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Changes in nasal airway resistance in response to controlled external respiratory obstruction

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Summary. A high degree of external nasal obstruction will by itself decrease nasal airway resistance. However, a controlled series of experiments in normal volunteers has shown that this lessened resistance is still not able to balance the level of the external obstruction present. The implications of this finding are discussed.

Key words: Hypercapnia – Severe nasal obstruction

Introduction

Tatum [12] first demonstrated that nasal airway resistance was decreased during breath holding in man and during experimentally induced asphyxia in animals; he suggested that this change was a reflex in nature that was controlled by the sympathetic nervous system of the nose. Dallimore and Eccles [2] further indicated that the hypercapnia produced by rebreathing was a major factor responsible for the decrease in nasal airway resistance.

In a recent animal experiment, Lung and Wang [4] confirmed that the nasal airway response to hypercapnia was partly due to a primary reflex action via the sympathetic nervous system and partly due to a local effect of carbon dioxide on the nasal capacitance vessels. They also suggested that it was likely that the hypoxic decrease in nasal airway resistance is usually masked or compensated for an increase in arterial blood flow. This latter occurrence resulted from a reflexive increase in systemic arterial blood pressure and ventilation through chemoreceptor stimulation.

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Cvetnic et al. [1] studied the effect of temporary nasal obstruction in healthy volunteers and found significant elevation of arterial pCO_2 accompanied by a depression of the arterial pO_2. Ramadan [9] reported that in animal experiments following bilateral nasal obstruction there was a marked decrease in pO_2 and a marked increase pCO_2.

From these previous observations, there is a possibility that nasal obstruction in and of itself may be responsible for the decrease in nasal airway resistance that results. In our present study, we aimed to determine whether or not a controlled external nasal obstruction was able to actually decrease internal nasal airway resistance.

Subjects and methods

Seven adult Japanese volunteers, aged 20–30 years, were used in our laboratory for this study. All had normal nasal anatomy and normal spirometric function values. They also denied having any history of allergic, cardiovascular or pulmonary disease. Total nasal airway resistance (NAR), end-expiratory CO_2 (FCO_2) and O_2 (FO_2) were measured simultaneously, as explained below.

NAR. Posterior rhinomanometry was performed using a rhinomanometer (Nihon kohden). This apparatus is a self-contained single unit that includes pneumotachograph, pressure transducer and storage oscilloscope. Nasal airflow was measured via a mask attached to the pneumotachograph. The pressure difference between the nasopharynx and the inside of the mask was measured with a polyethylene catheter placed in the posterior oropharynx. Pressure and flow were simultaneously displayed on the X-Y axes of the oscilloscope. NAR was calculated at a pressure of 1 cm H_2O during expiration to avoid the influence on NAR exerted during inspiration by the nasal valves.

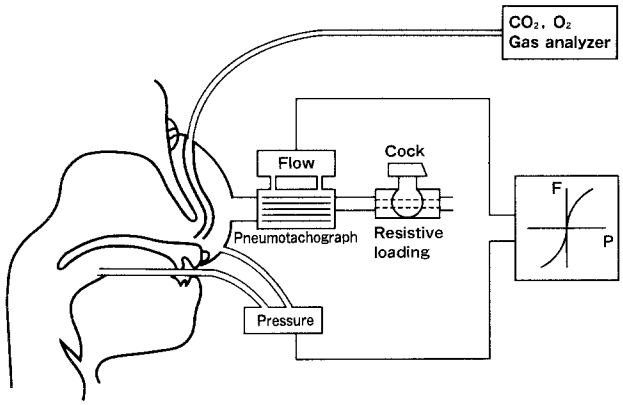

Fig. 1. Schematic drawing of the experimental set-up

Gas analysis. End-expiratory CO_2 (FCO_2) and O_2 (FO_2) were measured. The expired air was sampled at the nostril through a capillary tube, which was connected to a carbon oxide and oxygen gas analyzer. The capillary tube did not affect the NAR.

External nasal resistance. Several loads were added to obstruct the nose to differing degrees during nasal breathing. This was accomplished using a resistor, which was constructed of a variably sized cock and a polyvinyl pipe. The resistor was precalibrated approximately to 0 (R_0), 4.8 (R_1), 10 (R_2), and 19.5 (R_3) cm H_2O/l per second (linear up to 1l/s) and was connected directly to the pneumotachometer on the respiratory line.

