

Letters

Micrographia secondary to lenticular haematoma

Sir: A focal lesion of the lenticular nucleus is usually associated with hemidystonia, hemichorea and Parkinsonism. We describe the case of a patient with a writing difficulty as the only clinical manifestation of a lenticular haemorrhagic lesion.

A 59 year old man with a previous history of hypertension was admitted after developing right hemiparesis of acute onset. On examination, blood pressure was 210/120 mmHg; facial asymmetry with deviation of the mouth to the left, severe brachial weakness (0/5) and moderate (2-3/5) crural paresis were present. There was also a speech disorder characterised by dysarthria with impaired fluency and anomia. Tendon jerks were sluggish in the right arm and hyperactive in the right leg. Plantar response was extensor on the right and flexor on the left side. The rest of the neurological examination was unremarkable. CT of the brain at that time showed a large left basal ganglia haematoma with moderate oedema. The patient recovered motor function and speech over the next 2 weeks and was discharged.

Two months later a new examination found no weakness on the right side. Sensation, coordination and speech were normal. Tendon jerks were slightly asymmetrical, but not pathologically hyperactive in the right limbs. No abnormal postures were seen in the limbs. Finger counting and repetitive finger movements with the right hand, and simultaneous bilateral hand activation were all normal. The patient's only difficulty, as he volunteered, consisted in writing. He held the pen correctly and initiated writing with a normal speed and rhythm. However, his calligraphy became progressively smaller as he continued to write, until it was barely understandable (fig A). At this moment the patient complained of a feeling of tightness in the hand, but no abnormal posture was observed throughout the examination.

Electromyography of the forearm and hand muscles revealed a normal pattern of fast ballistic movements of the right wrist (either flexion or extension); movement time and reaction time were also normal when the same movements were initiated by an external somesthetic or auditory cue. During writing, EMG bursts in opponens pollicis, first interosseous, finger flexor and finger extensor muscles of the right limb were pathologically fractioned and decreased in amplitude as writing continued.

There was no excessive activation or cocontraction of antagonist muscles, as seen in dystonia. A new CT brain scan showed a linear (antero-posterior) low density lesion of the left lenticular nucleus (putamen mainly) and mild compensatory dilatation of the left anterior horn. (fig, b and c).

This patient showed a typical micrographic handwriting, similar in every respect to that observed in Parkinson's disease. Writing is known to be one single, although complex, motor program.¹ Recent physiological studies suggest that motor plans and simple motor programs are relatively intact in patients with Parkinson's disease;² however, there is an abnormal quantitative specification of the components of the motor programs.³ The characteristics of the abnormality shown by our patient indicate that he was capable of adequately selecting and starting the muscle activity necessary for writing, but failed to run the motor sequences adequately. This observation provides further clinical support for the contention that the striopallidal complex is involved in the automatic execution of simple and complex motor programs.⁴

The underlying reasons for lenticular lesions producing a single motor disturbance such as writing, or severe movement disorders such as hemidystonia and hemichorea, non-motor signs (that is psychic akinesia, aphasia) or no clinical manifestation at all, are not known. Understanding such an apparent paradox will probably lead to a better knowledge of the basal ganglia functional organisation.

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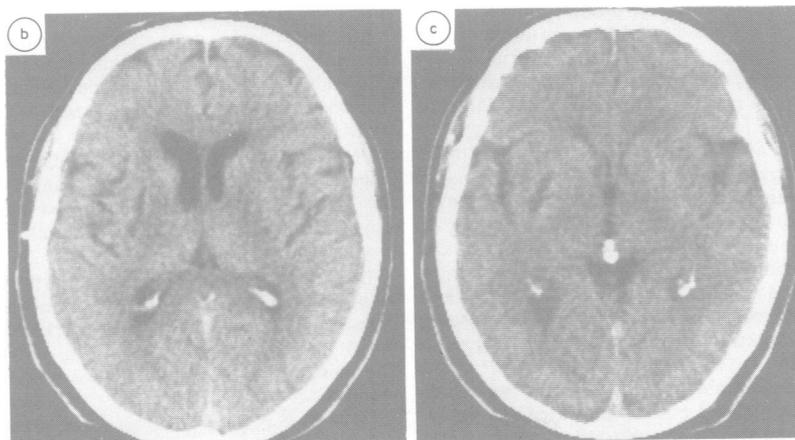


Figure (a) Example of patient's handwriting showing typical micrographia. (b and c) CT brain scan above and at the level of the third ventricle taken two months after stroke; a linear hypodense lesion in the left lenticular nucleus, mainly in the putamen, and mild compensatory dilatation of the anterior horn are shown.