

The alteration of nasal resistance before and after local exposure to heated aerosol in perennial allergic rhinitis

Kensei Naito¹, Sho Miyata¹, Ren Baba¹, Toshiko Mamiya¹, Yoshio Senoh¹, Shigenobu Iwata¹, Naoki Yokoyama², Satoshi Yamakawa³, Katsuro Iwata³

¹ Department of Otolaryngology, Fujita Health University, School of Medicine

² Department of Otolaryngology, Hekinan Municipal Hospital

³ Department of Otolaryngology, Shinshiro Municipal Hospital

SUMMARY

To determine the patho-physiological effects of heated vapour to the normal or allergic nasal mucosa, we measured the nasal resistance before and after a 10 min. exposure of hyperthermal (43.0°C) aerosol to the nasal mucosa in normal subjects and perennial allergic rhinitis patients. In the allergic patients the mean nasal resistances after hyperthermal stimulation were significantly higher than those resistances without stimulation, both in expiration or inspiration. No significant differences of nasal resistances in normal individuals during the whole schedule with and without heated aerosol stimulation were found on expiration or inspiration. The local heated aerosol exposure increases the nasal resistance in nasal allergic patients while in normal subjects no changes were found, and the reaction may have arisen from a non-specific hypersensitivity of the susceptible allergic nasal mucosa.

Key words: nasal resistance, nasal allergy, hyperthermia, nasal heated aerosol

INTRODUCTION

Yerushalmi et al. (1982) demonstrated the clinical efficacy of heated nasal aerosol for symptoms, including nasal stuffiness, of perennial allergic rhinitis. Ophir et al. (1988) and Grossman et al. (1988) also reported the effectiveness of nasal hyperthermal vapours to nasal allergy. However, the mechanism for this proposed beneficial efficiency of heated moisture is still unknown. Physiologically, inspired nasal air causes volume changes of the erectile venous sinus structures in the nasal mucosa depending on the temperature and humidity (Salman et al. 1971, Olsson & Bende 1985). There have been few reports concerning local physiological effects of hyperthermal aerosol on the nasal mucosa in normal or allergic individuals. To determine the pathophysiological responses of heated aerosol to the nasal mucosa, we studied the physiological effects of a 10 min. exposure of heated (43.0°C) aerosol to the nasal mucosa in perennial allergic rhinitis patients and normal subjects.

MATERIALS AND METHODS

Twenty four patients (14 males and 10 females, mean age of 24.8 years) with perennial nasal allergy were diagnosed by means of eosinophilia in the nasal secretion and the presence of serum specific IgE antibodies to mite and/or house dust as detected by the radio allergosorbent test (RAST) or an intradermal injection reaction to house dust, and 30 normal subjects (19 males and 11 females, mean age of 23.8 years). Nasal resistances were measured 10 min. before, right before, right after, 15 min. after, 30 min. after, 24 hr. after and 24 hr. & 10 min. after nasal heated aerosol stimulation (experiment 1). Both patients and normal subjects in the schedule of experiment 1 were exposed for 10 min. to 43.0°C heated aerosol with Skynar steam II (manufactured by Eisai Co., Ltd. Japan).

Nasal resistance measurements were performed by Rhinorheograph MPR-2100 (manufactured by Nihon Kohden Co., Ltd. Japan) using active anterior rhinomanometry using a nasal nozzle during relaxed breathing through the nose with the subject in a sitting position. Values of nasal resistance at the

peak flow point were determined. Total nasal resistances were calculated from the modified equation of Ohm's law for parallel resistors (Naito et al. 1990). Another 19 patients (12 males and 7 females, mean age of 24.7 years) and 10 normal subjects (5 males and 5 females, mean age of 25.2 years) were employed in a control study (Experiment 2). Nasal resistances in patients and normal subjects were measured in the same time course as Experiment 1 but without stimulation of heated aerosol. Summary measures (Matthew et al. 1990) were employed to analyse serial measurements and $p < 0.05$ was considered to be statistical significant in this study.

RESULTS

No significant differences in age (t-test) and sex (χ^2 -test) between the individuals with and without local hyperthermal stimulation in Experiment 1 and 2 were found. Alterations of mean values of inspiratory nasal resistance in the allergy patients with and without the nasal heated humidity challenge are shown in Figure 1. The values of nasal resistance after challenge were significantly higher than those in the patients without the nasal warm moist aerosol stimulation ($p < 0.02$). In expiratory patients, similar results compared to the inspiratory resistances were observed ($p < 0.01$). Alterations of mean values of inspiratory nasal resistances in the normal subjects with and without the nasal heated humidity challenge are shown in Figure 2. No significant differences of inspiratory nasal resistance between the normal subjects with and without the nasal heated vapour stimulation were found ($p > 0.65$). In expiratory nasal resistances of the normal subjects, similar results compared to the inspiratory resistances were observed ($p > 0.77$).

