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 midle 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 fontal lobes being supplied from the same side.

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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, but a more recent study failed to detect them in 31 patients with multiple sclerosis.[7]

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 0·5 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 Salonen[9] that interferon-α is not detectable in the serum or CSF of multiple sclerosis patients. Interferon has

recently been detected in the serum of patients with acquired immuno-deficiency syndrome,[11] systemic lupus erythematosis,[12] rheumatoid arthritis[12] and asbestosis[13] and it has been suggested that persistent lymphocyte stimulation due to an immune disorder may lead to high levels of interferon in the serum. Our study, using a reliable immunological assay, suggests that elevated CSF interferon-α concentrations are not present in multiple sclerosis. However, further studies using IRMAs for interferon-γ will allow further insight into the possible role of interferons in the aetiology of multiple sclerosis.

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Treatment of acute myelopathy in systemic lupus erythematosus with plasma exchange and immunosuppression

Sir: Central nervous system involvement occurs in up to 50% of patients with systemic lupus erythematosus (SLE). Happening early or late in the disease, alone or in combination with such manifestations as arthritis, nephritis and cutaneous lesions, it may include generalised epilepsy, disorders of mental function, central disturbances of extra-occular and pupillary innervation, tinnitus and vertigo. Hemiparesis affects about 5% of patients, and transverse myelopathy in SLE has only been reported in thirty cases in the world literature.[2–6] Clinical improvement in the published cases could not be reliably attributed to high dose corticosteroid therapy.[2 9] A slow recovery occurred in only three paraplegic and in one quadriplegic patient from a total of twenty six.[2]

Plasma exchange has been used in diseases where auto-antibodies and/or circulating immune complexes have a demonstrated role in pathogenesis.[10] Examples include myasthenia gravis (anti-acetyl choline receptor antibody),[11] type 1 diabetes mellitus (anti-islet cell antibody),[12] and SLE (anti-double stranded DNA antibody).[13] A good response has been observed in fulminating, steroid-unresponsive SLE, or where cytotoxic drugs were contra-indicated by haematological considerations. Numerous reports of the use of plasma exchange in neurological disorders have been unable to supply firm evidence of efficacy except in certain forms of myasthenia,[14 15] frequently because of the small numbers of patients with rare conditions, as in the case reported below.

A 49-year-old woman was referred in July 1982. She had experienced one week's progressive gait disturbance and impaired manual dexterity, culminating in a 24 hour onset of quadriplegia and acute urinary retention. Five years earlier she had been investigated for polyarthritis affecting the knees and interphalangeal joints. The latex test for rheumatoid disease was negative. Serum anti-nuclear factor titre was 1/1000 and SLE was diagnosed. A year later, a photosensitive skin rash settled spontaneously in a few weeks. She remained well until early 1982 when she again noticed hand and knee stiffness: this responded quickly to phenylbutazone. Soon after, transient attacks of vertical diplopia were reported. Early in July she developed unsteadiness, trembling, tingling sensations in both hands and visual blurring. Romberg's test was positive, but no other abnormality was found. Four days later she was admitted as an emergency, complaining of weakness and clumsiness of both arms and legs, and difficulty in initiating micturition with normal bladder sensation.

Systemic examination revealed facial erythema and telangiectasia with a scaly rash on the arms. The bladder was enlarged. She was well oriented and alert; speech was normal, although the content revealed a high degree of anxiety and repetition, not in keeping with her previous character. No abnormalities were found in the cranial nerves or upper limbs. Walking was impossible owing to an asymmetrical spastic paraparesis, denser on the right. The tendon reflexes were pathologically brisk, with extensor plantar responses. She was initially treated with prednisolone at 60 mg/day, increased after 24 hours to 120 mg/day. After continued deterioration over the next day, she was transferred to Guy's Hospital.

Examination again revealed a high degree of anxiety. She had a spastic tetraparesis greater on the right side. Sensation was impaired up to the C5 level, affecting both light touch and pain; two point discrimination in all fingers was absent, and vibration sense was lost below the tibial tuberosities. A myelogram was normal and CSF examination showed a white cell count of 165/mm^3, mainly polymorphs; the CSF protein concentration was 0·33 g/l. A diagnosis of transverse cervical myelopathy was made.

The first plasma exchange was performed on the night of admission, and in total, eight exchanges were made on alternate days; five litres initially and subsequently four litres of plasma were exchanged using a Haemonetics model 30 machine. Circulatory access was by a 'Vygon' 3·3 mm catheter placed in the left femoral vein; return was via a 14 gauge 'Abbocath' placed in an anticubital vein.