Complete visual inversion in vertebrobasilar ischaemic disease

Sir: The phenomenon of transient, complete 180° inversion of the visual image is exceedingly rare. It has been seen in association with Wallenberg's syndrome,1 vertebrobasilar transient ischaemic attacks (TIAs),2,3 measles encephalitis,4 toxic ingestion5 and severe head injury.6 In addition, behavioural evidence of a more permanent developmental disability with complete visual inversion has been described in children and adults.6 The mechanisms and anatomical sites that mediate this perceptual aberration in specific disease and developmental states are unknown. I describe three patients with vertebrobasilar transient ischaemic attacks and episodes of complete visual inversion. They share several distinct clinicopathological features.

A 73 year old right-handed man was hospitalised for recurrent syncopal attacks. In the past 3 months, there had been four episodes of transient loss of consciousness, one associated with prodromal nausea and light-headedness. In addition, several ill-defined episodes of gait instability and vertigo had occurred. During one of these "spells", which lasted for about 45 minutes, images suddenly appeared "completely upside down". People appeared to enter the room from the top walls and a carpet seemed magically suspended from the ceiling. The entire visual field was involved and other visuospatial and perceptual features were preserved. The patient had a long history of severe alcohol abuse and untreated hypertension. However, he had stopped drinking 3 years previously after an episode of bleeding from oesophageal varices. On the day of admission, he had experienced a typical "fainting spell" and subsequent transient slurred speech, unsteady gait and oscillopsia. Neurological examination 3 hours later revealed intact mentation, slight scanning speech, gaze-evoked nystagmus, mild bilateral dysmetria of the arms and widened base on gait testing. Brain computed tomography (CT), electroencephalogram (EEG), audiometric studies and brainstem auditory evoked responses (BAERs) during the hospital course were within normal limits. Carotid doppler echo was consistent with an alcoholic cardiomyopathy. Holter monitor showed multiple atrial premature contractions. During the hospitalisation, he had two episodes of gait instability and impaired arm coordination. Each occurrence was heralded by complete, sudden visual inversion. At the apex of one "spell", neurological examination revealed the following: preserved mentation and neuro-ophthalmological evaluation, mild ocular and bilateral arm dysmetria, impaired check with excessive rebound and widened base with ataxic gait. The patient was started on long-term anti-coagulation with cessation of symptoms.

A 67 year old right-handed woman was evaluated for "visual disorientation". For the past 2 days she had experienced six episodes of complete visual inversion, each lasting 10–15 minutes and preventing her from proper spatial navigation in her house or in the street. There were no associated symptoms. She had a history of atherosclerotic cardiovascular disease with chronic atrial fibrillation and moderate hypertension, treated initially with propranolol and later with captopril. Neurological examination was performed one hour after her last attack. She exhibited intact mentation, abnormal optokinetic nystagmus with exaggerated slow phase velocity to the right, irregular ocular tracking, mild skew deviation, impaired rapid alternating movements of both arms, and poor tandem gait. EEG, brain CT and MRI, audiometric studies and serial electrocardiograms (ECGs) were unrevealing. No additional therapeutic intervention was attempted. One year later, she had experienced only two brief recurrences of the visual inversions and no further neurological symptoms. Her neurological examination, at this time was completely normal.

A 47 year old left-handed man was hospitalised for vertebrobasilar transient ischaemic attacks. Over the past 2 days there had been three stereotyped episodes of transient, complete visual loss, gait unsteadiness and subsequent bilateral intention tremor. The patient had no prior medical history but an extensive family history of premature deaths from myocardial infarctions. Neurological examination on admission revealed normal mentation, corrected visual acuity of 20/50 bilaterally, gaze-evoked nystagmus to the left side, mild bilateral ocular dysmetria, bilateral intention tremor and impaired distal arm coordination, bilateral impaired check in the upper extremities, widened base with ataxic gait and generalised hypotonia. Brain CT, EEG, and BAERs during the hospital course were unrevealing. ECG showed evidence of an evolving subendocardial myocardial infarction. Holter monitor showed frequent atrial premature contractions and three brief runs of atrial fibrillation. During the hospital course he was awakened one morning by vertiginous sensations, and noted that the hospital room was completely upside down. On attempting to walk he noted that his legs were numb and unsteady. All visual features appeared intact but spatial forms were completely inverted and visual movements occurred in a direction opposite to normal in the vertical plane. Neurological examination was unchanged. Four-vessel cerebral angiography performed 3 weeks later was entirely normal as were repeat neuroimaging studies. During the next 6 months there was no recurrence of symptoms and repeat Holter monitor showed only occasional atrial premature contractions. Neurological examination at one year was completely normal.

Errors of visual egocentric localisation which induce tilts in environmental vision have been seen following ocular muscle or conjugate gaze pareses.7–10 With unilateral vestibular lesions there is constant lateralisation of the visual vertical in the absence of organic muscular pareses.10 The perceptual illusion is related to the vestibular imbalance which results in an altered resting position of the eyes. The latent illusion is made manifest only after elimination of the visual reference frame, and lateralisation is proportional to the extent of resting eye deviation.10 Marked deviations from the visual vertical towards the side of the lesion have been described with unilateral intracerebral haemorrhage processes.11 It is also known that vestibular reflexes elicited by a variety of stimuli at the labyrinthine recep-

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tors are modified and regulated by cerebellar connections. In Wallenberg's syndrome, both 180° visual inversions, and associated 90° inversions, and less dramatic defective perceptions of the vertical axes occur. A number of observations suggest that these two pathological processes may be nosologically distinct. One astute patient noted that, during a transient episode of visual inversion, his appendicular movements and general body habitus appeared normal, as if superimposed on an inverted landscape. This suggests a differential interface between the pathological substrate mediating complete visual inversion and neural mechanisms which govern the elaboration of egocentric, allocentric and extrapersonal space.

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MARK F MEHLER
Saul R Korey Department of Neurology,
Albert Einstein College of Medicine,
1300 Morris Park Avenue,
Bronx, New York 10461, USA

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Aneurysm of the cervical internal carotid artery following chiropractic manipulation

Sir: Aneurysms of the extracranial portion of the carotid artery are uncommon. Atherosclerosis and trauma are now considered to be the most common causes. Traumatic aneurysms of the carotid artery in the neck may be due to either open or closed injury. A case of internal carotid artery aneurysm at the origin following neck manipulation is reported and is to our knowledge the first reported account of such case.

A 40 year old male was admitted for a swelling of the left side of the neck, hoarseness of voice and mild drooping of the left eyelid with loss of sweating on the left side of face of one month's duration. He developed these symptoms within 1-2 hours of neck manipulation by a barber for the relief of pain in the neck. The neck manipulation included sudden jerky rotary movements to either side as well as extension and flexion.

On examination he was normotensive, all peripheral pulses were felt equally, there was a pulsatile globular swelling (3 cm x 2 cm) on the left side of the neck below the jaw. It was noncompressable and no bruit could be heard. There was Horner's syndrome on left side and hoarseness of voice, without vocal cord paralysis.

Syphilitic serology was negative. Lipid profile was normal. Carotid angiography showed a lobulated aneurysm at the origin of the left internal carotid artery (fig). The right carotid was normal. The left carotid bifurcation was explored 5 days after angiography. A thin walled highly friable 3 cm x 2 cm aneurysm of the distal carotid and proximal internal carotid was found. A clot could be felt within the sac of the aneurysm. A common carotid to internal carotid shunt was inserted before clamps were placed below and above the aneurysm. On opening of the aneurysm, a fresh thrombus was found.
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M F Mehler

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