DISCUSSION

Topical use of heated (43.0°C) vapour to the nasal mucosa without any medicine, is an effective and safe treatment for symptoms in infectious and allergic rhinitis (Yershalmi et al. 1982, Ophir et al. 1988, Grossman et al. 1988). Therefore, we can employ the local hyperthermal aerosol therapy for pregnant women with allergic and infectious rhinitis (Narita et al. 1994). On the contrary, Oppenheimer et al. (1993) did not demonstrate any evidence of therapeutic effects of heated, moisturised

air in the treatment of allergic rhinitis. Until now, the mechanism for the proposed benefit of heated moisture has been elusive. Monger & Schild (1957) found that anaphylactic histamine release was inhibited by both high and low temperature. However, Proud et al. (1992) reported that cold, dry air led to increased recovery of both histamine and tryptase, a specific mast cell enzyme, confirming that mast cell degranulation occurred during this reaction in the susceptible nasal mucosa. Togias et al. (1985) observed that cold, dry air caused a significant increase in histamine and prostaglandin (PG) D₂ in nasal lavage fluids and symptom scores as compared to baseline or to warm, moist air in subjects with a history of rhinorrhea and congestion upon cold or dry environmental exposure. Johnston et al. (1993) considered that the hyperthermia inhibited newly formed mediator production by mast cells in the nasal mucosa of allergic rhinitis. While, Yerushalmi et al. (1982) proposed that the hyperthermia decreased the severity of viral infections, considered an exacerbating factor of allergic reactions, and the phenomenon was suspected to influence the efficacy for allergic disorders. The normal nasal mucosa naturally changes the volume due to a reflex upon several stimuli and the rhythm of the autonomic nerve system is mainly regulated by blood flow in the mucosa (Olsson & Bende 1985). Ambient air-conditions, for example temperature and humidity, influence the alterations of the nasal passage (Salman et al. 1971). Furthermore, allergic nasal mucosa responds more to various ambient irritants (Proud et al. 1992). The non-specific incentive reaction in allergic rhinitis is being regarded as hypersensitivity. There are no experiments concerning the influence of a single exposure of 10 min. local heated (43.0°C) aerosol on normal and allergic mucosa, therefore we decided to determine the responses to intranasal warm, moist air exposure. In the present study, nasal resistances in normal subjects were not reduced, neither with or without local hyperthermal vapour. On the other hand, the resistances in perennial allergy patients were significantly increased after a local heated moisturised aerosol stimulation, while there were no changes of nasal resistance without the stimulation. The reaction occurred immediately, continued for at least 30 min. and disappeared within 24 hours after the stimulation. Only a single dose of 10 min. exposure to heated vapour might act irri-

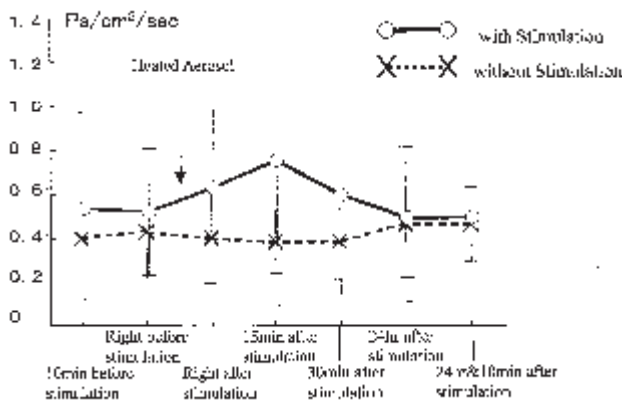


Figure 1. The Alteration of Nasal Resistance before and after Nasal Heated Aerosol Stimulation (Insp.) in Allergy Patients.

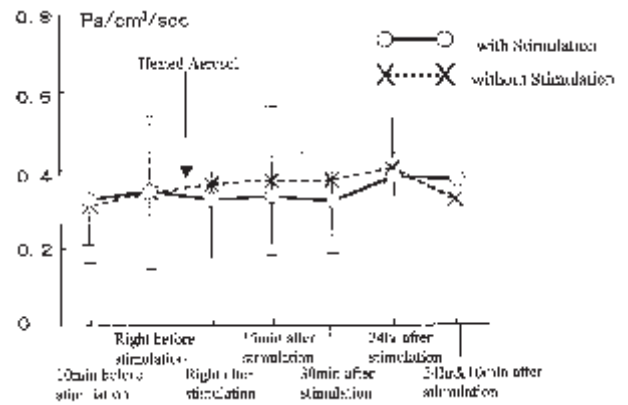


Figure 2. The Alteration of Nasal Resistance before and after Nasal Heated Aerosol Stimulation (Insp.) in Normal Subjects.

