Acute upside down reversal of vision in verteobasilar ischaemia

Acute upside down reversal of vision is an uncommon and little known phenomenon consisting of transient complete 180 degree inversion of the visual image. The pathogenesis and the anatomical sites of this dysfunction are unknown. Lesions involving cortical areas, mainly the parieto-occipital region, or the vestibulocerebellar system have occasionally been documented.\(^1\)

We observed two patients who experienced this bizarre visual illusion, both revealing features of verteobasilar ischaemia.

Patient 1, a 69 year old woman, was admitted because two weeks earlier she had experienced sudden malaise, sweating, nausea, vomiting, right occipital headache, followed by a 180 degree vertical inversion of the visual image, lasting about 20 minutes. Two similar episodes had occurred the day before admission. On admission the patient was alert, cooperative and had normal neurological examination. Blood parameters, urine, chest radiograph and ECG proved normal. Cervical radiographs revealed osteoarthrosis with osteophytes and narrowing of disc space C6-C7. EEG was normal. Brainstem auditory evoked potentials (BAER) revealed increased latency of V wave on right stimulation. Cerebral CT and MRI (figure) showed an ischaemic-like lesion, 2 cm diameter, in the right cerebellar hemisphere in the territory of the medial branch of the posterior inferior cerebellar artery (PICA), without mass effect. Moderate periventricular white matter abnormalities coexisted. Four vessel cerebral angiography revealed a right vertebral artery stenosis (50%) and two small arteriovenous malformations on the course of the right ascending cervical artery; a decreased flow in the basal artery was noted. Ticlopidine 250mg daily was given and the patient was discharged. No further attacks or other neurological disturbances occurred during the next two years.

Case 2, a 52 year old woman, with a 40 year history of bilateral chronic otitis with residual deafness, had recent recurrent episodes of sudden swelling, right occipital headache, dizziness, sometimes followed by a transient loss of consciousness. The whole episode usually lasted about 30–40 minutes. Frequently, at the height of dizziness, the patient experienced 180 degree vertical visual inversion of images. These episodes occurred monthly. On admission, the neurological examination was normal. Rare, isolated, left-sided jerks of horizontal nystagmus revealed by ENG. A 20 mmHg difference between right and left brachial arterial pressure (right > left) was noted. Ultrasound vascular investigations (Doppler cortico-vertebral echomography and cerebral transcranial Doppler) revealed a left subclavian artery stenosis with a steal syndrome. Cerebral SPECT, CT and MRI proved normal. BAER was unavailable due to the peripheral loss caused by chronic otitis. Cerebral angiography was refused. Fluorinize 10mg daily was given and the patient was discharged with a warning to avoid strenuous physical activities, especially those involving upper limbs and neck. No further episodes were reported in the subsequent six months.

These two women presented episodes of vertically inverted vision—upside down phenomena—associated with clinical signs and symptoms of verteobasilar insufficiency. Both reported transient visual inversion of 180 degrees, which was bilateral, of sudden onset and lacking subjective impression of movement (or reinversion). In the first patient, neuroimaging revealed a right hemispheric cerebellar infarction. In the second, a verteobasilar failure due to a left subclavian stenosis was detected. The pathogenetic mechanism underlying upside down visual inversion is unknown. Since the visual images enter the retina inverted, it may be assumed that the upside down phenomenon is associated with failure of the mechanisms mediating reinversion, even though the anatomical structures involved are unknown. In earlier observations,\(^2\) parietal and/or occipital lesions were sometimes associated with similar cortical origin of the dysfunction, probably affecting the integrative control of spatial vision. More recent cases,\(^3\)\(^–\)\(^6\) documented with neuroimaging techniques, revealed an association with vestibular/cerebellar lesions, that is, verteobasilar TIAs, Wallenberg’s syndrome and also cerebellar infarct in two cases.\(^7\) In our patients, the relationship between the horizontal visual inversion and the signs and symptoms of verteobasilar insufficiency, without evidence of cerebral damage, supports the idea that a transient inactivation of infratentorial structures may cause this visual phenomenon. Besides the integrity of the visual system, space visual perception needs a flow of extraretinal information, mediated by the vestibular and cerebellar systems.\(^8\)\(^–\)\(^10\) It has been suggested that damage to such structures may cause tilt and complete inversion of the visual space.\(^7\) The upside down phenomenon may occur following dysfunctions at various levels of the vestibulocerebellar-ocular system mediating the stabilization of the visual function so that cortical involvement is not indispensable.

Subcortical environmental reduplication: SPECT findings in a patient with a right thalamocapsular haemorrhage

Recently Nighoghossian et al\(^1\) reported the case of a patient with a previous history of a left fronto-basal haemorrhage, who developed environmental reduplication following an infarction of the retrolenticular portion of the right internal capsule. SPECT revealed right fronto-parietal cortical hypoperfusion. A similar disorientating phenomenon was described previously in a patient with a right thalamic haemorrhage, but its functional correlate using SPECT was not studied.\(^1\) We describe the neuroimaging and cognitive functioning of a case of environmental reduplication associated with a right thalamocapsular haemorrhage.

