SEVERE BRONCHOCONSTRICTION AFTER INHALATION OF BECLOMETHASONE AND BUDESONIDE

S C Poh
Y T Wang

Department of Medicine III
Tan Tock Seng Hospital
Moulmein Road
Singapore 1130

S C Poh, MB, MRCP (E)
Clin Assoc Professor and Senior Physician

Y T Wang, MB, MRCP, M Med
Physician

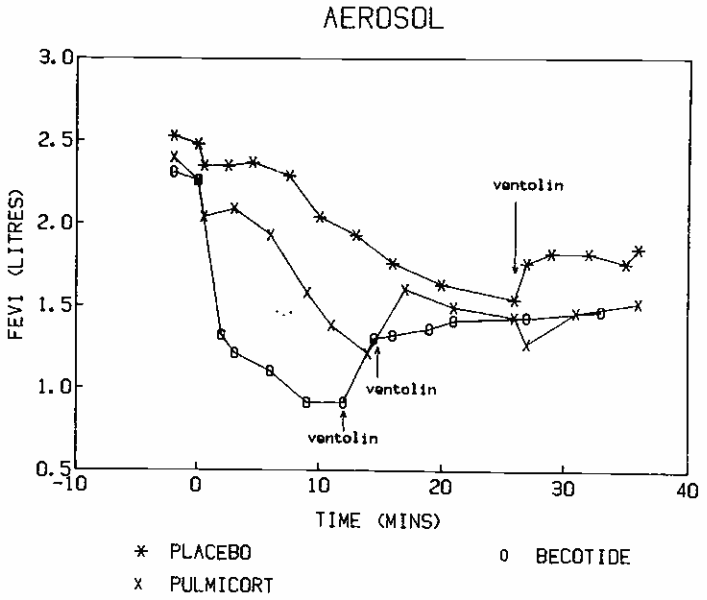
SYNOPSIS

Pressurised Freon-based aerosols containing sympathomimetic drugs and corticosteroids are now widely used in the management of bronchial asthma. Only four patients who developed severe bronchoconstriction following inhalation of a steroid aerosol have been reported. (1, 2) A further patient who developed severe bronchoconstriction after inhaling beclomethasone as well as budesonide is described in this report.

CASE REPORT

A 30 year old Caucasian housewife with mild asthma since the age of 15 years received regular treatment with a salbutamol pressurised aerosol (Ventolin) and a beclomethasone aerosol (Becotide) for nine months. She then noticed that the use of Becotide provoked fairly severe wheezing and was referred to one of us (SCP) by her family physician for further evaluation. She had previously been found to be atopic. She improved with prednisolone 10 mg every other day together with occasional puffs of Ventolin aerosol and remained well.

The following tests were made on different days in the respiratory function laboratory (her pre-challenge forced expiratory volume in one second [FEV_1] ranged from 2.31 L to 2.53 L on the different days): (1) the patient inhaled two puffs of Becotide from the same inhaler that she had used before. Within 10 mintues, she was dyspnoeic and very wheezy; the FEV_1 fell y 61% from 2.31 L, the forced vital capacity (FVC) by 53% from 3.25 L and the peak expiratory flow rate (PEFR) by 57% from 400 L/min. Her dyspnoea and expiratory flow rates improved rapidly following inhalation of a Ventolin aerosol. She was challenged on a separate occasion with a different Becotide aerosol and showed the same reaction; (2) the patient made two inhalations of the placebo aerosol which contained only the propellant vehicle. This caused the FEV_1 to fall by 39% (from a mean baseline of 2.53 L). The time course of the fall was slower and its severity was not as great as in test 1; (3) the patient inhaled two puffs of budesonide aerosol (Pulmicort). She became breathless and wheezy within 10 minutes. The fall in FEV_1, FVC and PEFR showed a similar pattern to that in test 1; (4) test 1 was repeated but on this occasion, the patient inhaled beclomethasone from a Becotide rotacap (containing beclomethasone and lactose base). Again she developed within 10 minutes dyspnoea and wheeze. Her FEV_1 fell by 46% from the stable basal value of 2.37 L, the FVC by 33% (from 3.47 L) and the PEFR by 32% (from 370 L/min.); (5) a challenge test from a placebo rotacap containing only the lactose base produced a fall in FEV_1 of 43% from an initial value of 2.45 L. The pattern of fall was similar to that of the placebo aerosol.



