Letters

Ideomotor apraxia arising from a purely deep lesion

Sir: Ideomotor apraxia arising from a purely deep lesion has not been reported frequently in the literature. Even in the few cases published,^{1,2} the lesions were not strictly confined to the basal ganglia. Systematic studies,^{3,4} however, have revealed the presence of ideomotor apraxia in about 15% of patients with lesions confined to thalamic or lenticular regions. We report the case of a patient with a haemorrhage confined to the head of the left caudate nucleus and the contiguous arm of the internal capsule, who had severe ideomotor apraxia and aphasia as well. Even though the haemorrhagic nature of the lesion renders the interpretation of the anatomoclinical correlations rather difficult, this case is interesting because of the coexistence of severe ideomotor apraxia and a deep lesion.

A 61-year-old right-handed male with 4 years of education suddenly developed aphasia and a right hemiparesis. Three subsequent CT scans performed on the 1st, 8th and 19th day after onset, showed a small area of increased density consistent with an intraparenchymal haemorrhage limited to the head of the left caudate nucleus, the anterior limb of the internal capsule and the medial part of the lenticular nucleus (fig). Neuropsychological assessments were performed 20 days after onset. His speech was dysarthric. In oral confrontation naming there were frequent word-finding difficulties with rare circumlocutions and semantic

paraphasias. Oral comprehension was impaired: he scored 15/36 on the Token Test. Spontaneous writing and writing to dictation were impossible: he produced isolated signs among which only few letters were recognisable. Repetition was good except for being dysarthric. He scored 5/36 on the Raven's Coloured Progressive Matrices. He had no oral apraxia. Testing for constructional apraxia was impossible: in every instance the patient tried to write instead of copying the model. He scored 35/72 (cut-off score: 53/72)⁵ on De Renzi⁶ ideomotor apraxia test. There was no difference between arm/hand movements (18/36) and finger movements (17/36). There was, however, a striking difference between single movements (25/36) and sequences (10/36).

Our patient also had a mild apraxia of use: for instance, when asked to light a candle he could not manage to light the match. Aphasia following a deep lesion has long been accepted⁷ and it will not be commented on. Ideomotor apraxia arising from a purely deep lesion has been occasionally reported in group studies. Our patient had a small and well localised lesion which caused, among other deficits, a severe ideomotor apraxia. It would seem that the role of deep structures in the genesis of apraxia, not taken into account by the majority of authors with few exceptions,^{8,9} deserves reconsideration.

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Unusual EEG pattern in rubella encephalitis

Sir: Encephalitis is a rare but well recognised complication of rubella infection.¹ The EEG findings in the acute phase²⁻⁵ are reported to show continuous slow activity with no specific features. We report a case of rubella encephalitis with unusual repetitive complexes.

A 15-year-old Japanese boy with no past history or family history of note, presented with a 4 day history of fever and rash. The rash started on the face, spread down to the body, and faded 2 days later. The day prior to admission, he developed headache and dizziness and within 24 hours had become drowsy and lapsed into coma. Examination revealed an unconscious, restless patient only responding semi-purposefully to painful stimuli. There was moderate neck stiffness but no focal neurological deficits. The pupils were 4 mm in diameter, and reacted to light. The fundi were normal. De-

Fig Post-contrast CT scan 8 days from onset. The luxury perfusion well defines the infarcted area.

tendon reflexes were normal and both plantar responses were flexor. Full blood count, serum electrolytes, liver function test, Paul Bunnell test, chest radiograph and CT scan were normal. CSF contained 31 white cells per mm³, mainly lymphocytes, protein 1.5 g/l and sugar 3.2 mmol/l. Blood sugar was 5.0 mmol/l.

The first EEG (fig) which was done immediately on admission, 4 days after onset of the rash, showed generalised slow activity at 1.5 to 2.5 Hz, 50 to 200 μ V in amplitude with no alpha rhythm and only traces of theta and fast activity. Some of the slow waves appeared as non-stereotyped triphasic complexes at intervals from 1 every 3 seconds to 1 every 5 seconds. At times they were prominent in the temporal leads but at others in the anterior leads. In view of the severity of the illness and the EEG appearance treatment with intravenous acyclovir 10 mg/kg, 8 hourly was commenced immediately and was continued for 10 days. Immunological studies showed changes diagnostic of rubella encephalitis with rubella CSF titres of 1/32, a significant fall of serum titre from 2048 units to 512 units two weeks later and a positive rubella specific IgM assayed by short and long term fixation methods (Dr Holzel). The patient made a rapid recovery regaining consciousness in 3 days at which time the EEG only showed intermittent frontal slow

wave disturbances but no complexes. He was discharged within 14 days.

The EEG in acute encephalitis usually shows continuous slow activity with no specific features.⁶ The presence of generalised, ill defined and asymmetric triphasic complexes probably reflects a generalised process affecting different parts of the brain to a different extent, and in the absence of an obvious cause of periodic complexes⁶ this raises the possibility of herpes simplex encephalitis. This was subsequently excluded by immunological studies. Although the EEG shows some features different from what is found in herpes simplex encephalitis,⁷⁻⁹ this diagnosis could not be excluded solely on EEG grounds. Typical EEG findings in herpes simplex encephalitis occur only in about 50% of cases.^{8,9} Hence it is important to treat suspicious cases as presumptive herpes simplex encephalitis. This case shows that periodic complexes are clearly not pathognomonic of herpes simplex encephalitis.

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Fig 1st EEG performed on the day of admission, 4 days after the onset of the rash showing repetitive triphasic complexes. The apparent right sided predominance is not constant and there is no definite lateralization throughout the record.