Minor hemisphere syndrome following left hemispheric lesion in a right handed patient

Some degree of unawareness of hemiplegia occurs in about one third of cases of right hemisphere patients with left hemiplegia. Anosognosia for hemiplegia may be associated with left hemisomatognosia. Such patients behave as if the left half of their body was no longer part of themselves. In contrast to the relative frequency of such phenomena following damage to the right hemisphere, anosognosia and hemisomatognosia have rarely been reported following left hemisphere lesions. None of the reported cases has concerned authentic right-handed patients and the degree of language impairment has often been unknown.

We describe a case of a strongly right handed patient with anosomatognosia and anosognosia for right hemiplegia. She exhibited other deficits relating to the so-called minor hemisphere syndrome, and had no language disorders.

On the thirteenth day after aortic valve replacement for aortic regurgitation, the patient, a 68 year old right handed woman, developed right hemiplegia. On the fourth day following onset, neurological examination showed massive right motor deficit affecting the face, the arm and the leg. Right plantar reflex was extensor, there was severe hypotonia and tactile extinction on the right side of the body, and right homonymous hemianopia on confrontation. Language and praxis were normal on bedside evaluation.

The patient's motor impairment was strongly tended to keep her head and eyes turned to the left, even on verbal stimulation from the right. CT scan at 10 days post onset revealed a left hemispheric infarct involving the territory of the middle cerebral artery, both deep and superficial, and the territory of the anterior choroidal artery.

The following observations were gathered during the first three weeks following the stroke, during which period the neurological condition of the patient remained essentially unchanged. Right sided visual neglect was seen on the dot cancellation test: she failed to cancel 7/9 dots in the right half of the test sheet, although she did not miss any of the eight dots in the left half. The patient was presented with a list of 40 pairs of items (10 pairs of digit names, 20 pairs of object names, 10 pairs of sentence fragments). She showed clearcut right auditory neglect: she correctly reported all of the 40 pairs presented to her left ear, but none of those presented to her right ear.

She was largely unaware of her hemiplegia. When asked if her right hand was strong, could she sew, or knits, she answered that she could. When asked if she could move her right leg, she answered positively and moved her left leg discarding the hand. Sometimes, the patient admitted that she needed some help with walking or running. Once, when asked if she could cut her meat and eat all by herself, she affirmed that she could, if only helped to sit in her bed.

She occasioned some abstract knowledge of her deficit. She said that doctors had told her she had hemiplegia, but that she did not believe it, since she was not paralysed and could walk.

The patient also showed a variable degree of right aphasisomatognosia. In several instances, when shown her right hand, she would answer that it was the hand of a corpse that had been introduced into her bed. However, she sometimes correctly identified the hand as her own. Touching it with her left hand apparently facilitated the identification. When asked to designate her right hand she was initially unable to find it in the bed. Twelve days after onset, she could correctly designate her right arm, leg, eye and cheek.

Her spontaneous speech was considerably aphasisic and she was initially unable to sing at all, although she was previously used to participate in an amateur choir. Two weeks after the stroke, her few attempts at singing were very much out of tune. Moreover, her perception of melodies was also impaired. Five days post-onset, she could not identify common tunes that were hummed to her, but recognised them readily as soon as the lyrics were added. Three weeks later, she could identify five out of 10 popular tunes whose melodies were sung to her.

The patient described herself as completely right handed, and denied having been forced to use her right hand as a child. There was no record of any history of cerebral trauma in birth or early childhood. She was submitted to a 24 item questionnaire about her preferred hand or foot in various everyday-life activities. She always unambiguously chose the right side (she was a girl). For pair-related grand-parents and two children we were all reported to be left handed.

Language evaluation was normal in all respects. Spontaneous speech, repetition, object and picture naming, designation, fluency in controlled association, comprehension, and reading were flawless. She was too awkward with her left hand to allow evaluation of writing. She had no left hand apraxia. Visual identification of objects, colours and faces was normal.

