Table Dosage of methylprednisolone and functional response in acute exacerbations of multiple sclerosis

Case	Age	Sex	Duration	* Disability grade before treatment	Treatment: iv methylprednisolone	* Disability grade after treatment	Marked improvement after (hours)
1	18	F	2 months	6	250 mg 5 days	3	24
2	25	F	2 years	8	250 mg 5 days	6	72
3	23	F	6 years	7	250 mg 5 days	6	48
4	33	M	1 month	9	500 mg 2 days 250 mg 3 days	2	24
5	53	M	8 years	8	1 gm 5 days	5	24
6	22	M	5 years	6	1 gm 5 days	3	24

*Score on Kurtzke Disability Scale * before treatment and 10 days after beginning iv methylprednisolone

methylprednisolone might produce rapid improvement in multiple sclerosis is unclear. Steroids have many biological actions,¹⁰ including anti-inflammatory and immunosuppressive effects; in an acute plaque there is inflammation and oedema with IgG synthesis.⁷ Local inflammation and oedema cause conduction block along an axon,¹¹ and the rapidity of the response in our patients is best explained on the basis of a reduction of inflammation and oedema, rather than an immunosuppressive effect.

The induction of a rapid remission might reduce the severity of residual disability following an exacerbation, and since CNS IgG synthesis is suppressed after high dose intravenous steroid therapy,^{12,13} it is conceivable that there may be a longer term beneficial effect. Conventional management with rest, intramuscular ACTH and physiotherapy in acute exacerbations is based on the controversial results of earlier trials.^{1,14} A controlled trial of high-dose, "pulsed" intravenous methylprednisolone treatment is warranted.

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Traumatic middle meningeal arteriovenous fistula and primitive trigeminal artery

Sir: It is well known that intracranial vascular abnormalities, such as aneurysms and arteriovenous angiomas, tend to occur together with a persistent primitive trigeminal artery. According to Jayaraman *et al*,¹ who reviewed 11 cases of primitive trigeminal artery and cerebral arteriovenous malformation, and added one of their own, an occult arteriovenous fistula has to be suspected when the artery is found in the course of intracranial bleeding of unknown origin.

Here I report the association of primitive trigeminal artery and middle meningeal arteriovenous fistula in a 42-year-old right handed man, who developed an inability to speak and to use his right arm and leg following a mild head injury. Skull radiographs were normal. Left carotid angiogram revealed a lenticular avascular parietal area and a dural arteriovenous fistula, which drained into the superior sagittal sinus. In addition, a carotid-basilar anastomosis of the trigeminal type was seen (fig). Two days later, evacuation of the extradural hematoma and electrocoagulation of the arteriovenous fistula were successfully performed.

In almost all instances, traumatic middle meningeal arteriovenous fistulae develop as a result of a skull fracture across the middle meningeal groove tearing the arterial wall. However, Nakamura *et al*² failed to note this finding and Markham³ recorded a case of arteriovenous fistula between the right middle meningeal artery and great petrosal sinus in a young female with no history of head trauma. These reports are

Fig. Angiogram showing carotid-basilar anastomosis (long arrow) and dural fistula (small arrow)

consistent with the hypothesis that dural arteriovenous fistula can be due to a malformation, as may be the case in the present observation. Indeed, the persistence of a trigeminal artery provides some indirect evidence that a structural defect may have predisposed to the disruption of the middle meningeal artery following a traumatic accident, which would have had little effect on a normal artery.

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Pronator syndrome due to thickened bicipital aponeurosis

Sir: The entrapment of the median nerve

at the elbow gives rise to a compression neuropathy commonly called the pronator syndrome.¹⁻⁵ We studied a patient presenting this symptom-complex caused by a thickened bicipital aponeurosis compressing the median nerve.

A 55-year-old brick-layer noted weakness of his left hand and numbness of the thumb, second and third finger, associated with pain at the left wrist, forearm and arm. These symptoms had an acute onset after a day in which the patient had carried heavy building material supported on his left forearm. When admitted, two months later, examination revealed a slight wasting of the thenar eminence and of the volar aspect of the forearm. Tinel's sign over the median nerve at the elbow was positive. There was weakness of the pronator teres, flexor digitorum sublimis, flexor digitorum profundus to the second and third finger, flexor pollicis longus, opponens pollicis, abductor pollicis brevis. The hand displayed a "benediction attitude" when the patient attempted to make a fist. Reduction of sensation was demonstrated over the median nerve distribution in the hand. Neurological examination failed to reveal any other abnormality. The electrophysiological study showed denervation and neurogenic atrophy in the flexor digitorum sublimis, flexor digitorum profundus, abductor pollicis brevis. Distal motor latency of the median nerve was 3.6 ms in the left side and 3.7 ms in the right side: motor conduction velocity across the elbow was respectively 48 ms⁻¹ and 67 ms⁻¹. Radiography of the chest and of the left upper extremity was normal, as were a complete blood count and the sedimentation rate. At operation, the median nerve, explored in the antecubital fossa, was found to be entrapped beneath a thickened bicipital aponeurosis. Section of the structure exposed a flattened portion of the nerve in the site of the compression, and a swelling of the nerve trunk just above it. The recovery of the patient was complete in few months. The most common causes of the median nerve compression at the elbow are an hypertrophied pronator teres, the passage of the nerve under both its heads, and the kinking against the sublimis bridge.²⁻⁴⁻⁵⁻⁷ In our patient, however, the weakness of the pronator teres muscle suggested that the entrapment of the nerve was indeed above the elbow. Besides acute traumatic incidents, some anatomical anomalies, such as a supracondylar process of the humerus, the Struthers ligament or a thickened bicipital

aponeurosis can be causative factors of this syndrome.⁵⁻⁷⁻⁸ Radiographs excluded the presence of a bony supracondylar process. Clinically it is not always possible to assess if an entrapment is caused by the Struthers ligament or by the bicipital aponeurosis. Our patient's symptoms represented the common features of the entrapment of the median nerve above the elbow, but did not give any suggestion about the structure restraining it.

Some manoeuvres are described which are supposed to be suggestive of the exact site of the compression:²⁻⁷ the entrapment by the Struthers ligament is described as usually associated with forearm pain elicited or increased during forceful extension of the wrist, while reproduction of pain by resistance to forearm supination and elbow flexion is considered a positive sign for entrapment at the bicipital aponeurosis. Both these manoeuvres failed to demonstrate an increase of the forearm pain in our patient. The electrophysiological findings were consistent with a compression of the nerve in the region of the elbow³⁻⁵⁻⁷ but they could not suggest the localisation of it.

Only the surgical exploration of the nerve in the antecubital fossa located the structure responsible for the entrapment, showing a thickened bicipital aponeurosis compressing the nervous trunk. Thus, thickened bicipital aponeurosis must be regarded as a rare cause of the uncommon syndrome of median nerve entrapment above the elbow.

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