Exercise-induced asthma without respiratory heat loss

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It is well documented that exercise-induced asthma is more pronounced when inspired air is cold and dry rather than warm and humid.^{1,2} The severity of exercise-induced asthma is closely dependent on loss of respiratory heat, needed to warm and humidify inspired air,³ and it has been suggested that airway cooling is the only stimulus triggering postexercise bronchoconstriction.³⁻⁷ We present a case of an asthmatic patient who has repeatedly developed exercise-induced asthma while breathing air at body temperature and humidity with no respiratory heat loss.

Case report

The patient is a 13-year-old asthmatic boy with a known history of bronchial asthma and hay fever for the last nine years. He was referred to us after his mother had observed him suffering multiple episodes of wheezing and shortness of breath resulting from normal playground activities. Subsequently he has been treated successfully with beclomethasone dipropionate aerosol for his perennial asthma and with salbutamol aerosol for his exercise-induced asthma. During his investigation a series of exercise tests were performed to learn about his exercise tolerance under various air conditions.

The patient came to the laboratory after refraining from taking any drugs for at least 12 hours before the visit. He performed a forced vital capacity manoeuvre to determine his baseline lung function. He then started breathing warm humidified air supplied to him from a thermally insulated circuit through a low-resistance one-way valve. Warm humidified air was obtained by the use of a conventional nebuliser heater (Ohio Medical Products). The temperature of the air measured at the inspiratory port of the mouthpiece was 37.1°C fully saturated (water content 44.2 mg per litre of air). There were free water drops throughout all the tubing.

After he had breathed the gas for 10 minutes, forced vital capacity manoeuvres were repeated and the patient then proceeded to perform a standard six-minute exercise test on a cycle ergometer (Lode Instruments) while continuously breathing the warm humidified gas. During the exercise test expired air was passed through a mixing chamber and pneumotachygraph system coupled with gas analysers and a microprocessor (PK Morgan Exercise Test System). This enabled us to monitor ventilation and gas exchange every half minute.

Lung function was measured at intervals after the end of exercise without alteration of the inspired gas by means of a Fleisch No 3 pneumotachygraph and an integrator (47804A Pulmonary Calculator System, Hewlett Packard). The subject made two or three attempts at forced vital capacity manoeuvres each time and the best forced expired volume in one second (FEV₁) was chosen. All measurements were performed while the patient was seated on the cycle ergometer. The severity of exercise-induced asthma was calculated as the maximum percentage fall of FEV₁ after exercise:

$$\Delta \text{FEV}_1(\%) = \left(1 - \frac{\text{minimum postexercise FEV}_1}{\text{pre-exercise FEV}_1} \right) \times 100,$$

where the pre-exercise value was that measured immediately before starting exercise while the boy was breathing the warm humidified air. On a second occasion the test was repeated under identical conditions but after full sterilisation of the equipment. On a third occasion the test was repeated while the patient was breathing cold, dry air which was supplied to him by passing compressed air through calcium chloride and a simple refrigeration unit. All tests were performed on separate days over an eight-day period.

FEV₁ before and at intervals after a six-minute cycling exercise under different respired air conditions: hot, humid air test (1)—control; repeat hot, humid air test (2) after sterilisation of respiratory test system; cold, dry air test.

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Values of pre-exercise FEV₁ and exercise respiratory parameters for the three tests

	Warm humid air test 1	Warm humid air test 2	Cold dry air test
Pre-exercise FEV ₁ (l)	1.13	0.95	1.04
Minute ventilation (l/min)	42.0	40.8	42.0
Heart rate (beats/min)	158	162	159
Oxygen consumption (ml/min)	1318	1294	1258

In the first test, during the breathing of warm humid air, the baseline FEV₁ was 53% of predicted normal.⁸ Average minute ventilation, heart rate, and oxygen consumption over the last three minutes of exercise were 42 l/min, 158 beats/min, and 1318 ml/min respectively, the oxygen-consumption being 60% of predicted maximum.⁹ The mean expired air temperature was 37.3°C, compared with 37.1°C for the inspired gas, and thus the respiratory heat loss was negligible. The maximum fall in FEV₁ was 55%. The changes in lung function are shown in the figure. The second test was identical to the first in all respects and the response was very similar, as the figure shows. Measurement of lung function was stopped 15 minutes after the end of exercise when FEV₁ was still falling and the patient was given a bronchodilator. This was done because he showed severe shortness of breath coupled with signs of nausea. He was tested for the third time while breathing cold and dry air (5.7°C, water content 2.05 mg H₂O/l air). With the exception of the inspired air conditions, this third test was identical to the first two. He again developed exercise-induced asthma similar to that which he had developed previously in the two tests with warm humidified air, as shown in the figure, the maximum fall in FEV₁ being 47%.

Discussion

We report the case of a 13-year-old boy with bronchial asthma who consistently developed exercise-induced asthma while breathing warm and humid air at body temperature and humidity. This observation is contrary to previous reports,¹⁻⁴ which have attributed exercise-induced asthma to airway cooling, since in our case respiratory heat loss was negligible. To exclude the possibility that the attack was caused by an immunological rather than a mechanical stimulus the whole system was fully sterilised

before we undertook the repeat test. Before sterilisation multiple cultures showed a growth of mixed cocci and *Alternaria* spp, but after sterilisation only cocci were cultured. The patient was tested for skin sensitivity to *Alternaria* but did not react. As a final check on the possibility that asthma was caused by the apparatus, the patient breathed the same total volume of gas at the same temperature and humidity at rest as he had during his exercise tests. No detectable bronchoconstriction occurred.

We believe this to be the first case in which a patient was not protected by the warm humidified environment during exercise and developed as severe exercise-induced asthma as during exercise while breathing cold dry air. Possibly his response was related to his relatively low baseline level of FEV₁—most subjects reported previously have had more normal baselines. The protective effect of warm humid air might thus have been somewhat exaggerated by testing relatively fit patients.

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