Variance—Abnormal variance of fibre size within muscle types was found in eight cases.

Discussion

The few studies on skeletal muscle in acromegaly have presented conflicting results. Lundberg *et al*⁷ found no significant changes in any patient whom they investigated. In another series⁸ clinical and electromyographic evidence of myopathy was found in five patients, but the muscle biopsy specimens were reported as normal. Using a histochemical technique closer to our own, Mastaglia *et al*^{9, 10} found type 1 hypertrophy and type 2 atrophy, but they did not attempt to correlate the results with individual growth hormone levels.

Our commonest finding was hypertrophy of type 1 fibres and atrophy of type 2 fibres, particularly type 2A. Atrophy was not that of simple disuse, which affects predominantly type 1 fibres,¹¹ while the hypertrophy was not that seen after exercise, which is most often present in type 2 fibres.⁶ Hypertrophy also did not seem to be due to compensation for the loss of function in the smaller atrophic fibres, since in seven of the 10 patients with hypertrophy there was no atrophy of the other fibre types. In view of the experimental findings of Greenbaum and Young¹ hypertrophy is probably a direct result of excess growth hormone secretion.

Whether increased growth hormone levels are also directly responsible for the atrophic appearances is not clear. Although atrophy probably follows hypertrophy, in six patients the picture was that of atrophy only. It is perhaps significant that of the two patients with normal biopsy findings one had only slightly raised hormone concentrations and the other was clinically little affected by the disease, which appeared to be of recent onset. The duration of the disease would seem to be more important than the actual height of the growth hormone concentrations, which did not correlate directly with the muscle appearances. Mastaglia¹⁰ has suggested that a fall in growth hormone levels

produced by treatment may be responsible for the atrophic changes, but our findings do not support this view since hypertrophy was maintained in four patients despite a prolonged period during which normal levels had been restored.

The possible relation of the skeletal muscle changes to acromegalic cardiomyopathy is important and requires further study. In the only patient in our series who died from heart failure due to cardiomyopathy (case 4) the skeletal muscle changes shortly before death were those of atrophy with considerable variation in type 1 fibres. An earlier biopsy specimen from this patient had shown considerable phagocytosis of individual muscle fibres indicative of severe myopathy. We have no evidence that restoring normal growth hormone concentrations can reverse the muscle abnormalities, since even a prolonged period of normal hormone concentration was still associated with considerable changes. Nevertheless, the history of the condition can be ascertained only by serial sampling, and the relatively atraumatic needle biopsy technique is particularly suitable for this purpose.

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SHORT REPORTS

Aortic incompetence in systemic lupus erythematosus

Aortic incompetence as a sole manifestation of endocarditis is uncommon in patients with systemic lupus erythematosus (SLE).¹ We report a case where this developed in a patient with SLE treated with corticosteroids.

Case report

An obese 28-year-old woman with a previous history of polyarthralgia was seen by a psychiatrist in 1969 when she complained of depression. She was treated with amitriptyline and diazepam. Two months later she developed fever, polyarthralgia, a butterfly rash, diffuse alopecia, and pericarditis. Her blood pressure was 120/80 mm Hg. Investigation showed haemoglobin 10 g/dl, erythrocyte sedimentation rate 100 mm in first hour, LE cells positive, and antinuclear antibody and Rose-Waaler tests both positive. She was given 60 mg prednisone orally per day. Her illness subsided, and the prednisone was gradually reduced to a maintenance dose of 10 mg daily. A minor exacerbation of her articular symptoms in July 1971 necessitated increase of prednisone to 15 mg daily for one month.

In September 1973 she was asymptomatic, but a high pitched early diastolic murmur behind the left sternal edge in the third and fourth intercostal spaces was heard, and the blood pressure was 195/60 mm Hg. The haemoglobin level was 11.9 g/dl, the Wasserman reaction was negative, and blood cultures were negative. In October 1975 exertional dyspnoea developed, the diastolic murmur was found to have persisted, and electrocardiography showed left ventricular hypertrophy. In December 1975 the exertional dyspnoea was increasing, and an ejection systolic murmur in addition to the

diastolic murmur was heard at the left sternal edge. Cardiac catheterisation confirmed the presence of aortic incompetence without stenosis. Cardiac cineangiography showed moderately severe aortic regurgitation with satisfactory left ventricular function, good movement of the aortic valve cusps, and some dilatation of the ascending aorta. Treatment with 10 mg prednisone and a diuretic was continued. When reviewed in March 1976 she continued to do well.

