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

Some degree of unawarness of hemiplegia occurs in about one third of cases of right hemisphere patients with left hemiplegia.1,4 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.1 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 mixed 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 a right hemispheric lesion. 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 hypoaesthesia 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 right hemispheric injury and 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 colors, 10 pairs of sentence fragments). She showed clearcut right agraphy 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 she could move her hand, sew, or knit, she answered that she could. When asked if she could move her right leg, she answered positively and moved her left leg as a proof. Sometimes, the patient admitted that she had helped 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 occasionally showed 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 asomatognosia. 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 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, and eye.

Her spontaneous speech was considerably aposodic and she was usually unable to sing at all, although she 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 previous 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 hand for almost all tasks, grand-parents and two children as reported to be right 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, for instance. 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 hemispheric syndrome. Such are right hemispatial neglect, right hemisomatognosia, anosognosia for right hemiplegia, motor aprosodiA and a severe impairment in identifying and producing musical tunes but no aphasia. Two main conclusions can be drawn concerning the patient's pattern of cerebral dominance. First, her right hemispheric was certainly dominant for language. If the left hemisphere was dominant for language, or if language was bilaterally represented, the extensive left hemispheric softening would have caused aphasia, according to Kertesz. This was not the case. Second, her left hemisphere was strongly dominant for manuality, spatial attention, and body schema."

Several cases of right hemisomatognosia or anosognosia for right hemiplegia have been reported. Drunker and Knight1 have described a left handed patient with clear-cut anosognosia for right hemiplegia, right sparing apraxia, and anosognosia for right hemispheric infarct. Cramber et al. have reported a similar case, but their patients seems to be ambidextrous. Such is also the case of the patient reported by Hermann and Potrel. Among their patients with right hemisphere lesions, Hecaen and Sauguet mention right hemisomatognosia in two out of 47 left-handed patients, but in none of 293 that were right-handed. Gross and Kaltenback1 have described two patients with such a denial of right hemiplegia, including one case with hemisomatognosia. These patients were both aphasic, but there is no mention of their right hemisphere. Hakstian has described three instances of denial of hemiplegia out of 22 testable right hemiplegic patients. Their manual dominance and degree of language impairment, however, are not reported.

The remaining hemiplegic patients with body schema is usually the right hemisphere. However, as we have just seen, considerable evidence exists of reversed dominance, as revealed by right somatosensory and anosognosia for right hemiplegia. The left hemisphere is usually dominant for manuality and language, although reversed dominance in this domain is also well known. But are the different patterns of co-dominant in concern ing language, manuality and body-schema biologically possible? Or are some of these functions necessarily supported by the same hemisphere (or by opposite hemispheres)? We shall consider each pair of functions in turn. First, the left hemisphere is usually dominant for both language and manuality. Our patient falls into the 4% of those that are right handed with right hemispheric dominance for language and where attention, and Milner's figures (for discussion, see Geschwind and Galaburda13). Second, manuality and body-schema are usually dissociated since in most right handed subjects, the right hemisphere is dominant for body schema. The patient reported by Drunker and Knight shows exactly the opposite pattern of dominance. However, Hecaen and Sauguet1 mention several left handed patients with left anosomatognosia, suggesting that the right hemisphere can occasionally support both functions. The pattern of impairment in our patient demonstrates that this may also occur for anosognosia. Third, language and body-schema are usually dissociated, but one hemisphere can occasionally be dominant for both functions, as illustrated by the patients of Gross and Kaltenbach,1 who have a preserved aphasia and anosognosia for hemiplegia following left hemispheric lesion. Our patient's dominance pattern is the exact opposite of the pattern which prevails in cases where the right hemisphere is right handed, since her left hemisphere is dominant for body schema, and her right hemisphere for language.
that each can be supported by one hemi-
phere or the other.

LAURENT COHEN
PHILIPPE RÉMY
ANNE LEROY
CHRISTIAN GENY
JEAN-DENIS DEGOS
Clinique des Maladies du Système Nerveux,
*Hôpital de la Salpêtrière,
Paris, France
Service de Neurologie, Hôpital Henri Mondor,
Créteil, France
Correspondence to: L Cohen, Clinique des
Maladies du Système Nerveux, Hôpital de la Sal-
pêtrière, 47 Bd l'Hôpital, 75651 Paris Cedex 13,
France.