Experimental procedures. All parameters were recorded on a four-channel polygraph (Nihondenki-Sanei, 1H26). Each subject was seated upright and underwent three tests, using different levels of external loading resistances (R_1, R_2, and R_3) for 2 min each. A 2-min control (unobstructed) test (R_0) was performed before and after each of the three tests. The baseline values for NAR, FCO_2 and FO_2 were measured immediately before each of these tests, following which NAR, FCO_2 and FO_2 were measured every 30 s during and after each test. The experimental design of the study is illustrated in Fig. 1.

Results

The control values for FCO_2, FO_2 and nasal airway resistance ranged from 4.7% to 5.8%, from 14.1% to 16.2% and from 2.7 cm H_2O/l per second to 4.3 cm H_2O/l per second, respectively. Figure 2 shows the mean ± SD values of FO_2, FCO_2 and nasal airway resistance for the controls and for every 30 s during which the noses were obstructed. The responses of FCO_2 and FO_2 to the three different external respiratory obstructions were remarkable. The differences between the control values and the values during each study were examined by the paired *t*-test. The results are shown in Fig. 3. Both FCO_2 and FO_2 recovered to the control level within 90 s after removal of the external nasal obstruction. Significantly different nasal airway resistances were first detected when

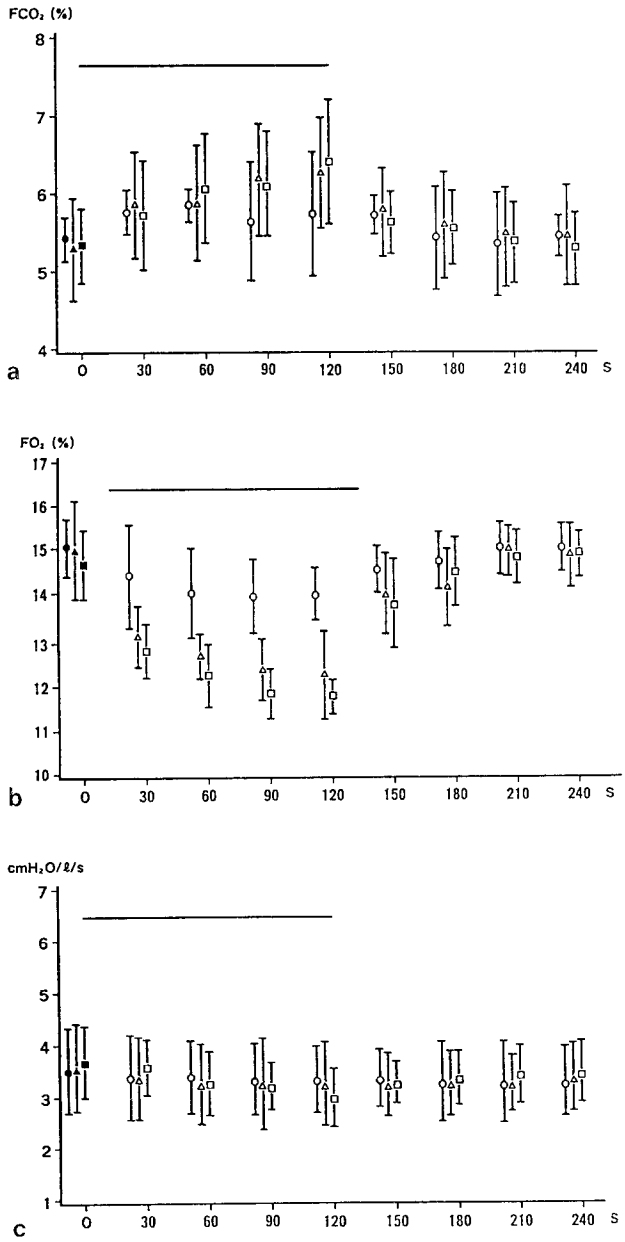

Fig. 2a–c. Relationship between the different loadings and magnitude of variation (mean ± SD) in FCO_2 (**a**), O_2 (**b**) and nasal airway resistance (**c**). *Circles* (R_1), *triangles* (R_2) and *squares* (R_3) indicate each of three tests using different degrees of obstruction: 4.8, 10 and 20 cm H_2O/l per second, respectively. *Solid markers* indicate the control values. *Bars* show the external obstructive loadings

the external nasal obstruction was R_3: that is, about 20 cmH_2O/l per second. Scheffè's method [10] was used to determine whether or not the different values for external respiratory loading (namely R_1, R_2 and R_3) had any significant influences on the difference in FCO_2 between the control level and that 120 s before the removal of the external obstruction. Significant differences in FCO_2 changes were observed between

