

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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References

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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 ACTH¹ 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 Cook⁵ 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 score⁶ 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 vision 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.⁶ 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 methylprednisolone 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