Motility Disorder
1/60/M	Bilateral occipital and cerebellar CVA	3 h	Right cerebellar syndrome	Hypertension and IHD	Right horizontal and rotatory nystagmus
2/70/M	Vertebrobasilar TIA	30 min	Left cerebellar syndrome	Hypertension and DM	Left horizontal nystagmus and decreased right OKN
3/79/M	Brainstem CVA	10 min	Optic ataxia and gaze apraxia	Hypertension	Gaze apraxia
4/56/F	Basilar migraine	45 min (3)	Right clumsy hand	Hyperlipidemia and hypothyroidism	Vertical nystagmus
5/54/M	Complex partial seizure	5-10 min (3)	Transient left hemianopia and left hemihypoesthesia	Hyperlipidemia, DM, and IHD	Disturbed pursuit eye movements
6/75/M	Brain concussion and left occipitotemporal fracture	30 min (2)	NA	NA	NA

\* CVA indicates cerebrovascular accident; IHD, ischemic heart disease; TIA, transient ischemic attack; DM, diabetes mellitus; OKN, optokinetic nystagmus; and NA, not applicable.

tion because it shows that certain stimuli can correct and reinvert the visual scene. A functional dissociation exists in the human brain of near and far visual space perception. The movement of a hand in the peripersonal space might activate neuronal networks with pericutaneous receptive fields.<sup>23</sup> These networks, which process information from the peripersonal space, might correct RVM, which is primarily brought about by lesions to networks related to far space perception. Four patients described in the literature had transient recoveries from RVM usually in response to eye closure or changes in body position.<sup>5,6,14,15</sup> Polysensory neurons that respond to visual stimuli and are selective to the spatial characteristics of hand movements24 and neurons that respond to visual and somatosensory stimuli in area 7b<sup>25</sup> might also be involved in the process of recovery. Stratton<sup>22</sup> showed that after a few days of wearing goggles that inverted the retinal image, subjects could realign and reinvert the visual scene without taking off the goggles. This demonstrates the enormous plasticity of the specific neuronal networks involved in spatial frame perception.

None of the patients in our series had visual field defects at the time of the RVM. Only 1 patient (case 5) had transient hemianopsia before metamorphopsia occurred, which is consistent with an early observation of Critchley,<sup>26</sup> who pointed out that metamorphopsia occurs when lesions approximate the visual cortex and the geniculostriate radiations but do not actually involve them.

It was difficult and at times impossible to determine the geometrical relationship of rotations and reflections of the visual world to chirality in our patients and those described in the literature. Metamorphopsia was reported along each of the principal orthogonals, although it was rare in the sagittal and horizontal planes.<sup>5,7,11,15</sup> The most common distortion is a rotation along the coronal plane, which flips the top and bottom and the left side of objects to the right and vice versa, but keeps a left hand looking like a left hand appearing in the contralateral part of the space (Figure 1, C). In contrast, an inversion along the sagittal plane would result in a top-bottom mirror reflection, making a left hand look like a right hand. Although chirality is inverted, a left hand would stay in the left visual field (Figure 1, D). One patient (case 4) described an incomplete (<180°) inversion of the visual field in the sagittal plane. Horizontal-plane metamorphopsia would not bring about a top-bottom RVM but a right-left reversal, such that a right hand would appear to be a left hand in the wrong (contralateral) part of space (Figure 1, E).

Three patients had oblique vision, 1 with clockwise and 2 with counterclockwise rotation of the visual field (cases 1 and 4 [Figure 1, A and B], and 3). This phenomenon accompanying RVM was reported in 5 cases in the literature.<sup>5,7,8,10,12</sup> In these cases, the visual scene was tilted less than 180°. Case 1 in our study is particularly interesting in this regard. This patient had asymmetrical bilateral stroke in Brodmann areas 17 and 18. Consequently, the patient experienced both RVM and counterclockwise tilting of vision simultaneously in different parts of his visual field. This might indicate that both phenomena are interrelated and that a separate central integrator of visuospatial orientation exists in each hemispheric visual cortex, possibly within the posterior parietal and occipital areas.

One patient (case 3) had RVM in the coronal plane with a reaching disorder. Apparently, he executed contralateral reaching movements that correspond to Figure 1, C. He did not have self right to left confusion, and he was able to identify the chirality of hands presented to him. We did not observe right to left confusion in our patients, suggesting that this phenomenon is not related to RVM.

In all patients, vomiting, dizziness, and feelings of malaise were reported. Ocular motility disorder was found in 5 of 6 of our patients (**Table**). This condition has been recognized in many other patients, <sup>5,8,14,19-21</sup> but its pathophysiological significance remains unclear.

