Acta Physiol Scand. 1996 Dec; 158 (4): 343-7.

Inhalation of nasally derived nitric oxide modulates pulmonary function in humans.

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Abstract

The vasodilator gas nitric oxide (NO) is produced in the paranasal sinuses and is excreted continuously into the nasal airways of humans. This NO will normally reach the lungs with inspiration, especially during nasal breathing. We wanted to investigate the possible effects of low-dose inhalation of NO from the nasal airways on pulmonary function. The effects of nasal and oral breathing on transcutaneous oxygen tension (tcPO2) were studied in healthy subjects. Furthermore, we also investigated whether restoring low-dose NO inhalation would influence pulmonary vascular resistance index (PVRI) and arterial oxygenation (PaO2) in intubated patients who are deprived of NO produced in the nasal airways. Thus, air derived from the patient's own nose was aspirated and led into the inhalation limb of the ventilator. In six out of eight healthy subjects tcPO2 was 10% higher during periods of nasal breathing when compared with periods of oral breathing. In six out of six long-term intubated patients PaO2 increased by 18% in response to the addition of nasal air samples. PVRI was reduced by 11% in four of 12 short-term intubated patients when nasal air was added to the inhaled air. The present study demonstrates that tcPO2 increases during nasal breathing compared with oral breathing in healthy subjects. Furthermore, in intubated patients, who are deprived of self-inhalation of endogenous NO. PaO2 increases and pulmonary vascular resistance may decrease by adding NO-containing air, derived from the patient's own nose, to the inspired air. The involvement of self-inhaled NO in the regulation of pulmonary function may represent a novel physiological principle, namely that of an enzymatically produced airborne messenger. Furthermore, our findings may help to explain one biological role of the human paranasal sinuses.