Discussion

This 28-year-old patient developed SLE in 1969 and was treated with corticosteroids. Four years later despite apparent reasonable control of the disease she developed aortic incompetence with no other demonstrable valvular defect. Aortic incompetence was not explicable on the grounds of cardiac failure secondary to anaemia, nor were the length of the subsequent history or the results of investigations compatible with a diagnosis of subacute bacterial endocarditis. The patient did not have ankylosing spondylitis, and rheumatic heart disease seems a less likely diagnosis with neither evidence of other valvular involvement nor history of acute rheumatic fever previously. It seems most likely that this patient developed aortic incompetence as a complication of her SLE. When endocardial involvement by SLE occurs it most commonly effects the mitral valve,^{1, 2} and aortic incompetence without mitral valve disease in SLE is extremely rare. Bernhard *et al*³ reported three such patients, while Schulman and Christian⁴ described three cases with aortic incompetence and mitral valve disease. Aortic incompetence in our patient developed despite apparent control of other features of SLE and underlines the suggestion³ that corticosteroid therapy may play a part in the development

of these valvular defects. However, our patient continues to thrive on a low dose of prednisone and diuretics.

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³ Schulman, H J, and Christian, C L, *Arthritis and Rheumatism*, 1969, **12**, 138.

⁴ Bulkley, B H, and Roberts, W C, *American Journal of Medicine*, 1975, **58**, 243.

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Severe bronchoconstriction provoked by sodium cromoglycate

The inhalation of sodium cromoglycate (SCG) is an effective and widely used treatment for asthma. Rarely, reactions to this drug, such as pulmonary eosinophilia, allergic granulomatosis, anaphylaxis, angioneurotic oedema, urticaria, and skin eruptions, have been reported.¹ Although mild wheeze may develop in some patients after inhaling SCG, in only one patient has the provocation of severe bronchospasm been fully documented.² A further patient who developed severe bronchoconstriction after inhaling SCG is described in this report. In this patient bronchospasm was prevented by the administration of salbutamol and an antihistamine but not by atropine.

Case report

A man aged 42 years with chronic asthma was treated with SCG, 20 mg four times daily for 12 months, during which time there was clinical improvement. SCG was then replaced by a corticosteroid aerosol. After 15 months SCG was restarted because of recurrence of asthma. A month later he suddenly became very breathless and wheezy immediately after inhaling

SCG. Treatment with this drug was then stopped and oral prednisolone substituted. While this suspected reaction to SCG was being investigated, maintenance treatment consisted of beclomethasone 100 µg four times daily and prednisolone 5 mg once daily.

Prick skin testing showed that the patient was atopic. The serum IgE concentration was, however, normal. The results of patch and prick skin testing with solutions of SCG were negative. The changes in forced expiratory volume in one second (FEV₁) after inhalation of 20 mg of SCG with the lactose base and the lactose base alone via Spinhaler are shown in the figure. Five minutes after inhalation of the active preparation the FEV₁ fell from a pretreatment level of 2.75 l to 0.7 l. At ten minutes the FEV₁ was 0.6 l and since he was in considerable respiratory distress salbutamol by intermittent positive-pressure ventilation (IPPV) was given. There was an immediate improvement and the FEV₁ rose to 2.75 l. After inhalation of the lactose base alone a slight fall in FEV₁ from 2.9 l to 2.3 l was observed. The figure also shows the changes in FEV₁ when salbutamol, chlorpheniramine, and atropine were administered before 20 mg of SCG was inhaled. An aerosol of an 0.5% solution of salbutamol given by IPPV 20 minutes before the SCG prevented a fall in FEV₁, which in fact increased from 2.4 l to 4.0 l. No significant changes in FEV₁ were observed when chlorpheniramine (10 mg) was given intramuscularly 40 minutes before the inhalation of SCG. Nevertheless, atropine (1.2 mg) given intramuscularly 40 minutes before SCG failed to prevent a dramatic fall in FEV₁ from 3.5 l to 0.9 l. Again, severe breathlessness was promptly relieved by the administration of salbutamol by IPPV.

Discussion

A profound fall in FEV₁ associated with distressing breathlessness was observed in this patient after the inhalation of SCG but not after inhalation of the lactose base alone. Similar responses were seen in the patient described by Muller and Kowalski.² In their patient, however, the fall in FEV₁ was prevented by prior intramuscular injection of 1 mg of atropine and it was, therefore, postulated that bronchoconstriction was reflexly mediated through the vagus nerve. In our patient a fall in FEV₁ was not prevented by atropine. Chlorpheniramine, however, prevented the fall in FEV₁, which suggests that histamine release may have been involved in the development of the bronchoconstriction provoked by the inhalation of SCG. If that is so, this observation is surprising because of the widely accepted view that the beneficial effects of SCG in asthma are due to prevention of release of histamine and other substances from mast cells in the bronchial wall.³

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Changes in FEV₁ after inhalation of SCG and lactose base alone, and the effect of prior treatment with salbutamol, chlorpheniramine, and atropine.

Terminal ileitis due to *Yersinia pseudotuberculosis*

Yersinia pseudotuberculosis has been recognised as a cause of mesenteric adenitis and terminal ileitis for many years.¹ Most cases present with a short history suggestive of appendicitis, the true diagnosis being made at laparotomy.²⁻⁴ Our patient, however, had had symptoms for several months that clinically simulated Crohn's disease. It is important to distinguish yersinial ileitis from Crohn's disease as the prognosis is very different.

Case report

A 24-year-old clerical worker was admitted with a three-day history of colicky lower abdominal pain, nausea, and fever. For six months he had