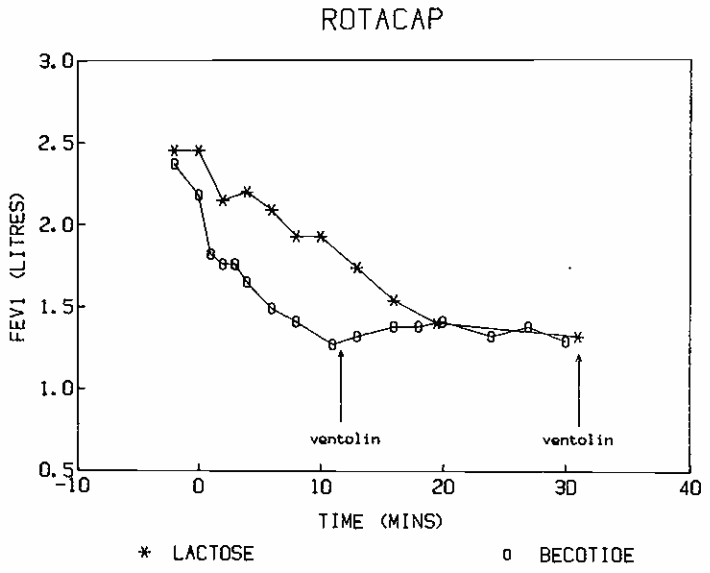


Fig. Changes in FEV_1 after inhalation of aerosol Becotide, placebo and Pulmicort and after rotacap Becotide and lactose base.

DISCUSSION

In 1969, Sterling and Batten (3) reported that both normal and asthmatic subjects showed a fall in specific airway conductance after the inhalation of aerosol propellant vehicle; they concluded that the slight bronchoconstriction was insufficient to cause symptoms of wheezing and was less than that caused by inhalation of a single cigarette. Three asthmatic patients who experienced increased breathlessness and wheezing after inhalation of Becotide were investigated by Bryant and Pepys. (1) They found that although a placebo aerosol (containing only the propellant vehicle trichlorofluoromethane and dichlorodifluoromethane) produced similar bronchoconstriction, inhalation of the gaseous fluorocarbons obtained by heating the liquid propellant to its boiling point or as a compressed gas failed to produce a reaction, indicating that other agents were responsible. These included extractives from rubber components of the metering value, contaminants from the aluminium can and surfactants (oleic acid in Becotide). Another asthmatic patient who developed bronchoconstriction after inhaling Pulmicort was reported by McGivern and Macfarlane. (2) Inhalation of a placebo aerosol (containing only the propellants trichlorofluoromethane, dichlorodifluoromethan and dichlorotetrafluoroethane) gave a similar but smaller reaction.

Our patient developed severe bronchoconstriction to both in the Becotide and Pulmicort aerosol and to the Becotide given via a rotahaler. The induced bronchoconstriction could be readily reversed by salbutamol from a pressurised aerosol supporting the suggestion that the presence of a bronchodilator in the aerosol would be sufficient to overcome the bronchoconstriction. (13) The cause of the more rapid and severe reaction to the inhaled steroids than to the placebo inhalation is uncertain and might be only a reflection of a greater response to the drugs.

This is the first reported case of severe bronchoconstriction after inhalation of Becotide (both as a pressurised aerosol and in a power form) as well as Pulmicort. It is likely that idiosyncrasy is responsible for the reaction.

ACKNOWLEDGEMENT

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