Response of the nose to exercise in healthy subjects and in patients with rhinitis and asthma

Joan Serra-Batelles, Josep M Montserrat, Joaquim Mullol, Eugeni Ballester, Antoni Xaubet, César Picado

Abstract

Background – Although the nose and the bronchi are both involved in the process of regulating respiratory heat exchange, thermal changes may precipitate airway obstruction during exercise but rarely cause nasal obstruction in patients with rhinitis. The cause of the different response of the nose and bronchial tree has hardly been investigated. This study was performed to assess the response of the nose during exercise in the presence of rhinitis, asthma, and in normal controls.

Methods – Ten healthy subjects (group 1), 15 patients with asthma and rhinitis (group 2), 10 with rhinitis only (group 3), and 11 with asthma only (group 4) were included in the study. Exercise was performed on a bicycle ergometer for six minutes, reaching a heart rate of 80% of predicted. Bronchial and nasal responses were measured by forced expiratory volume in one second (FEV₁) and posterior rhinomanometry, respectively. A drop in the FEV₁ of 20% or more was considered a positive exercise induced asthma challenge test.

Results – Heart rate and ventilation increased by a similar proportion in the four groups. The FEV₁ significantly decreased in asthmatic patients (groups 2 and 4) but it did not change in healthy subjects (group 1) or those with rhinitis (group 3). Thirteen asthmatic patients developed exercise induced asthma. Nasal patency increased with exercise by a similar proportion in all groups, and no differences were detected between those with rhinitis (groups 2 and 3) and those without (groups 1 and 4). Nasal patency had returned to basal values at 25 minutes after completion of exercise in the four groups. The nose of patients with exercise induced asthma, however, remained significantly more patent than in patients without exercise induced asthma between 10 and 30 minutes after exercise.

Conclusions – These results suggest that the nose responds differently from the bronchi during exercise induced airway obstruction: whereas the bronchial tree responds by becoming narrowed, the nose becomes more patent. These findings suggest that the mechanisms regulating the response of the nose to exercise are different from those involved in the response of the bronchial tree.

The lamina propria of the nasal mucosa contains capacitance vessels which are venous sinuoids that form the erectile elements of the nose. While the patency of the nasal cavities is regulated by the erectile tissue, the lumen of the bronchi varies, at least in part, according to relaxation or contraction of the smooth muscle contained in their walls.

In asthmatic patients exercise induces an initial bronchodilation which is often followed by bronchospasm. The bronchospastic response usually occurs after stopping exercise. The severity of the asthmatic response increases with increasing work load and also with increasing duration of exercise. It is generally accepted that airflow obstruction after exercise is related to the thermodynamic events that occur in the tracheobronchial tree during exercise.

Although the nose and the bronchi both participate in the conditioning of inhaled air, thermal changes during exercise may cause bronchoconstriction in asthmatic patients but rarely, if ever, cause nasal obstruction in patients with rhinitis. Interestingly, in asthmatic subjects with rhinitis, nasal resistance decreases with exercise and remains low even though the patient may develop severe bronchospasm.

Although the possible mechanisms responsible for exercise induced asthma have been extensively investigated, the response of the nose to exercise has received much less attention. The effect of rhinitis on the nasal response to exercise in the presence or absence of bronchial asthma has only been evaluated in poorly defined groups of patients and for very short periods. In order to improve the understanding of the nasal response to exercise we studied 46 subjects divided into four groups: healthy subjects, patients with rhinitis alone, patients with asthma, and those with both asthma and rhinitis.

Methods

Population

Forty six non-smoking subjects were included in the four groups of the study which comprised 10 healthy subjects (group 1), 15 patients with rhinitis and asthma (group 2), 10 patients with rhinitis only (group 3), and 11 with asthma only (group 4).

Patients with asthma had a typical history of paroxysms of dyspnoea, wheezing, and coughing which improved after the administration of a bronchodilator. An increase in the FEV₁ exceeding 15% predicted had been obtained at least once following 200 µg salbutamol. None
Nasal response to exercise in rhinitic and asthmatic patients

Mean (SD) data of subjects in study

<table>
<thead>
<tr>
<th></th>
<th>Healthy subjects</th>
<th>Rhinitis and asthma</th>
<th>Rhinitis</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (M:F)</td>
<td>8:2</td>
<td>9:6</td>
<td>9:1</td>
<td>7:4</td>
</tr>
<tr>
<td>Age (years)</td>
<td>26 (7)</td>
<td>25 (9)</td>
<td>24 (7)</td>
<td>26 (10)</td>
</tr>
<tr>
<td>FEV(_1) (%)</td>
<td>103 (8:5)</td>
<td>81 (21)*</td>
<td>105 (16)</td>
<td>89 (16)*</td>
</tr>
<tr>
<td>Nasal flow at 150 Pa (cm(^3)/s)</td>
<td>493 (152)</td>
<td>379 (206)*</td>
<td>496 (235)</td>
<td>460 (183)</td>
</tr>
</tbody>
</table>

*\(p<0.005\) compared with healthy subjects and patients with rhinitis.

Results

Details of the subjects are summarised in the table. Basal FEV\(_1\) was significantly lower (\(p<0.005\)) in the asthmatic patients (groups 2 and 4) than in the healthy subjects and those with rhinitis (groups 1 and 3). No significant differences were found in nasal patency among the four groups.
EXERCISE TEST
There were no differences in baseline heart rate among the four groups. Heart rate increased during exercise in a similar manner in all subjects, suggesting that the work done was similar (data not shown). Basal ventilation was similar in all groups and increased in a similar proportion during exercise. There were no differences in ventilation during the last minute of exercise (data not shown).

