

neuromuscular disorder or of central nervous system lesion. A disturbance of corollary discharge or of efference copy can thus be ruled out. Since no abnormality of sensation of position or of movement was evident before operation, the abnormality seems directly attributable to the lateral meniscectomy. The small area of altered cutaneous sensation in the region of the scar seems unlikely to be relevant since it is so slight. McCloskey *et al*² have discussed the possible errors in Moberg's interpretation that the relatively extensive cutaneous sensory disturbance noted in his patients was important in their altered position sense.

In the cat there is evidence that both muscle receptors and joint afferents are necessary to achieve entrainment, that is correct phase relationships, between limb movement and muscle activity in locomotion.³ This results from neuronal activity at various levels in the nervous system, probably including cord and cortex, at which convergence of input from joint, skin and muscle receptors occurs.⁴

There seem to be two possible explanations for my experience. Firstly, it can be suggested that joint receptors play a role in movement sensation and that this sensation is more complex than static position sense itself. The latter is perhaps only part of the substrate of the former. Movement sensation may thus depend on input from several classes of receptors of which joint receptors are only one component. The abnormality of motor control I have experienced might thus result from disturbance of sensory function. Alternatively, this disturbance of motor control might result from inappropriate reflex corrections occurring after limb displacement. On this view the perceived abnormality would represent out of phase movements causing inappropriate displacement of the limb.

Persistent minor degrees of impairment of function are widely recognised after knee surgery of various types but no systematic evaluation of them seems to have been made. A study of the effect of total knee arthroplasty revealed no change in joint position sense, although abnormalities of this sensation were noted in control knees damaged by degenerative joint disease,¹¹ an observation that might indicate that sensory abnormality resulted from mismatch between disturbed sensory input and motor output in the damaged knees, an abnormality that was removed when this joint was surgically excised and replaced with a prosthesis. It may thus be of particular interest to investigate joint

sense and kinaesthesia before and after joint surgery in previously healthy young people, as well as in an older population in whom surgery has been performed for long standing degenerative joint disease.

MICHAEL SWASH
The London Hospital,
Whitechapel,
London E1 1BB, UK

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Transient paraparesis—a manifestation of ischaemic episodes in the anterior cerebral artery territory

Sir; Transient paraparesis or sudden unexpected falling due to leg weakness without loss of consciousness ("drop attack") occurs in about 15% of patients with vertebro-basilar insufficiency.¹ It is usually associated with symptoms due to brainstem or occipital ischaemia and rarely occurs as an isolated event. Ischaemic episodes in the anterior cerebral territory due to emboli from the internal carotid artery are uncommon, but might give rise to transient paraparesis if multiple emboli affected both anterior cerebral arteries simultaneously. We describe a patient presenting with this symptom in whom angiography showed severe right internal carotid stenosis, both anterior cerebral arteries being supplied from the right internal carotid.

A 49-year-old man was admitted for investigation of episodes of transient neurological dysfunction which began six months previously. These were described as follows: (1) Two episodes of sudden weakness of the right lower limb lasting about ten minutes with complete recovery. (2) Four episodes of sudden weakness of both lower limbs causing him to fall. Strength gradually returned over a few minutes and he was able to walk normally again. In one of the attacks, the patient became very "dizzy" and lost consciousness for some minutes. (3) Ten episodes of "weakness" and numbness in the left hand lasting up to one hour; several occasions these were associated with numbness in the upper and lower lips on the left.

Relevant past history was a blind right eye since birth from congenital cataract. He smoked 56 g of tobacco each week. On examination he was jovial and his behaviour rather disinhibited. He was fully orientated and higher mental functions were normal on simple testing. Cranial nerves were normal; however, the right fundus could not be seen because of severe cataract. In the motor system there was a mild spasticity with some drifting of the outstretched left arm. There was mild weakness of left wrist extension, finger extension and finger abduction; plantar responses were both flexor. There was some impairment of joint position sense, stereognosis and two point discrimination in the left hand. In the cardiovascular system, the radial pulse was 68 per minute and regular. Blood pressure was 165/95 mm Hg and equal in both arms. There were no

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bruits. A soft ejection systolic murmur was present over the left sternal border. Routine blood tests were normal. A fasting cholesterol was slight raised at 8.6 mm/l. ECG and echocardiogram were normal. CT scan showed small low density lesions in the right frontal and parietal lobes consistent with small infarcts. Digital subtraction angiography was technically unsuccessful and four vessel angiography was done under general anaesthetic which showed severe irregular narrowing of the right internal carotid artery with a minimum diameter of less than 1 mm. There was severe hypoplasia of the proximal segment of the left anterior cerebral artery, the distal parts of both anterior cerebral arteries being supplied from the right carotid. There was atheroma without stenosis of the proximal left internal carotid. The vertebral arteries were normal.