The patient was a right handed woman, with no family history of left handedness. After a left parieto-temporal infarction, she showed several symptoms that usually follow right hemispheric lesions, that is, symptoms encompassed in the so-called minor hemisphere syndrome. She had right hemispatial neglect, right hemisomatognosia, anosognosia for right hemiplegia, motor aphasias and a severe impairment in identifying and producing musical tunes but no aphasia.

Two main case favours the hypothesis that there is no obligatory link between any two of the three cerebral functions we have considered, and
that each can be supported by one hemisphere or the other.

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Pure sensory stroke due to midbrain haemorrhage

After reading the interesting case reported by Azzuvi et al, we would like to report a similar case, but in our patient the underlying disease was a cavernous angioma.

A 42 year old woman was admitted to our hospital because of a mild occipital pain, and dysaesthesia in the left part of her body. Neurological examination showed a fully oriented patient, loss of touch and pain sensations then involved the entire left side of her body, including the face. Vibration and position sensations were normal as were motor and cerebellar functions. Tendon jerks were symmetrical and plantar responses were flexor. Laboratory examinations, ECG and chest radiographs were normal. A CT showed a haematoma in the right dorsal and lateral aspect of the pons. The angiographic study of both vertebral arteries showed no abnormalities. The neurological disorder resolved within three months. An MRI performed three months later revealed a hypodensity, of 0.8 x 1.8 cm diameter, in the right dorsal and lateral aspect of the pons, suggesting a cavernous angioma (fig.).

Since the first description in 1977, only eight cases of haemorrhagic pure sensory stroke (PSS) have been described. All of them were secondary to small haematomas in the thalamus, internal capsule or pons. As far as we know, this is the first case of haemorrhagic PSS secondary to cavernous angioma located in the pons. In our patient, as in the one reported by Azzuvi et al, the damage was restricted to the right dorsal spinothalamic tract without involving the medial lemniscus. We agree with the authors that small haematomas located on the sensory pathways, before they reach the thalamus, can produce partial PSS. MRI is useful in detecting vascular malformations, even when, as in our case, angiographic studies are normal.

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Parkinsonism and defects of praxis following methanol poisoning

The neurological sequelae of methanol intoxication shows marked individual variation, but Parkinsonism, optic atrophy and focal cranial nerve deficits have been described. We report a patient with Parkinsonism and dyspraxia who recovered from acute severe methanol poisoning. The MRI scan showed damage of the deep grey nuclei.

A 28 year old woman with a history of depression and recurrent alcohol abuse experienced nausea, vomiting, ataxia and blurred vision for 24 hours before she was admitted supine, with dilated pupils that did not react to light. The optic fundi were normal, and there were no localising neurological signs. The liver was enlarged. She had a leucocytosis of 12900/mm³ and a severe metabolic acidosis (pH 7.15; bicarbonate 2.8 mM Eq/l; pO₂, 129 mm Hg; pCO₂, 8 mm Hg).

With the possibility of methanol poisoning in mind, she was given intravenous infusions of sodium bicarbonate and ethanol, and haemodialysis. The level of methanol in the blood was 2.85 g/l. After three hours, the acidosis was controlled. When the patient’s level of consciousness improved, she consented drinking some 200 ml of methanol in the past 48 hours. She left the hospital after four days without apparent neurological or ocular abnormalities.

She was examined two years later, complaining of motor slowness and loss of memory. She had been working at the same factory at which she was employed before taking the methanol. The work required sequential and repetitive movements of both hands and feet. The employers stated that her productivity was reduced by 50%.

Neurological examination showed a mild dysarthria, a Parkinsonian-like syndrome, with an expressionless face, limp bradykinnesia, and abnormal postural reflexes. Rapid alternating finger movements were poorly performed. The glabella tap sign was positive and palmar-mental and peri-oral reflexes were present; her gait was characterised by poverty of associated movements, with reduced arm-swinging and body turning. The optic fundi displayed bitemporal pallor. Visual acuity was normal. Chest radiographs, ECG and EEG were normal. An unenhanced CT scan showed bilateral areas of decreased density, especially in the putamen. An MRA scan (fig.) showed bilateral lesions involving the claus-
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