BRONCHIAL RESPONSE
FEV₁ did not change after exercise in the healthy subjects (group 1) or in the patients with rhinitis (group 3) but decreased significantly in those with asthma (fig 1). No differences were found in the airway responses between asthmatic patients, with and without rhinitis (data not shown). Thirteen (50%) of the 26 asthmatic patients studied developed a drop in FEV₁ of 20% or more from the baseline value and were considered positive for exercise induced asthma. Among the 15 patients with a positive clinical history for exercise induced asthma, 11 (five with asthma and rhinitis and six with asthma alone) showed a positive exercise challenge test. Only two patients with a negative clinical history developed bronchoconstriction after the exercise test.

NASAL RESPONSE
Although patients with rhinitis showed a lower nasal flow at 150 Pa pressure than the remaining three groups, the difference did not reach statistical significance (table). Nasal patency increased with exercise by a similar proportion in all groups and progressively returned to the normal range, returning almost to baseline values 25 minutes after the completion of exercise (fig 2). No differences in nasal responses were found when patients with and without rhinitis were grouped together, nor between asthmatic and non-asthmatic patients. However, the nasal response in asthmatic patients who developed exercise induced asthma differed from those who did not. In patients with exercise induced asthma nasal patency decreased at the same rate as non-responders during the first 10 minutes, but between 10 and 60 minutes the nose of patients with exercise induced asthma remained more patent and consequently the flow at 150 Pa was significantly higher in these patients than in the non-responding asthmatics (fig 3). The intensity of exercise and baseline nasal airway resistance was similar in both groups of patients.

Discussion
Our study shows that the nose dilates during exercise and that this response is independent of the presence of rhinitis. We have also shown that, when exercise precipitates bronchocon-
striction in asthmatic subjects, the nasal response is completely dissociated from the bronchial response as the nose never develops obstruction. The nose remains significantly more dilated during bronchoconstriction, however, reinforcing the impression that it behaves inversely to the bronchi during exercise induced airway obstruction.

Why does the nose, which is morphologically and functionally closely linked to the upper and lower airways, behave in such a different manner? There is one study that might help to explain, at least in part, the peculiar response of the nose. Rubinstein et al.10 investigated the response of the airways to exercise in asthmatic and healthy subjects using an acoustic reflection technique which allows the measurement of the diameter of the upper as well as the central airway. They found that, in healthy subjects, exercise did not change FEV1, but caused a significant increase of approximately 55% in the cross-sectional area of the intrathoracic trachea. No significant changes, however, were found in the bronchial tree. In asthmatic patients with exercise induced asthma, exercise resulted in a significant reduction in FEV1, and a concomitant decrease in the bronchial cross-sectional area: however, the extrathoracic and intrathoracic tracheal areas showed an unexpected and significant increase. These findings provide clear evidence that the bronchi are the main site of airway narrowing in exercise induced asthma and that tracheal dilation can occur at the same time. These observations fit well with our findings and suggest that the nasal response to exercise, both in healthy subjects and in patients with rhinitis and asthma, parallels that of the trachea. This increase in the upper airway diameter may explain the bronchoconstrictor response immediately after exercise in both asthmatic and healthy subjects.12,13 Taken together, these findings suggest that two different mechanisms regulate the patency of the upper and lower airways.

Although the role of vessels in the regulation of tracheal and main bronchi patency has received little attention, several studies have shown that the tracheobronchial mucosa has an extensive system of blood sinuses.14 Since the response of the trachea to exercise closely imitates the nasal response which is dependent on changes in the volume of capacitance vessels, it is reasonable to assume that the enlargement of the trachea during exercise might also be caused by a vasoconstrictor phenomenon of the sinus network present in the tracheal mucosa.

Although at present there is no clear explanation for the dichotomous response of the different parts of the upper and lower airways, there is evidence in the literature to suggest that both parts show a different regulatory system. Syabbalo et al.6 found that alpha-adrenergic agonists reduce blood flow in the dog trachea, suggesting

that both the nose and trachea are controlled by the same adrenergic system. In contrast with the upper airway, adrenergic nerve fibres seem to play a minor part in the regulation of lower airways in both man and animals.17

In our study we found that the nose remains significantly dilated in asthmatic patients who develop exercise induced asthma, suggesting the possible existence of a reflex from the lower airway to the nose. It is tempting to speculate that the open nose during exercise induced asthma observed in this study might represent a protective reflex mechanism to diminish airway resistance during peripheral airway obstruction. Syabbalo et al.6 did not detect this response of the nose in asthmatic patients suffering exercise induced asthma, probably because they included in their study only four patients with exercise induced asthma in whom measurements of nasal flow were taken for 10 minutes after the completion of exercise. As differences in the nasal response between patients with exercise induced asthma and asthmatics without exercise induced asthma are detected after 10 minutes, this difference in methodology between the two studies probably accounts for the different results.

A rebound phenomenon characterised by an increase in nasal resistance starting approximately 20 minutes after exercise has been described in healthy subjects and in patients with either asthma or rhinitis.6 The mechanisms responsible for the rebound response are unknown. We did not detect any late increase in nasal airway resistance in our study. The rebound mainly occurs with mild degrees of exercise and is not usually detected when the work load is increased.18 Since our subjects were submitted to an intense level of exercise, this probably accounts for the lack of any late increase in nasal resistance in our study.

In summary, we have shown that the nose, unlike the bronchi, never develops obstruction in patients suffering from rhinitis and asthma when challenged with exercise. We also found that the nose remains dilated during exercise induced bronchoconstriction in asthmatic patients. This finding suggests the presence of a reflex from the lower airway to the nose which increases nasal patency in these patients. Since a previous study has also shown a dichotomous response between the trachea and central/peripheral bronchi in exercise induced asthma, these results suggest that the mechanisms regulating the response of the upper airway (nose and trachea) to exercise differ from those controlling the central and peripheral airways.

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