SHORT REPORTS

Patient error in use of bronchodilator metered aerosols

Aerosolised bronchodilator drugs in pressurised canisters delivering metered doses are widely used, easy to carry, and apparently easy to operate. The instructions with each canister are reasonably clear, stating that each puff must be deeply inhaled and the breath held afterwards. Since the drug intake depends mostly on the patient's ability to follow the instructions, we thought it was important to find out whether asthmatics used their aerosols correctly.

Patients, methods, and results

Twenty asthmatic patients (12 men, 8 women) with a mean age ($\pm$ SD) of 44.8 ± 15.1 were studied. All had been treated with bronchodilator sprays and knew how to operate them. They were not examined during an acute attack, although all of them had some bronchial obstruction when tested. They received no medication for 24 hours preceding the first test.

Specific airway resistance (S_{Raw}), which is the resistance corrected for lung volume,¹ was measured in a whole-body constant volume plethysmograph at a thoracic gas volume close to functional residual capacity, using the panting technique.² Each subject was tested, by different procedures, on two successive days between 2 and 5 pm if the initial value of S_{Raw} was comparable. On both days S_{Raw} was measured before and 5 and 10 minutes after bronchodilator aerosol inhalation (salbutamol 100 μ g/puff in 10 patients and fenoterol 200 μ g/puff in 10 others). Bronchodilation was greatest after 10 minutes, therefore only measurements taken at that time were used.

On the first day the patients were told to inhale two puffs of a bronchodilator spray in their customary way. On the second day two puffs of the same bronchodilator spray were administered by the physician. Wearing a nose clip, the patients opened their mouth wide, expired deeply, and then inspired deeply. One puff of drug was delivered at the beginning of the inspiration with the canister about 3 cm from the lips. After fully inspiring the patients closed their mouths and held their breath for four seconds. A second puff was administered in the same way. Repetition of the first-day procedure on different occasions by some patients showed there was no systematic error due to the tests not being randomised.

The bronchodilation achieved by the two procedures was similar in only 5 patients. In the 15 others it was greater when the aerosol was given by the physician. Observation showed that only the 5 patients in whom bronchodilation was similar by both methods correctly inhaled (that is, at the beginning of a deep inspiration) on self-administration. The other 15 patients failed either to inspire deeply or hold their breath afterwards, or both, or poorly co-ordinated the puff and the inspiration.

The table shows the mean values of S_{Raw} observed. The control values were similar on the two tests. After inhalation the mean decrease of control S_{Raw} was significantly greater with physician administration (-54%) than with patient administration (-45%).

Bronchodilation measured by specific airway resistance after patient administration and after physician administration of bronchodilator metered aerosol in 20 patients (mean values $\pm$ SEM)

Administration by:	S _{Raw} (cm H ₂ O $\times$ s)		Decrease	
	Control	10 min after inhalation	Absolute	%
Patient	22.6 $\pm$ 3.0*	12.3 $\pm$ 1.6	10.3 [†]	45
Physician	23.3 $\pm$ 3.1*	10.5 $\pm$ 1.4	12.8 [†]	54

*Difference between patient and physician administration not significant.

[†]Difference between patient and physician administration significant ($P < 0.001$ by Snedecor's f test).

Discussion

Penetration and deposition of aerosol is influenced by numerous factors, some related to the physical characteristics of the aerosol and others to the patient. Important among the latter are a deep inspiration to ensure deep penetration,³ and breath-holding to ensure good retention of particles.⁴ Only about 10% of a puff is inhaled,⁵ and in most cases, because of poor inhalation technique,

probably only a fraction of that. The bronchodilator drugs currently available are so active that even with minute doses airway dilatation is noticeable. Nevertheless, our findings show that the bronchodilation achieved by self-administration may be less than the maximum possible and that, in general, more careful inhalation yielded significantly better results. Since asthmatic patients seem either not to read or to be unable to follow the instructions supplied with bronchodilator metered aerosols we suggest that chest physicians should show their patients how to use the canister correctly and emphasise the importance of the proper method.

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Use of pressurised aerosols by asthmatic patients

Drugs administered by inhalation now play a major role in the treatment of bronchial asthma. It has been shown that some patients have difficulty inhaling drug aerosols from pressurised canisters¹ and this may, of course, be a reason for treatment failure. The aim of this study was to find the number of patients attending an asthma clinic who were not using their inhalers satisfactorily, in spite of apparently adequate tuition in the technique by members of the medical staff before treatment with drugs in aerosol form was started. We also assessed whether patients who were unable to use a pressurised inhaler correctly could use a dry powder inhaler (Spinhaler). The breath-actuated pressurised inhaler now marketed may be easier to use than the conventional pressurised canister, but since this device is not yet available for aerosol corticosteroid administration it was not included in this study.

Methods and results

A trained respiratory technician assessed 321 asthmatic patients (195 females and 126 males) in the use of their inhalers. All the patients were asked to use their inhaler under observation and their technique was classified as: (a) efficient; (b) doubtfully efficient; and (c) inefficient.

The results showed that 45 (14%) patients were unable to use their inhalers satisfactorily (see table). Of this total 26 (8.1%) were classified as inefficient and 19 (5.9%) as doubtfully efficient inhaler users. Thirty-five (77%) of the 45 patients were females and their average age was 56.5 years (range 14-75 years) compared with an average age for the male group of 49.4 years (range 13-82 years). The average age of the 10 male patients classified as inefficient or doubtfully inefficient was the same as that for the male group as a whole. When the 45 patients in categories 2 and 3 who were

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

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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.

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