unable to inhale correctly from a pressurised cannister were asked to use the Spinhaler 43 (95.5%) had no difficulty in using this device.

Efficiency of technique in using aerosol inhalers in 321 asthmatic patients

		Females	Males	Total	
				No	%
Efficient		160	116	276	85.98
Doubtful		12	7	19	5.92
Inefficient		23	3	26	8.10

Discussion

In spite of careful tuition before the prescription of drugs by aerosol, 45 (14%) of our patients used their inhalers inefficiently. It is not always possible to take the time to teach patients how to use a pressurised aerosol, and the proportion of "untaught" patients not using their inhalers correctly must be much greater than that found in this study. Now that corticosteroids by aerosol are being used extensively in the treatment of bronchial asthma, it is of paramount importance that patients should be taught how to use their inhalers. This study, however, indicates that even after initial tuition some patients cannot use an inhaler correctly, and regular checks of inhalation technique are therefore necessary. We have now adopted the practice of checking every patient's technique at every visit to the asthma clinic. This is done by the respiratory technician at the time ventilatory function tests are recorded, since we now consider that it is as important to check inhalation technique as it is to measure ventilatory function.

The fact that 95.5% of the patients unable to inhale correctly from a pressurised cannister could use a Spinhaler efficiently confirms our clinical impression that patients have little difficulty in using a Spinhaler whereas many have problems with the pressurised cannister. There must be many asthmatic patients who are being denied the benefits of corticosteroid and bronchodilator therapy by inhalation because they cannot use a pressurised inhaler correctly.

The results of this small study suggest that many patients would benefit if corticosteroids and bronchodilators were made up in dry powder form for inhalation with a device like the Spinhaler. Another advantage of this method of administering drugs to the bronchi would be the reduction in fluorocarbon aerosol propellants inhaled by patients since there still remains slight doubt about the safety of these compounds.²

We thank our colleagues Miss Mary Cursiter, Miss Shirley Armstrong, Mrs Linda Sutherland, and Mr William Witherspoon for their help in this study and Miss I A McCall for typing the transcript.

¹ Saunders, K B, *British Medical Journal*, 1965, 1, 1037.
² *Lancet*, 1975, 1, 1073.

Respiratory Unit, Northern General Hospital, Edinburgh

I C PATERSON, MB, MRCP, registrar
G K CROMPTON, MB, FRCPD, consultant physician

Addison's Disease presenting with chronic low backache

Though abdominal pain and muscle cramps are recognised in Addison's disease¹ persistent low backache is not a feature. We describe here a patient with Addison's disease who presented with chronic low backache which steroid replacement treatment rapidly relieved.

Case Report

A 42-year-old man presented with a nine-months history of low backache of gradual onset, continuous by day and often disturbing his sleep. It

had become extremely severe, resulting in prolonged absence from work. He further admitted to one year of anorexia, weight loss, and depression. Examination showed loss of lumbar lordosis and painful limitation of lumbar spinal movements.

Haemoglobin, white cell count, and erythrocyte sedimentation rate (ESR) (Westergren) were normal. X-ray films showed disc space narrowing at L 4-5 and L 5-S 1 with osteophytes, but no evidence of spondylitis, sacroiliitis, or tuberculosis.

Disc degeneration was diagnosed and an Oswestry-Goldthwaite support prescribed. As there was no improvement he was referred to the rheumatology clinic two months later, where additional history ascertained included weakness and lassitude for a year, postural giddiness, and increased pigmentation. Examination showed pigmentation, especially in the palmar creases and on the lips. Blood pressure was 100/70 mm Hg.

Investigations showed haemoglobin 13.2 g/dl; white cells $7.4 \times 10^9/l$ ($7400/mm^3$), 28% neutrophils, 66% lymphocytes; ESR 9 mm; he gave a negative Rose-Waaler test result and was negative for antinuclear factor. Serum sodium was 135 mmol/l (135 mEq/l) (normal 135-145 mmol/l), potassium 4.5 mmol/l (4.5 mEq/l) (normal 3.6-5.0 mmol/l) and blood urea 7.9 mmol/l (48 mg/100 ml) (normal 2.5-7.1 mmol/l). Serum calcium, phosphorus, and alkaline phosphatase were normal. X-ray examination of chest, spine, and sacroiliac joints provided no fresh information. No adrenal calcification was seen. Urinary oxogenic steroids were 16 μ mol/24 h (4.6 mg/24 h) (normal 30-60 μ mol/24 h (9-17 mg/24 h)). Plasma 11-hydroxycorticosteroids were 50 nmol/l (1.8 μ g/100 ml) (normal 200-650 nmol/l (7-24 μ g/100 ml) at 9.00 am) and 30 minutes after 250 μ g of tetracosactrin intramuscularly were 60 nmol/l (2.2 μ g/100 ml).

Addison's disease was diagnosed and treatment started with cortisone acetate 37.5 mg/day and fludrocortisone 0.1 mg/day. His backache disappeared overnight and had not recurred five months later. He regained energy, appetite, and weight. His pigmentation faded and his blood pressure was 130/80 mm Hg. No change has occurred in disc space width at the levels of disc degeneration.

Discussion

Except for the backache, the history, signs, haematological, and biochemical findings were typical of Addison's disease. The subnormal plasma and urinary steroids with absence of response to adrenal stimulation confirmed the diagnosis.²

The backache was of the type seen with lumbar disc disease and radiographic evidence supported this diagnosis. The lack of response to symptomatic treatment and the dramatic relief of pain by corticosteroid replacement suggested that adrenal cortical insufficiency was responsible. Chronic low backache is, however, not typically found in Addison's disease. There was no evidence to suggest inflammatory joint disease, metabolic bone disease, or spinal tuberculosis.

Possibly the pain was related to disc degeneration and caused by muscular weakness of corticosteroid insufficiency adversely affecting the supportive action of the paraspinal musculature. Steroid treatment, abolishing this weakness, resulted in relief. Alternatively, loss of body water due to Addison's disease may have caused dehydration of the discs, exaggerating the loss of volume and support of the degenerate discs. Steroid therapy might have restored some volume to the disc, thus relieving pain. Dessication of the disc is an accompaniment of disc degeneration,³⁻⁵ and excised discs have affinity for water.⁴ This hypothesis, however, was not supported by an increase in disc space width at the appropriate levels three months after starting therapy. It is arguable that the response to treatment was too rapid for these explanations to suffice and either there is another explanation linking disc degenerative symptoms with adrenal cortical insufficiency or the pain was unrelated to the disc changes and was simply an unusual form of the muscular pain that may occur in Addison's disease.

¹ Rowntree, L G, and Snell, A M, *A Clinical Study of Addison's Disease*. Philadelphia and London, Saunders, 1931.

² Wood, J B, et al, *Lancet*, 1965, 1, 243.

³ Keys, D C, and Compere, E L, *Journal of Bone and Joint Surgery*, 1932, 14, 897.

⁴ Charnley, J, *Lancet*, 1952, 1, 124.

⁵ Hendry, N G C, *Journal of Bone and Joint Surgery*, 1958, 40B, 132.

Leeds (St James's) University Hospital, Leeds LS9 7TF

P SHERIDAN, MB, MRCP, lecturer in medicine, university department of medicine
C F V MURRAY-LESLIE, MB, MRCP, senior registrar in rheumatology
J R GOLDING, DM, FRCP, consultant rheumatologist and senior clinical lecturer