Short Report

Nostril widening improves arterial oxygenation: a role for nasal nitric oxide?

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Background

Healthy humans normally breathe mostly through the nose, whereby the nasal cavity serves to regulate the temperature and humidity of the inhaled air. However, the nasal airways may not only function as an ingenious heat exchanger and humidifier; recently it was shown that the potent vasodilator nitric oxide (NO) is present in high concentrations in the nasal airways of healthy subjects (1). During nasal breathing substantially more NO reaches the lungs as compared to oral breathing and when inhaled this NO has been suggested to modulate pulmonary function (2,3). The anterior part of the nose is the narrowest passage in the upper airway and it accounts for more than half of the total airway resistance during nasal respiration (4). Therefore, nostril widening devices have been developed in order to improve nasal breathing. These include nasal tapes and were originally developed to be used by subjects suffering from snoring or sleep apnoea syndrome. Recently, these tapes have also been used by athletes in order to aid nasal breathing during physical exercise, although the beneficial effects of this concept have not been evaluated.

Methods

We studied the effects of nostril widening on arterial oxygenation in a group of 11 spontaneously breathing patients in a neurosurgical intensive care unit. The majority of the patients were unconscious and no instructions were given regarding breathing technique. All were in the supine position throughout the experiment and oxygen supplementation was withdrawn 30 min before start. A nostril-widening tape (Breathe-right®, 3M Pharmaceuticals) was applied to the patients for 30 min. Arterial oxygenation (PaO₂) was measured together with carbon dioxide (PaCO₂) at the end of this period and the values were compared to those obtained before the application and 30 min after the plaster had been removed.

Results

PaO₂ was higher during the period of nostril widening compared to before the treatment period whereas ventilation, as measured by PaCO₂, remained unchanged throughout the experiment. Basal PaO₂ was 10·18 ± 0·55 and rose to 10·81 ± 0·42 (P < 0·02). Thirty minutes after the plaster was removed, PaO₂ had decreased to 10·41 ± 0·57 (Fig. 1).

Discussion

Inhaled exogenous NO has been shown to improve oxygenation and reduce pulmonary hypertension in patients with pulmonary disorders (5). The amounts of NO that reach the lung during nasal breathing (2) are in the same range as those proven to be effective during treatment with exogenous NO (5). A recent study shows that NO of nasal origin is involved in modulation of pulmonary function under normal as well as pathological conditions. Enhancement of nasal breathing with, for example, a nostril widener will lead to higher concentrations of NO in inspired air which may explain the results obtained in this study. This mechanism may add to the other well-known beneficial effects of nasal breathing.
References