Ataxic hemiparesis and mirror movements

SIR,—We have recently seen an elderly male with a unique combination of ataxic hemiparesis and mirror movements.

Case report A 56-year-old hypertensive male experienced an episode of weakness of right arm and leg and unsteadiness of gait six years prior to admission. There was no headache, unconsciousness, seizure or speech difficulty during the episode. A week after the ictus the patient began to improve, and after six months the only disability was clumsiness of right hand. On examination he was alert and oriented with normal speech. The visual fields were full and the optic fundi showed early arterial changes of essential hypertension. The pupils were equal and reacted to light. Positive neurological signs were a fine horizontal nystagmus on lateral gaze to each side, a mild right hemiparesis with hyper-reflexia and extensor plantar response, and intention tremor, dysmetria and dysidiadochokinesis confined to the right extremities. Relevant negative findings included normal ocular movements, absence of facial asymmetry, intact sensation and no Rombergism. It was observed that active pronation and supination of the forearm, flexion-extension movements at the wrist, independent finger movements and rapid finger tapping induced similar movements on the opposite side. These mirror movements were induced by voluntary movements on either the right or the left side. Similar movements were not present in the lower limbs and he could open or close one eye only. The patient was aware of these abnormal movements and was certain that they were not present before the episode of hemiplegia and no other member of the family had similar disorder. The blood pressure was 170/106 mm Hg. The

carotid pulsations in the neck were equal and there was no arterial bruit. Routine urine analysis, haemogram and blood chemistry were within normal limits. Serology for syphilis was negative. Cerebrospinal fluid and electroencephalography were normal. Electrocardiography showed occasional ventricular ectopics. Radiographs of the cervical spine, craniovertebral junction and chest were normal. He was discharged after adequate control of hypertension.

Ataxic hemiparesis as the stroke syndrome of elderly hypertensive individuals is a distinct clinicopathologic entity. It is established that the lesion responsible for ataxic hemiparesis lies at the junction of upper one-third and lower two-third of the basis pontis on the side contralateral to the pyramidal and cerebellar signs.¹ The clinical history and neurological findings in our patient suggest ataxic hemiparesis. Though tumour and demyelinating process can produce the syndrome, the clinical profile in the present case is indicative of a vascular episode.

Mirror movements are bimanual synkinesis consisting of involuntary imitative movements of one limb when the opposite limb carries out rapid movement. Mirror movements are present during normal development, decrease with maturation and rarely persist beyond the age of 14 years.² They may persist into adult life, as the result of early cerebral damage as in childhood hemiplegias,³ congenital malformations of the cervical spine and spinal cord as in Klippel-Feil syndrome and related myelodysplasias,⁴ or on a genetic basis transmitted as autosomal dominant.⁵ One unproven hypothesis to explain congenital mirror movements is that there is a less complete pyramidal decussation with a much higher proportion of uncrossed ipsilateral pyra-

midal tract fibres.^{4,5} It is speculated that in the presence of inadequate decussation of the pyramidal tracts, a lesion involving discretely the pyramidal fibres as in ataxic hemiparesis might be responsible for the reappearance of mirror movements at a later age. This remains as the plausible explanation for the appearance of mirror movements in our patient after the stroke syndrome, since the role of crossed and uncrossed pyramidal fibres in the distal synkinesis is at present entirely unknown. Pathological confirmation in similar cases might provide further important insight into the neurology of intentional movements, in relation to its normal development and reorganisation after nervous system damage, both in terms of the site and time of occurrence after maturation.

References

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