Acute upside down reversal of vision in vertebrobasilar 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 cervical arteries, mainly the parieto-occipital region, or the vertebrobasilar system have occasionally been documented. 

We observed two patients who experienced this bizarre visual illusion, both revealing features of vertebrobasilar 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 well-oriented. The neurological examination was normal. In particular, no neuroophthalmological abnormalities were found on clinical 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 basilar 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 sweating, an episode of 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 noticed a 180 degrees vertical visual inversion of images. These episodes occurred monthly. On admission, the neurological examination was normal. Rare, isolated, left-sided jerks of horizontal nystagmus were revealed by ENG. A 20 mmHg difference between right and left brachial arterial pressure (right > left) was noted. Ultrasound vascular investigations (Doppler cortico-vertebral echography 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 presence of bilateral chronic otitis. Cerebral angiography was refused. Flunarizine 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 vertebrobasilar insufficiency. Both reported transient visual inversion of 180 degrees, which was bilateral, of sudden onset and lacking subjective impression of movement (or vertical disorientation). In the first patient, neuroimaging revealed a right hemispheric cerebellar infarction. In the second, a vertebrobasilar 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—associated with transient failure of the mechanisms mediating reinnervation, even though the anatomical structures involved are unknown. In earlier observations, parietal and/or occipital lesions were sometimes associated with cortical origin of the dysfunction, probably affecting the integrative control of spatial vision. More recent cases, documented with neuroimaging techniques, revealed an association with vestibular/cerebellar lesions, that is, vertebrobasilar TIAs, Wallenberg's syndrome and also cerebellar infarct in two cases. In our patients, the relationship between the horizontal visual inversion and the signs and symptoms of vertebrobasilar 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. It has been suggested that damage to such structures may cause tilt and complete inversion of the visual space. The upside down phenomenon may occur following dysfunctions at various levels of the vestibulo-cerebellar-ocular system mediating the stabilisation of the visual function so that cortical involvement is not indispensable.

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

Recently Nighoghossian et al 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 dismutation syndrome was described previously in a patient with a right thalamic haemorrhage, but its functional correlate using SPECT was not studied. We describe the neuroimaging and cognitive functioning of a case of environmental reduplication associated with a right thalamicocapsular haemorrhage.

A 71 year old ambidextrous 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. His sensorium was clear, 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 nonbeing in 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 unaided despite his left hemiplegia. He was alert and oriented to time and person, but not to place. While he

Figure  Axial MR T2-weighted image showing a high signal area in the territory of the medial branch of the right PICA.
named. Although he remained in the city he was located in neighbouring cities (Torremolinos, Fuengirola, Marbella).

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Evidence for presynaptic inhibition on trinigeminal primary afferent fibres in humans

In a recent study we have shown that a conditioning electrical stimulus applied to the trigeminal afferent fibres of intensity below the reflex threshold (Th) which is 0-95 times the perceived Th of the function of the left peripheral branch is also reported. There is a clear strict similarity of the curves of the R2 and soleus H reflex inhibition. Identical findings have been observed in two other normal subjects. By exploring the upper curve in fig 1 it is apparent that the long-lasting inhibition of the soleus H reflex is preceded by an early short-lasting facilitation (see also the upper curve in fig 2). This early facilitation is due to mechanical spreading of the presynaptic inhibition to spinous spindles causing homonymous Ia facilitation in the soleus motor neurons. This explains why this early facilitation is lacking in the R2 reflex curve (lower curve in fig 1).

The histograms on the right in fig 1 show the enhancement of the R2 and H reflex depressions after acute intravenous administration of TRH. It has been demonstrated that a single subcutaneous injection of high-dose TRH (1-2-2-5 mg/kg) produces dramatic and long-lasting (1-2 hours) increase of the soleus H reflex after TRH in patients with amyotrophic lateral sclerosis. We have observed a similar but short-lasting (10-20 minute) enhancement of the soleus Ia presynaptic inhibition in normal 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 thyrotropin releasing hormone (TRH), a substance shown to increase presynaptic inhibition in humans, 3) has been tested in parallel on both spinal Ia presynaptic inhibition and R2 reflex inhibition. Thus, spinal Ia presynaptic inhibition and trigeminal R2 inhibition were studied in a patient affected by chronic progressive spinobulbar spasticity, a rare disease presenting with spastic paralysis of facial muscles and lower limb muscles, due to a progressive and parallel involvement of corticobulbar and cortico-spinal projections. Many patients with spasticity show a reduced or absent R2 reflex.
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