occurs, measuring creatine kinase concentration may give a better 'overall' impression of the muscle damage than do myoglobin levels.

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Ankylosing spondylitis and middle ear impairment

Sir, The publication in your journal of a case report by Magaro et al, in which a middle ear conductive impairment was observed in association with ankylosing spondylitis (AS), drew our attention to the subject. We studied the auditory function of 94 ears from 48 patients with AS diagnosed according to the New York diagnostic criteria. There were nine women and 39 men, with ages ranging from 15 to 58 years, and an average age of 35 years. In all of them otoscopy, Weber’s test, Rinne’s test, pure tone audiometry, verbal auditory, tympanometry, and a stapedial reflex test were performed. In some patients a brain stem evoked response audiometry (BERA) examination was made. Ears in which previous disease could affect the results were rejected (two ears with chronic otitis media). We did not find any middle ear impairment, but sensorineural hearing loss was found more frequently in patients with AS than in the control population.

We think the results presented by Magaro et al do not provide sufficient evidence for concluding that the conductive defect was due to involvement of the fibrocartilaginous articulations between malleus and incus or incus and stapes and not to the stapes fixation seen in otosclerotic ears. The case presented by Magaro et al could be common otosclerosis in a patient with AS. An exploratory tympanotomy should make it clear which structures are responsible.

Our study suggests that middle ear involvement either does not occur in patients with AS or is very uncommon (less than 1% of ears in patients with AS). On the other hand, the sensorineural component deserves further research, and this is being conducted in our hospital.

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Thyroid disorders in systemic lupus erythematosus

Sir, The observations by Goh and Wang in the Annals that the prevalence of thyroid disorders is greater in a population of Malaysian patients with systemic lupus erythematosus (SLE) than in the general population are confirmed by our own experience. In a recent survey in Oxford of 64 patients with SLE (61 female, three male) 10 women were found who had also suffered clinical thyroid disease. Seven had had thyrotoxicosis, predating the diagnosis of SLE in six by one to 17 years. All were treated with either carbimazole or Neo-Mercazole, and two required radiolabelled iodine for subsequent flares. Three patients had suffered hypothyroidism, predating the SLE in one, and were treated with thyroxine. Thyroid antibody levels were not available.

The prevalence of overt thyroid disease in our group was 11–5% for thyrotoxicosis and 4–9% for hypothyroidism in women, compared with 1–9 and 1–0% respectively for a British population. There were no clinical features of this subgroup which distinguished them from the total SLE population. Goh and Wang suggest a possible association between the antibodies responsible for the false positive Wassermann reaction and the presence of thyroid disease. None of our thyroid subgroup had a false positive Wassermann reaction, and the frequency of anticardiolipin antibody was no different in these patients from that in the total group. It is possible that the thyroid disorders were related to the presence of thyroid stimulating and inhibiting immunoglobulins which have been demonstrated in patients with SLE.

Two patients showed a third autoimmune disorder; pernicious anaemia in one and Addison’s disease in the other. Among the SLE patients without overt thyroid disease were one with insulin dependent diabetes mellitus and one with myasthenia gravis, predating the SLE by 10 and eight years respectively. Clearly there is considerable overlap between the organ specific and non-organ specific autoimmune disorders.

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Treatment of Wegener’s granulomatosis with cyclosporin

Sir. Cyclosporin has been used successfully as an immunosuppressive drug in allografted patients. In addition, data have been published on the beneficial influence of cyclosporin in autoimmune diseases such as uveitis,1 myasthenia gravis,2 and polymyositis.3 Furthermore, there is evidence for the effectiveness of cyclosporin in patients with diabetes mellitus type I.4

In this report we present a patient with Wegener’s granulomatosis in whom conventional therapy with cyclophosphamide and prednisone failed. With the administration of cyclosporin, however, remission of the disease was achieved.

In January 1986 a 61 year old man was admitted to our hospital for evaluation of progressive respiratory distress, cough, and severe sinusitis with rhinorrhoea. Eleven months earlier the diagnosis of Wegener’s granulomatosis had been made elsewhere on the basis of clinical symptoms and characteristic histopathological findings of an extensive destructive granulomatous reaction with necrotising vasculitis of the upper and lower respiratory tracts. Consequently, treatment with cyclophosphamide (150 mg/day) and prednisone (40 mg/day) was started. At that time no renal involvement was found. Although initially this treatment appeared to be successful, his complaints about dyspnoea and pain behind his right eye were progressive in November 1985. A computed tomographic (CT) scan showed severe destruction of the right concha inferior and sinus maxillaris and part of the right orbita. For this reason the doses of cyclophosphamide and prednisone were increased to 200 mg and 100 mg a day respectively. Nevertheless, his condition deteriorated further, which made admission to hospital necessary in January 1986. On admission he was very dyspnoeic at rest, with severe rhinorrhoea and nasal discomfort. Laboratory studies, including erythrocyte sedimentation rate, C reactive protein, liver and kidney function, were normal. There were no signs of infection. An x-ray examination of the chest showed the presence of bilateral fibrosis without hilar adenopathy. Examination of pulmonary function showed obstructive abnormalities. Review of the nasopharynx and lung biopsy specimens taken earlier confirmed the diagnosis of Wegener’s granulomatosis. Since the disease appeared to be progressive despite optimal therapy we decided—after informed consent of the patient—to start treatment with cyclosporin (10 mg/kg body weight/day). At the same time cyclophosphamide was stopped and prednisone tapered off slowly. He has now been receiving cyclosporin and a low dose of prednisone for nine months and is doing very well. Rhinorrhoea and nasal discomfort have disappeared completely and dyspnoea becomes manifest only on major physical exercise; he is even able to cycle without difficulty. A control CT scan showed that the process of destruction of the upper airway organs and orbita has been stopped. Trough levels of cyclosporin (using the high performance liquid chromatography method) range from 140 ng/ml to 180 ng/ml, measured in whole blood. Serum creatinine has been increased slightly to 130–150 μmol/l (normal <120 μmol/l) during treatment.

In this case cyclosporin appears to have been a very effective drug in the treatment of Wegener’s granulomatosis. We suggest, therefore, that this drug be considered as an alternative to cyclophosphamide in patients who are resistant to conventional therapy or suffer severe side effects.

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