A 71 year old amnestic man suddenly developed a left-sided weakness and mild dysarthria. He had had hypertension but no history of previous cerebrovascular events. Neurological examination revealed a dense left hemiplegia, and a left sensory loss affecting all modalities above the knee. The modality of pain was preserved, as was temperature and vibration, and there was no evidence of visual or auditory extinction on double simultaneous stimulation. He showed left hemispatial neglect on drawing, and on a letter cancellation task he only crossed targets on the right side of the paper. He did not deny his left hemiplegia, but he had a tendency to attribute it to previous “chest problems”. He reported a feeling of nonbelonging to his paralysed left arm, and also said that he had three left legs and a strange left arm crossed over his chest. The patient said that he could walk almost normally and repeatedly tried to walk under his left hemiplegia. He was alert and oriented to time and person, but not to place. While he...
classified. Although he remained in the Memory was at 10%).

percentile scores on tests 4-6), Trail-making impairment on gamma camera.

were normal (99 points; his scores on immediate history recall and associated learning were average though his performance on the visual reproduction subset was bel-camera. Focal blood flow were analysed semiquantitatively in twelve circular regions of interest which were placed over the cortical mantle in three successive slices. Asymmetry indices (AI) for each lobe were calculated using the following formula: (R - L)/(R + L) × 200. A marked decrease of perfusion was observed in the right thalamus and basal ganglia as well as in the left cerebellum. Hypoperfusion was also noted in widespread cortical regions of the right hemisphere affecting mainly the frontal lobe (AI = −29-6), and to a lesser extent the parietal (AI = −10-8) and temporal (AI = −10-) lobes (negative AI values indicated higher hyperactivity relative to the right sided activity).

The assessment of neuropsychological functions in our case of subcortical environmental reduplication revealed more permissive deficits than those observed in the patient reported by Nighoghossian et al., but similar to those of previous cases showing evidence of environmental reduplication and cortical involvement. Moreover, the combination of deficits on nonverbal memory, awareness, visuospatial skills and reasoning abilities supports the view that environmental reduplication revealed more permissive multifactorial delusional misidentification syndrome.4

Some previous reports emphasised the association of admission to hospital, the superimposition of bilateral frontal-lobe and right hemisphere cortical involvement, while others suggested that unilateral (right) lesions of either the fronto-parietal or parieto-temporal cortices are sufficient to cause it.5,6 In our patient, right thalamocapsular damage may have induced functional depression of various distant but anatomically connected cortical areas. Data from SPECT, however, revealed secondary cortical desactivation affecting mainly the right frontal cortex. In this context, we suggest that functional desactivation of the right cortical mantle, in addition to thalamocap- sular activity, may underlie reduplication and its associated neuropsychological deficits. In addition, given that environmental reduplication probably requires preexisting brain pathology (for example, cortical atrophy) besides the specific sites of brain damage in the right hemi-

sphere,1 the presence of leuoaraisis in the case by Nighoghossian et al. and in our own patient might be another risk factor for developing it after acute stroke.7

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1 Nighoghossian N, Trouillas P, Vighetto A, et al. Spatial delirium following a right subcor-

2 Leiguarda RC. Environmental reduplication associated with a right thalamic haemor-


5 Kapur N, Turner A, King C. Reduplicative paramnesia: possible anatomical and neu-

Evidence for presynaptic inhibition on trigeminal primary afferent fibres in humans

In a recent study we have shown that a cond-
ing electrical stimulus applied to the trigeminal primary afferent fibres of intensity below the reflex threshold (Th) evoked by presynaptic inhibition in the perceptive Th) produces unexpected changes of test trigemino-facial reflex responses.1 In particular, for the R2 response, there is a monotonous depression starting at 20–30 ms of interstimulus interval, reaching a maximal value at 50–100 ms and recovering within 300–400 ms (figure 1). Based on the similarity of the time-course of the R2 response and of the time-course of the trigeminal primary afferent depolarisation described in the cat,2 we propose that presynaptic inhibition from low-Th trigeminal afferent fibres was the primary factor contributing to the depression of the test response. We describe experimental evidence to support this hypothesis.

Two complementary experimental designs, approved by the Local Ethical Committee, have been employed: 1) the time-course of the R2 inhibition was compared, in the same subject, with that of the soleus Ia presynaptic inhibition; 2) the effect of intravenous administration of thyrotopin releasing hormone (TRH), a sub-

stance shown to increase presynaptic inhibition in humans,3 has been tested in parallel on both spinal Ia presynaptic inhibi-
tion and R2 reflex responses. In addition, spinal Ia presynaptic inhibition and trigeminal R2 inhibition were studied in a patient affected by chronic progressive spino-bulbar spasticity, a rare disease presenting with spastic paralysis of facial, upper extremity and lower limb muscles, due to a progressive and parallel involvement of corticobulbar and cortico-spinal projections.4 Many patients with spasticity show a reduced or absent reflex.
Subcortical environmental reduplication: SPECT findings in a patient with a right thalamocapsular haemorrhage.
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