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Letters

Facial atrophy during sotalol treatment

Sir: Cold extremities is the most common side effect of "betablockers" and, during treatment, intermittent claudication often gets worse.[1] The reason probably is arterial constriction in voluntary muscle.[2] Peripheral vasoconstriction also may damage facial muscles, at least in cold weather, as the following case-report suggests. The patient is a 44-year-old male, who had been healthy until the spring of 1979, when hypertension was detected. Sotalol (160 mg once a day) was prescribed and, on regular treatment, the blood pressure was controlled. In March 1980 the patient was skiing for four hours on the ice of the sea. He was an eager rambler and was used to skiing in any weather during the winter. The temperature outside was 10°C below zero and the wind was cold. The patient felt exceptionally cold on the arms, up to the elbows, in the thighs and on the face, but no frost-bite developed. One week later his face became swollen and penicillin and naproxen were prescribed. After two weeks of treatment the patient recovered. In June 1980, however, the patient's face became atrophic. On examination in November 1980, the physical and neurological findings were normal, except that the cheeks were obviously atrophic and there were dimples on them. The patient had also difficulty in opening his mouth completely, indicating weakness of the lateral pterygoid muscles. A blood count, blood glucose, serum CK and aldolase were normal. EMG of the facial and left arm muscles also was normal. No findings of a general muscle disease or of a peripheral neuropathy were detected.

We suggest that the facial oedema, and the atrophy which developed later, resulted from an ischaemic lesion of the facial muscles caused by the combined vasoconstrictive effect of coldness and the beta-blocking drug, sotalol. Patients taking beta-blockers should be informed of the possibility of "frost-bite" on the face with a permanent harmful cosmetic sequel.

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Treatment of acute exacerbations of multiple sclerosis with intravenous methylprednisolone

Sir: The role of steroid treatment in acute exacerbations of multiple sclerosis has been examined in several studies using regimes consisting of intramuscular ACTH[1] or oral steroids.[2] Recently, short-term, high dose intravenous steroid has been used in acute exacerbations of diseases such as systemic lupus erythematosus and acute nephritis,[3 4] without the side effects normally associated with chronic corticosteroid administration. Downing, Bosch, and Cook[5] reported encouraging results with such a regime in acute exacerbations of multiple sclerosis. We have observed a remarkable clinical response to high-dose intravenous methylprednisolone in six consecutive patients with severe acute exacerbations of this disease.

Six consecutive patients aged 18-53 years were studied. The diagnosis of multiple sclerosis was definite in five, probable in one. The disease had been active for from one month to eight years. In each patient an acute exacerbation of 24 hours to three weeks' duration necessitated admission to hospital. Four patients had had previous exacerbations of comparable severity; these had been treated with bed rest or ACTH. The mean disability score[6] before treatment was 7·3, and 10 days later 4·2. (table). Each of these patients improved within a few hours of beginning intravenous steroids. The rapidity of this improvement is illustrated in the following case report of one of these patients.

A 22-year-old man had noted numbness in his left arm with some blurring of visition five years before admission. These symptoms cleared within a month, but since that time he had had four similar exacerbations. Three weeks before admission he found that his left leg had become weak. A week later his right leg became weak, and numbness developed in the legs and trunk, with a level at T6. He was treated at home with a two-week course of daily intramuscular ACTH (60 units) injections without effect. On admission there was bilateral Grade 1 horizontal nystagmus. The left arm was ataxic. There was a spastic paraparesis, more marked on the left than on the right, with hyperreflexia and bilateral extensor plantar responses. Pin prick sensation was absent below the knee on the left and to a level at T3 on the right, with sacral sparing. Vibration sense was absent to the anterior superior iliac spine bilaterally, and position sense was absent in both legs. He could walk a few yards with assistance. He was treated with intravenous methylprednisolone 1 g daily, and with graded physiotherapy. He improved overnight, and a week later co-ordination was normal in his arms, and strength was normal in his legs. He could walk unaided. Spasticity was no longer evident, but the plantar responses remained extensor. Minimal ataxia was found in both legs. The sensory level was now at T11 on the right, and on the left only the L5 dermatome was abnormal.

Because of the fluctuating course of multiple sclerosis assessment of the effectiveness of any therapy is fraught with difficulty.[8] Acute exacerbations of multiple sclerosis have differing durations and outcomes,[9] and it is possible that the improvement in our patients was fortuitous and would have occurred without methlyprednisolone treatment. However, the rapidity and extent of the improvement including reversal of weakness, ataxia and sensory disturbances, was quite unexpected when compared with that which commonly occurs spontaneously or after ACTH treatment. Indeed, in one patient treatment with ACTH had been ineffective. All the patients thought that they had improved more quickly with intravenous methylprednisolone than with previous regimes.

The mechanism by which intravenous

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

Case	Age (yr)	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 [6] 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 this 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 develops 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 Markham[3] 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