Shortly following admission the patient complained of sudden weakness in the left hand and re-examination at that time showed a global paralysis of the left hand including wrist flexion and extension; strength gradually returned to its previous level after a few hours. He also had an episode of weakness and numbness in the left leg which returned to normal after twenty-four hours. Carotid endarterectomy was performed. He made a good recovery and has had no further episodes of transient neurological deficit since the time of operation ten months ago even though he continued to have a mild sensory deficit in the left hand.

The pattern of our patient's neurological deficit was initially interpreted as being due to ischaemia in the territory of the vertebrobasilar system though the absence of other features of brainstem disturbance was unusual. There was no evidence on clinical examination or on an echocardiogram of a cardiac source of emboli. It seemed probable from the angiographic findings that the severe stenotic lesion of the right internal carotid artery was responsible for his symptoms. Since the severe hypoplasia of the proximal segment of the left anterior cerebral artery left both anterior cerebral arteries dependent on the right internal carotid artery, emboli from the latter could clearly cause ischaemia in the territory of the middle cerebral artery on the right and of both anterior cerebral arteries and would explain the episodes of transient weakness affecting both lower limbs. The critical nature of the stenosis also raised the possibility of the attacks being "haemodynamic" in origin though

symptoms suggestive of generalised hypoperfusion were only recorded in one of the attacks.

Severe hypoplasia of the proximal segment of the anterior cerebral artery (A1 segment) has an incidence of 4%.² Embolisation from the internal carotid artery into the anterior cerebral artery is also rare with an incidence of about 7%; the remaining 93% occurring in the middle cerebral artery and its branches.^{3,4} However, in situations when multiple emboli have been thrown off, the anterior cerebral artery is affected in about 50% even though experiments using spherical steel emboli showed that the first embolus was never lodged in the anterior cerebral artery and it was concluded that only after occlusion of the middle cerebral artery or its branches is flow redirected towards the anterior cerebral artery.⁴

The latter reasoning can partly explain the disproportionately high incidence of transient ischaemic attacks affecting the anterior cerebral circulation (approximately 40%) in our patient since there was both clinical and radiological evidence of infarction in the right middle cerebral artery territory. One can also postulate that the common origin of both anterior cerebral arteries had in some way biased flow towards that circulation possibly due to the anatomical variation but also possibly related to an increase in blood flow secondary to both frontal lobes being supplied from the same side.

RTK HO
MJG HARRISON
CJ EARL

Department of Neurology,
The Middlesex Hospital,
Mortimer Street,
London W1N 8AA, UK

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Absence of immunoreactive interferon- α in CSF from patients with multiple sclerosis

Sir; It has been suggested that multiple sclerosis may be due to an infectious agent (a virus) with occurrence only in people immunogenetically at risk.¹ The interferons are antiviral proteins which also modulate immune systems and cell growth. Interest has arisen in the possible therapeutic role of interferons in multiple sclerosis,² but a number of studies of interferons in multiple sclerosis by biological assay have produced conflicting results. Thus some laboratories reported reduced capability of lymphocytes from multiple sclerosis patients to produce interferons *in vitro*,^{3,4} whereas others failed to confirm these findings.^{5,6} Early reports also suggested that measurable quantities of interferons were present in serum and CSF of some multiple sclerosis patients,^{7,8} but a more recent study failed to detect them in 31 patients with multiple sclerosis.⁹

We have used a recently developed highly sensitive immunoassay for immuno-reactive interferon- α to study patients with multiple sclerosis. This is a two-site immunoradiometric assay (IRMA) ('Sucrosep'; Boots Celltech Diagnostics Ltd., Slough) based on a monoclonal antibody (Yok5/19) to interferon- α . The assay can detect as little as 0.3 IU interferon- α /ml serum/CSF and shows no cross reactivity to human interferons β and γ . We measured interferon- α in CSF from 15 patients with multiple sclerosis (10 female, 5 male, mean age 42.3 years). All had definite multiple sclerosis according to recent diagnostic criteria.¹⁰ Twelve had relapsing and remitting and three chronic progressive disease. In the former group lumbar puncture was performed in the early stages of an acute relapse, and prior to the commencement of ACTH treatment. In 11 patients serum was taken at the time of lumbar puncture and assayed for interferon- α . All specimens were stored at -20°C prior to assay, conditions under which interferon- α is stable. We detected no interferon- α by IRMA in any of the specimens of CSF or serum.

Previous discrepancies in studies of interferons in multiple sclerosis probably relate to technical differences in the bioassays used. These assays depend on cell culture and virus challenge and are thus very prone to biological variability. Our results using a highly specific, sensitive and reproducible immunoassay confirm the findings by bioassay of Salonen⁹ that interferon- α is not detectable in the serum or CSF of multiple sclerosis patients. Interferon has