Discussion

These results show that AH 7725 when given by mouth can inhibit the immediate bronchial response to inhaled antigen in asthmatic patients. It has no bronchodilator effect (Gayrard, 1974) and its activity is most likely to be explained by the inhibition of release of mediators of immediate-type allergic responses, which has already been shown in laboratory work.

AH 7725 has similar inhibitory properties to those of disodium cromoglycate and also to those of its own predecessors AH 6556 and AH 7079 (Assem, 1973; Assem and McAllen, 1973 b). It is, however, exceptional in being active when given by mouth. So far as we know this is the first report on a drug for the prophylaxis of asthma which is active when given by mouth.

Our results encourage the hope that AH 7725 has a potential in future clinical practice. Many asthmatic patients would prefer to take a prophylactic drug by mouth rather than by inhalation, and this new compound may be of particular value in very young children with asthma and in those adults who cannot master the necessary inhalation technique for using disodium cromoglycate. The possible use of AH 7725 in allergic conditions which require systemic treatment awaits investigation.

We wish to thank Dr. E. P. Woodings and Allen & Hanburys Limited for supplies of AH 7725 and for allowing us to report the results on the plasma levels of this drug.

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MEDICAL MEMORANDA

Mitral Regurgitation due to Rupture of Chordae Tendineae by Calcified Atrial Myxoma

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Patients with left atrial myxoma characteristically present with a history of progressive shortness of breath, often punctuated by syncopal attacks, and physical signs of mitral valve obstruction (Goodwin, 1963; Greenwood, 1968; Harvey, 1968). Predominant or "pure" mitral regurgitation due to left atrial myxoma has been reported but is uncommon (Cohen et al., 1963; Penny et al., 1967; Wittenstein et al., 1959). This report describes a patient with a calcified left atrial myxoma who presented with sudden severe mitral regurgitation due to ruptured chordae tendineae.

Case Report

A 44-year-old machinist, previously well and active, abruptly developed dyspnoea and orthopnoea during the first week of December 1972. He was admitted to his local hospital where he was found to have cardiac enlargement, pulmonary oedema, and bilateral pleural effusion. The electrocardiogram showed sinus rhythm with evidence of left atrial enlargement. A heart murmur had been heard one year earlier, but he was told "not to worry" about his heart. There was no history of rheumatic fever, chest pain, calf tenderness or swelling, or fever or chills. Digitalis (digoxin 0.25 mg/day) and a diuretic (Lasix

40 mg/day) were given but dyspnoea, orthopnoea, and fatigue persisted, atrial fibrillation developed, and he was referred for consultation.

On examination he was very thin and dyspnoeic at rest. Atrial fibrillation was present, the blood pressure was 110/60 mm Hg, there were no signs of peripheral embolism, and the optic fundi were normal. The carotid pulse was small and rapid, the venous pressure was raised, and there was prominent systolic pulsation of the neck veins. The apex beat was in the mid-clavicular line and there was a prominent, diffuse systolic pulsation in the third to fifth interspaces midway between the sternum and the apex. The pulmonary valve closure sound was increased and the aortic component normal. A loud first heart sound was followed by a grade III/VI regurgitant murmur heard best at the apex. There was a prominent early third heart sound and a faint mid-diastolic apical murmur. The lungs were clear. The liver was palpable 2 cm below the right costal margin in the mid-clavicular line. Chest x-ray examination showed enlargement of the left ventricle and left atrium, pulmonary venous congestion, and costophrenic septal (Kerley's B) lines.

After some initial improvement progressive dyspnoea and orthopnoea had recurred after three months. Cinefluoroscopy showed a mobile, calcified mass in the left atrium which appeared to enter and partially obstruct the mitral valve orifice during diastole. Pulmonary capillary wedge pressure and right heart pressure were raised (see table) and there was evidence of mitral and tricuspid regurgitation. At operation the left atrium was found to contain a large, calcified myxoma (see fig.) attached by a 1-cm pedicle to the atrial septum just below the limbus of the fossa ovalis. The mitral valve leaflets were thickened and damaged and many chordae tendineae were ruptured.