Five of our patients (cases 1-3, 5, and 6) had signs and/or results from imaging studies or electrophysiological studies consistent with occipital or parietooccipital lesions. A stroke in the occipital region, Brodmann area 18, was found in 2 patients (cases 1 and 2, according to the Damasio plates<sup>27</sup>). Brainstem auditory

evoked response was unilaterally or bilaterally abnormal in 2 patients (cases 1 and 4). Four patients had vertigo, cerebellar signs, or even brainstem cerebrovascular accident prior to metamorphopsia (cases 1-4). One patient (case 3) had signs of Balint syndrome. Although most of our patients had a cortical parieto-occipital syndrome, such conditions were not reflected in previous cases. When the site of the insult was documented in patients with RVM, it was found to involve 2 main regions, the parieto-occipital region and the brainstem.<sup>6</sup> If we include our patients, 7 (16%) of 36 patients (30 previous cases and our own 6 patients) had a brainstem lesion.<sup>17,19,20,22</sup> Three patients had a medullary insult,<sup>8,13</sup> 4 had a cerebellar lesion,<sup>12,14,19,20</sup> and 7 had a parietooccipital lesion.7,9,11,15 One patient had an unusual frontal lesion<sup>6</sup> (in addition to the case of Halpern<sup>7</sup> with cysticercosis involving mostly the parieto-occipital region but also the frontal lobes). One patient (case 3) had transient vertebrobasilar ischemic attacks with brainstem and occipital lobe syndrome.<sup>21</sup>

Thus, 15 (41%) of 36 patients described in the literature had infratentorial lesions, whereas only 8 patients (22%) had parieto-occipital lesions. However, in many of the cases with brainstem syndromes, the cause was either a vertebrobasilar transient ischemic attack or a cerebrovascular accident. It is plausible that the actual phenomenon of RVM in patients with brainstem syndromes occurred when ischemia affected the parieto-occipital area rather than brainstem structures. Most of the cases of RVM were reported before the era of modern imaging. Therefore, it is conceivable that some of the patients with brainstem syndromes had concomitant parieto-occipital lesions.

What are the mechanisms underlying RVM? The spatial representation of visual stimuli in the central nervous system is formed by combining information from various modalities, both retinal and extraretinal. To create a valid representation of space, a series of coordinate frame transformations is performed. Retinal inflow is combined with ocular position information to produce head-centered representation.<sup>28-30</sup> To represent body-centered coordinates, the 7a parietal cortex and the intraparietal area have neurons that respond to the eye position as well as the head position.<sup>30</sup> The relationship between the relevant representation of visual stimuli and RVM is based on world-centered coordinates (allocentric). Information about the position of the head derives from otolith and vestibular inputs together with proprioceptive information from neck muscles. These signals, when combined with eye and retinal position information, can encode locations in world-based coordinates.<sup>30,31</sup> Experiments in rats typified "direction tuned" cells, the activity of which were modulated by the orientation of the whole body. These cells in the posterior parietal region are multimodal cells. They are activated by both visual and integral vestibular signals to form the representation of space.<sup>30,32</sup> We know that this orientation system is widely distributed in the central nervous system.<sup>30</sup> This might explain the various locations of insults causing RVM. It is clear that both retinal and extraretinal disturbances can bring about a mismatch of information that would lead to

RVM. Any transient inconsistent correspondence of vestibular information or lack of information due to a brainstem lesion would potentially lead to RVM. In particular, a disturbance of the central otolith pathway that would deprive higher brain centers of necessary information about the head position in the roll plane, which is the plane relevant to the phenomenon of RVM in most patients. Distinct areas in the brainstem that mediate vestibular tone in the horizontal, vertical, and roll planes have recently been discovered.<sup>33</sup> Dysfunction of these specific regions in the brainstem might lead to vestibular tone imbalance, and that in turn would induce RVM along 1 or more of the principal orthogonals.

In conclusion, failure to perceive space in an allocentric coordinate frame, particularly in the coronal roll plane, is potentially the critical event underlying RVM. Reversal of vision is always a transient visual illusion caused by either brainstem or parieto-occipital lesions. It is a pure disorder of far space perception that should be clearly differentiated from certain body scheme disorders and other types of visual metamorphopsia. The polymodal nature of the neuronal network that governs the perception of allocentric coordinates and their widespread distribution in the central nervous system might allow for a rapid recovery from RVM when correct and new information is processed.

#### Accepted for publication March 23, 1998.

This work was supported in part by the Sol Irwin Juni Memorial Fund and the Hilda Katz Blaustein Fund, Jerusalem, Israel.

We would like to thank Jane Loitman, MD, for critically reviewing the manuscript, and Izchak River, MS, for helping with the translation of the non-English articles.

Reprints: Yaron River, MD, Department of Neurology, Hadassah University Hospital, Hebrew University-Hadassah Medical School, PO Box 12000, Jerusalem 91120, Israel.