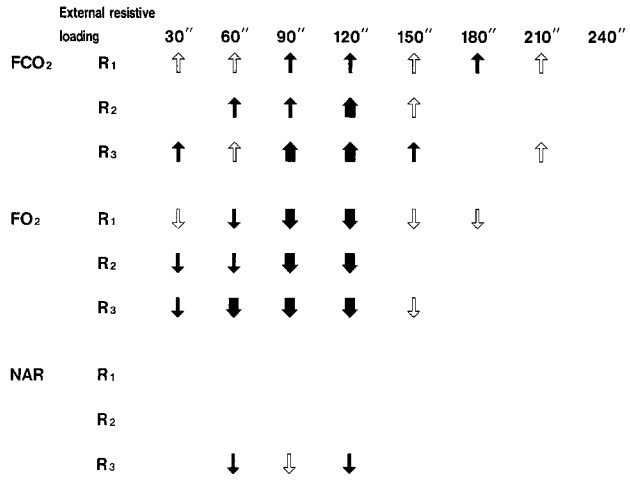

Fig. 3. Difference between the control values and the values in FCO_2, FO_2 and nasal airway resistance every 30 s during and after each of the external obstructive loadings. ⇧⇩, ⬆⬇, ⬆⬇ indicate significant increase and decrease at 5%, 1%, and 0.1% level, respectively

R_1 and R_2, and between R_1 and R_3, but not between R_2 and R_3. The same tendencies were also found in the FO_2 levels.

Discussion

Our study elucidated the following points: when nasal breathing takes place in the presence of significant obstruction, there is an increase in FCO_2, a decrease in FO_2, and a decrease in nasal airway resistance. In other words, when nasal breathing is forced to push against significant obstruction, the effect of this obstruction appears to be that of decreasing the nasal resistance.

Since Tatum [12] reported that nasal patency is increased by asyphyxia, many investigators have reported that hypercapnia decreases nasal airway resistance [2, 4, 5, 6, 8, 11]. In order to establish hypercapnic conditions, nasal breathing has been subjected to rebreathing circuits or to an atmosphere with a high CO_2 partial pressure. However, such conditions are hardly common in normal life. We induced hypoventilation in our subjects and found a subsequent increase in FCO_2 and a decrease in FO_2, using controlled external nasal obstruction (and resembling that seen under clinical conditions). In this state, we examined whether the nasal airway resistance itself was changed. A decrease in nasal airway resistance has been reported by Takagi et al. [11] and was induced by having subjects inhale an atmosphere containing 7% CO_2. Similar findings were described by Mertz et al. [8], using O_2 mixtures containing 5% CO_2. In our present study, a decrease in nasal airway resistance was observed at CO_2 levels of about 6% or more, which approximately coincides with the levels recorded by Takagi and Mertz.

The difference in the decrease of FCO_2 between the R_2 and R_3 loadings was not significant in our study. It was interesting to us to find that the decrease in nasal airway resistance was observed only under R_3 loading. Although hypercapnia may be the major factor related to the decrease in nasal airway resistance, other factors must also be taken into consideration. In dog experiments, McCaffrey and Kern [6] found that the pulmonary Hering-Breuer reflexes and chest wall receptors did not participate to any great extent in the reflex control of normal nasal airway resistance. In contrast, with a high degree of external nasal obstruction, such as that used in our study, both air inflow and outflow were interrupted, leading to a decrease in the movements of the chest wall. According to Hoshino [3], there are cold and warm receptors for the recognition of nasal flow sensation. McBride and Whitelaw [7] have demonstrated that the upper airway flow sensitive receptors, which seem to be the same as the thermosensitive receptors found by Hoshino, influence respiration. Under pronounced external obstruction to the nose, the flow sensitive receptors seem to be much less activated and as a result influence the respiratory center in the brain.

It has been generally recognized that the nasal airway can be a variable and high-resistance segment of the upper airway, and is very labile to various stimuli influencing respiration. In fact, when minute ventilation increases, the nasal airway resistance decreases [5, 6, 8]. The high degree of external respiratory obstruction presented to the nose in our study probably decreased minute ventilation through nasal breathing. Hypercapnia with low minute ventilation through nasal breathing is essentially different from that found in other investigations [5, 6, 8]. Our findings show that acute severe nasal obstruction in humans itself tends to decrease nasal airway resistance, but it is impossible for us to say that the decrease occurring in nasal airway resistance balances the nasal obstruction present.

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