table in perennial nasal allergy patients, though the warm, moist air was assumed to be a treatment of nasal allergy. Our results, indicate that the beneficial effectiveness of the heated aerosol to nasal allergy may not be due to normal autonomic nerve reflexes. Most likely, other therapeutic mechanisms of nasal hyperthermal aerosol acting on the allergic nasal mucosa are suspected. It is possible that nasal lavage washes out antigens from the nasal cavity, an inhibition of allergen-induced release of histamine (Monger & Schild, 1957) or release of other newly formed mediators (Johnston et al. 1993), a reduction of the sensitivity of the vasculature to inflammatory mediators (Johnston et al. 1993) and/or defence potentials against viral infections causing to exacerbate the allergic reactions and their symptoms (Yerushalmi et al. 1982).

ACKNOWLEDGEMENT

We wish to express our thanks to Professor Philip Cole Department of Otolaryngology, Airflow Laboratory, University of Toronto) for his help in the preparation of this paper.

REFERENCES

- Grossman, J., Ball, R., Shyulan, D. and Porcella, J (1988) Treatment of seasonal allergic rhinitis with vapour induced nasal hyperthermia (Abstract). *J Allergy Clin Immunol* 81: 175.
- Johnston, S.L., Price, J.N., Lau, L.C.K., Walls, A.F., Walter, C., Feather, I.H., Holgate, S.T. and Howarth, P.H. (1993) The effect of local hyperthermia on allergen-induced nasal congestion and mediator release. *J Allergy Clin Immunol* 92: 850-856.
- Matthews, J.N.S., Altman, D.G., Campbell, M.J. and Royston, P. (1990) Analysis of serial measurements in medical research. *British Med J* 300: 230-234.
- Monger, J.L. and Schild, H.O. (1957) Effect of temperature on the anaphylactic reaction. *J Physiol* 135: 320-338.
- Naito, K., Cole, P. and Humphrey, D. (1990) Unilateral and bilateral nasal resistances: a supplement. *Rhinology* 28: 91-95.
- Narita, S., Asakawa, K. and Kataura, A. (1994) Local hyperthermia for allergic rhinitis in pregnancy (in Japanese with English abstract). *Practica Otologica* 87: 1739-1744.
- Olsson, P. and Bende, M.. (1985) Influence of environmental temperature on human nasal mucosa. *Ann Otol Rhinol Laryngol* 94: 153-155.
- Ophir, D., Elad, Y., Fink, A., Fishler, E. and Marshak, G. (1988) Effects of elevated intranasal temperature on subjective and objective findings in perennial rhinitis. *Ann Otol Rhinol Laryngol* 97: 259-263.
- Oppenheimer, J., Buchmeier, A. and Nelson, H.S. (1993) Double-blind trial of a heated nasal aerosol in the treatment of perennial allergic rhinitis. *J Allergy Clin Immunol* 92: 56-60.
- Proud, D., Bailey, G.S., Naclerio, R.M., Reynolds, C.J., Curz, A.A., Eggleston, P.A., Lichtenstein, L.M. and Togias, A.G. (1992) Tryptase and histamines as markers to evaluate mast cell activation during the responses to nasal challenge with allergen, cold, dry air, and hyperosmolar solutions. *J Allergy Clin Immunol* 89: 1098-1110.
- Salman, S.D., Proctor, D.F., Swift, D.L. and Evering, S.A. (1971) Nasal resistance: description of a method and effect of temperature and humidity changes. *Ann Otol* 80: 736-743.
- Togias, A.G., Naclerio, R.M., Proud, D. Fish, J.E., Adkinson, Jr. N.F., Kagey-Sobotka, A., Norman, P.S. and Lichtenstein, L.M.. (1985) Nasal challenge with cold, dry air results in release of inflammatory mediators possible mast cell involvement. *Am Soc Clin Invest* 76: 1375-1381.
- Yerushalmi, A., Karman, S. and Lwoff, A. (1982) Treatment of perennial allergic rhinitis by local hyperthermia. *Proc Nat Acad Sci USA* 79: 4766-4769.

Kensei Naito, M.D.
 Department of Otolaryngology
 Fujita Health University
 School of Medicine
 1-98 Kutsukake Toyoake
 Aichi 470-1192
 Japan
 Tel: +81-562-93-2195
 Fax: +81-562-95-0566