#### REFERENCES

- Walsh FB, Hoyt WF. *Clinical Neuro-ophthalmology*. Vol 1. Baltimore, Md: Williams & Wilkins; 1969:753-754.
- Brown JW. Hallucinations: imagery and microstructure of perception. In: Vinken PJ, Bruyn GW, Klawans HL, eds. *Handbook of Clinical Neurology*. Vol 45. Amsterdam, the Netherlands: Elsevier Science Publishers; 1984:351-372.
- Winslow F. On Obscure Diseases of the Brain and Disorders of the Mind. 4th ed. London, England: John Churchill & Sons; 1868.
- Pearson G. Inverted position in children's drawings: report of two cases. J Nerv Ment Dis. 1928;68:445-449.
- Klopp HW. Uber Umgekert und Verkehrtschen. Dtsch Z Nervenheilkd. 1951;165: 231-260.
- Solms M, Kaplan-Solms K, Saling M, Miller P. Inverted vision after frontal lobe disease. *Cortex*. 1988;24:499-509.
- Halpern F. Kasuitischer Beitrag zur Frage des Verkehrtsehens. Z Gesamten Neurol Psychiatrie. 1930;126:246-252.
- Hornsten G. Constant error of visual egocentric orientation in patients with acute vestibular disorder. *Brain.* 1979;102:685-700.
- Phelps E. Die diagnostische Verwerthung der Schalleitungsveranderungen des Schadels bei intra-und extracraniellen Herderkrankungen. Arch Psychiatrie. 1908; 43:577-632.
- Pick A. Ueber eine besondere Form von Orientierungsstorung und deren Vorkommen bei Geisteskranken. Dtsch Med Wochenschr. 1908;47:2014-2017.

ARCH NEUROL/VOL 55, OCT 1998 1367

- Gerstman J. Uber eine eigenartige Orientierungsstorung im Raum bei zerebraler Erkrankung. Wien Med Wochenschr. 1926;76:817-818.
- Wilder J. Über Schief-und Verkehrtsehen. Dtsch Z Nervenheilkd. 1928;104:222-256.
- Luers T, Potzel O. Uber "Verkehrtsehen" nach insult: beitrag zu den zerebral bedingten Sehestorungen. Wien Klin Wochenschr. 1941;54:626-628.
- 14. Potzel O. Uber Verkehrtsehen. Z Gesamten Neurol Psychiatrie. 1943;176: 780-792.
- 15. Penta P. Due casi di visione capovolta. Cerevello. 1949;25:377-389.
- Bender MB, Rudolph SH, Stacy CB. The neurology of the visual and oculomotor systems. In: Baker AB, Joynt RJ, eds. *Clinical Neurology*. Philadelphia, Pa: Harper & Row; 1985;1:42-43.
- Oppenheim H. Lehrbuch der Nervenkrankheiten. Berlin, Germany: S Karger AG; 1913:815-816.
- Stracchiari A, Guarinc M, Ciucci G, Pazzaglia P. Acute upside down reversal of vision in vertebrobasilar ischaemia. *J Neurol Neurosurg Psychiatry*. 1993;56: 423-429.
- Steiner I, Shahin R, Melamed E. Acute "upside down" reversal of vision in transient vertebro-basilar ischemia. *Neurology*. 1987;37:1685-1686.
- Charles N, Froment C, Rode G, et al. Vertigo and upside down reversal of vision due to an infarct in the territory of the medial branch of the posterior inferior cerebellar artery caused by dissection of a vertebral artery. *J Neurol Neurosurg Psychiatry*. 1992;55:188-189.
- Mehler MF. Complete visual inversion in vertebrobasilar ischaemic disease. J Neurol Neurosurg Psychiatry. 1988;51:1236-1237.
- Stratton G. Vision without inversion of the retinal image. *Psychol Rev.* 1897;4: 341-360, 463-481.

- Rizzolatti G, Scandolara C, Matelli M, Gentilucci M. Afferent properties of periarcuate neurons in macaque monkeys. *Behav Brain Res.* 1981;2:147-163.
- Taira M, Mine S, Gergopoulos AP, Murata A, Sakata H. Parietal cortex neurons of the monkey related to the visual guidance of hand movements. *Exp Brain Res.* 1990;83:29-36.
- Leinonen L, Nyman G. Functional properties of cells in anterolateral part of area 7, associative face area, of awake monkeys. *Exp Brain Res.* 1979;34: 321-333.
- Critchley M. Metamorphopsia of central origin. *Trans Ophthalmol U K.* 1949;69: 111-121.
- Damasio H, Damasio AR. Lesion Analysis in Neuropsychology. New York, NY: Oxford University Press Inc; 1989.
- Galletti C, Battaglini PP. Gaze-dependent visual neurons in area V3A of monkey prestriate cortex. J Neurosci. 1989;9:1112-1125.
- Robinson DL, McClurkin JW, Kertzman C. Orbital position and eye movement influences on visual responses in the pulvinar nuclei of the behaving macaque. *Exp Brain Res.* 1990;82:235-246.
- Anderson RA, Essick GK, Siegel RM. Encoding of spatial location by posterior parietal neurons. *Science*. 1985;230:456-458.
- Horn G, Hill RM. Modifications of receptive fields of cells in the visual cortex occurring spontaneously and associated with bodily tilt. *Nature*. 1969;21: 186-188.
- Sakata H, Kusunoki M. Organization of space perception: neural representation of three-dimensional space in the posterior parietal cortex. *Curr Opin Neurobiol.* 1992;2:170-174.
- Brandt T, Deiterich M. Vestibular syndromes in the roll plane: topographic diagnosis from brainstem to cortex. Ann Neurol. 1994;36:337-347.

©1998 American Medical Association. All rights reserved.