1 Gross H, Kaltenbäck E. Die Anosognosie.
Wiener Zeitschrift für Nervenheilkunde
2 Cutting J. Study of anosognosia. J Neurol
3 Bisach E, Vallar G, Perani D, Papagno C, Berti
A. Unawareness of disease following lesions of
the right hemisphere: anosognosia for hemi-
plegia and anosognosia for hemianopia.
Neuropsychologia 1986;24:471–82.
4 Hecaen H. Introduction à la neuropsychologie.
5 Heilman KM, Watson RT, Valenstein E. Neglect
and related disorders. In: Heilman KM, Valenstein E, eds. Clinical neuropsy-
chology. New York: Oxford University Press,
1985.
6 Ross ED. The aprosodias: Functional-anatomical
organisation of the affective components of
language in the right hemisphere. Arch Neurol
1951;38:561–9.
7 Zatorre RJ. Musical perception and cerebral
function: a critical review. Music Perception
184;2:196–221.
8 Donkers NF, Knight RT. Right-sided neglect
in a left-handed: Evidence for reversed hemi-
spheric specialisation of attention capacity.
9 Cambier J, Mason M, Goujon M, Robine B.
Neglélige droite avec hemisomatognosie,
confusion mentale, apraxie et apragnosie sans
10 Hermann G, Pötzl O. ÜberAgraphie und ihre
inhaltlichen Begleiterscheinungen. S Karger:
Berlin, 1926.
11 Hecaen H, Sauguet J. Cerebral dominance in
12 Rasmussen T, Milner B. Clinical and surgical
studies of the cerebral speech areas in man.
In: Zulch KJ, Creutzfeldt O, Galtbräuch GC, eds.
Ostitied Foerster symposium on cerebral lateral-
13 Geschwind N, Galaburda AM. Cerebral lateral-
isation. Biological mechanisms, associations,
and pathology. 1. A hypothesis and a program

Pure sensory stroke due to midbrain haemorrhage

After reading the interesting case reported by Azouvi et al.,1 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 radiographs were normal. A CT showed a haematoma in the right dorsal and lateral aspect of the pons. An arteriographic 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 0.6 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 1977,2 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.3 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 Azouvi et al.,4 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.

J ALVAREZ-SABIN
J MONTALBAN
M TINTORE
A CODINA
Department of Neurology,
Hospital General Vall d'Hebron,
Barcelona, Spain
Correspondence to: Dr Alvarez-Sabin, Rda San
Antonio, 5 Ppal, 08011 Barcelona, Spain.

1 Azouvi Ph, Tougeron A, Hussonois C,
Schouman-Cheys E, Bussel B, Hedel JP.
Pure sensory stroke due to midbrain haemor-
rhage limited to the spinothalamic pathway. J
2 Groothuis DR, Duncan GW, Fisher CM.
The human thalamocortical sensory path in the
internal capsule: evidence from a small cap-
sular haemorrhage causing a pure sensory

Parkinsonism and defects of praxis following methanol poisoning

The neurological sequelae of methanol in-
toxication shows marked individual varia-
tion, but Parkinsonism, optic atrophy and focal cranial nerve deficits have been de-
scribed.1 We report a patient who presented with Parkinsonism and dyspraxia and who recovered from acute severe methanol poisoning. The MRI scan showed damage of the 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 to university hospital in a condition where she did not react to light. The optic fundi were normal, and there were no localising neo-
logical signs. The liver was enlarged. She had a leucocytosis of 12900/mm3 and a severe metabolic acidosis (pH 7.15; bicarbonate 2.8 mmol/l; pO2 129 mmHg; pCO2 8 mmHg). 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 was advised to drink 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, com-
plaining of motor slowness and loss of
memory. She had been working at the same factor 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 dysharmonia, a Parkinsonian-like syndrome, with an expressionless face, limb bradykin-
esia, and abnormal postural reflexes. Rapid alternating finger movements were poorly performed. The gait was wide-based and palmar-mental and peri-oral reflexes were present; her gait was characterised by poverty of associated movements, with reduced arm-
waving and body turning. The optic fundi displayed bitemporal pallor. Visual fields were normal. Chest radiographs, ECG and EEG were normal. An unenhanced CT scan showed bilateral areas of decreased density, especially in the putamina. An MRI scan (fig) showed bilateral lesions involving the claus-

Figure: Axial and sagittal T1 MRI showing a cavernous angioma in the right dorsal part of the pons.