Short report

Non-paralytic motor disturbances and speech disorders: the role of the supplementary motor area

HJ GELMERS

From the Department of Neurology, Pr Irene Ziekenhuis, Almelo, The Netherlands

SUMMARY A right-handed patient with a lesion demonstrated by CT to involve the right medial frontal cortex is described. He exhibited a strong contralateral grasp reflex, motor perseveration and the presence of purposeful movements that appeared to be dissociated from conscious volition. In addition, there was a disorder of speech consisting of a lack of spontaneous speech production, with preserved ability to imitate. It is suggested that these disorders are due to damage to the supplementary motor area.

Experimental studies in man indicate that there are a number of cortical fields involved in motor control, among which is the supplementary motor area. Although its functional distinctiveness has been demonstrated, there are few descriptions of the results of supplementary motor area damage. The purpose of this case history is to describe the clinical symptoms caused by a lesion involving the right medial frontal cortex and to outline the "supplementary motor area syndrome".

Case history

A 69-year-old right-handed man had sudden onset of difficulty in speaking, together with weakness of the left leg. Examination on admission showed him to be awake, alert and oriented. He had a mild left-sided lower facial weakness at rest and on volition, with a striking weakness after laughing. There was total paralysis of the left leg. Although the left arm showed a global inertia and no spontaneous movements, there was no paresis. The patient found it an immense effort to move. When he moved, the movements were correct but slow and deliberate. A strong grasp reflex was noted in the left hand. Tendon reflexes were exaggerated on the left and plantar responses were extensor on the left and equivocal on the right. Visual fields and ocular movements were full. Pinprick and vibratory sensation was normal as were detection of passive movements and identification of palpatated objects. The patient did not initiate conversation. He spoke very haltingly in reply to questions. The responses were characterised by a great deal of difficulty in initiation. Comprehension of speech was good, as was the ability to imitate complex phrases sentences. There were no localising phenomena indicating parietal lobe damage; the interpretation of body-perception was normal. Computed tomograms demonstrated an area of increased density involving the right medial frontal cortex (2nd day after onset of symptoms) (fig). Ten days after onset there was still a strong grasp reflex in the left arm. The left leg had improved in strength, but remained weak. The patient was observed to reach out spontaneously with the left arm and then would not be able to release his grip voluntarily. He began to perseverate with his left arm when using a knife and fork during meals, or when handling a haircomb or an electrical razor. This motor perseveration was inhibited to some extent by concentrated effort. After five weeks the patient began to walk unaidsed, but he still displayed motor perseveration of the left arm. This phenomenon was indeed perseveration and not tonic innervation or an inability to release objects on account of the grasp reflex.

Discussion

This patient displayed a strong grasp reflex of the left hand initially preceded by a global inertia. He was observed to have motor perseveration (inappropriate repetition of a simple movement) of the left hand. Later it was noted that apparently purposeful movements appeared to be occurring independent of conscious volitional control. Additionally, the patient exhibited a speech disorder, consisting of a lack of spontaneous speech with preserved
Non-paralytic motor disturbances and speech disorders

Penfield and Jasper reported that stimulation of the supplementary motor area produced vocalisation, speech arrest, complex coordinated or repetitive movements usually appearing in the contralateral arm and arrest or slowing of voluntary action following which it was noted that the patient was "usually puzzled by his inability to execute" the action he had intended. Continuous electrical stimulation of anterior cingulate cortex in epileptic patients is followed by complex, highly integrated forms of motor behaviour most often in the contralateral arm. Recently, regional blood flow studies in man have demonstrated bilateral activation of supplementary motor area in association with various complex movements of the extremities, as well as with speech. From these studies it has been suggested that the supplementary motor area is involved in the assembly and execution of time-ordered sequences of motor instructions.

Our patient also exhibited a speech disorder, consisting of a lack of spontaneous speech production, with preserved ability to imitate. This type of speech disturbance is often designed as transcortical motor aphasia and has been described in patients who sustained damage to the left medial frontal cortex. Although similar disturbances of speech of patients have been described by others, no mention was made of the site of the involving medial frontal lesion. Since the lesion in our patient was localised in the right hemisphere, it is best not to describe this speech disorder as an "aphasia". Moreover, it covers the recently proposed term "transcortical motor aprosodia".

There seems now sufficient clinical and experimental evidence to conclude that lesions of the medial frontal parts of the hemisphere, including the supplementary motor area, give rise to non-paralytic motor and speech disturbances.

The author is grateful to Professor Dr J Willemse, Department of Child Neurology, Faculty of Medicine, University of Utrecht, for support and advice in this study.

References