Cardiac Catheterization Data

Site of Sample	Oxygen Saturation (%)	Pressure (mm Hg)
Pulmonary capillary (wedge) . .	—	V — 45 (mean* 30)
Pulmonary artery	40, 41	85/45 (mean 44)
Right ventricle (body)	36, 37	85/10, Right ventricle end diastolic pressure — 20
Right ventricle (infundibulum)	37, 38	—
Right atrium	34	C — 25 } (mean 18)
Radial artery	—	V — 23 } (mean 80)
		110/70 (mean 80)

*Mean pressures are derived from the phasic pressures by electronic integration.

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Lobulated calcified myxoma (4 x 5 cm) removed from left atrium.

The myxoma was excised and the mitral valve replaced with a No. 3 Starr-Edwards prosthesis (6120). Convalescence was uneventful and subsequent cardioversion restored sinus rhythm.

Comment

Harvey (1968) used the term "wrecking ball" to describe the movements of a calcified pedunculated right atrial myxoma, likening it to the swinging iron ball suspended from a crane used for demolishing buildings. In the patient he described the tricuspid valve had been almost completely destroyed by the movements of the tumour. Similar patients with incompetent tricuspid valves damaged by calcified right atrial myxoma have been reported by Oliver and Missen (1966) and Fluck and Lopez-Bescos (1968). Predominant mitral regurgitation, though unusual, has been described in a few patients with myxoma of the left atrium (Cohen *et al.*, 1963; Penny *et al.*, 1967; Wittenstein *et al.*, 1959). It was attributed to partial herniation of the tumour through the mitral valve interfering with valve closure. Ruptured chordae tendineae were not seen. The present patient, unlike most others with left atrial myxoma, presented with a sudden onset of severe mitral

regurgitation and pulmonary oedema. Probably the chordae tendineae ruptured at that time and the rupture was related to the "wrecking ball" effect of the heavily calcified, mobile tumour. The sudden onset of mitral regurgitation suggested ruptured chordae tendineae but atrial myxoma was not considered.

A variety of auscultatory findings have been described in patients with left atrial myxoma, including systolic and diastolic apical murmurs, accentuated pulmonary valve closure sounds, ejection sounds and diastolic filling sounds (Goodwin, 1963; Greenwood, 1968; Harvey, 1968). The diastolic filling sound, the so-called "tumour plop," occurs earlier in diastole than the usual third heart sound, near the timing of the opening snap of the mitral valve with which it is sometimes confused (Greenwood, 1968; Abbott *et al.*, 1962; Pitt *et al.*, 1965). This early diastolic sound is thought to be produced by the sudden deceleration of the tumour on its pedicle as it enters the left ventricle (Pitt *et al.*, 1965). The "third heart sound" heard in our patient was quite early and may have been produced in this way. Though the combination of ruptured chordae tendineae and left atrial myxoma has not been previously reported the association should not be unexpected, especially when the tumour is calcified. While some mitral regurgitation can be caused by a left atrial myxoma, the sudden appearance of severe regurgitation would be unusual and misleading. The presence of an early third heart sound ("tumour plop") may be a clue in this situation, but it is difficult to distinguish from the usual third heart sound heard in some cases of mitral regurgitation.

The patient was referred to Dr. Peter M. Yurchak at the cardiac unit, Massachusetts General Hospital.

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Testicular Torsion in Henoch-Schonlein Syndrome

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The Henoch-Schönlein syndrome or anaphylactoid purpura is common in children. The presenting symptoms are non-thrombocytopenic purpura, arthralgia, abdominal pain, and sometimes renal abnormalities similar to those of poststreptococcal glomerulonephritis (Silber, 1972). Uncommon and atypical clinical presentations have been reported, including facial nerve palsy due to oedema and convulsions, and hemiparesis and coma due to hypertensive encephalopathy (Silber, 1972). Gastrointestinal complications have also been reported and include chronic gastrointestinal obstruction, perforation, massive haemorrhages, bowel necrosis, (Silber, 1972), and intussusception (Gairdner, 1968). Roy (1972) has reported a case of steatorrhoea and malabsorption complicating the Henoch-Schönlein syndrome. Fitzsimmons (1968) described two boys with testicular pain and scrotal swelling, a complication which had not been reported previously. Sahn and Swartz (1972) found testicular involvement in 38% of their 20 cases. We report an unusual complication of testicular involvement.

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Case Report

A 4½-year-old boy was admitted from the casualty department on 15 September 1973 with a painful swelling on the left side of the scrotum of four-hours duration. Haemorrhagic spots had appeared on both ankles on 12 September 1973, followed by

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