

Laryngeal resistance immediately after panting in asthmatic subjects

M YANAI, T OHROI, K SEKIZAWA, H SASAKI, T TAKISHIMA

From the First Department of Internal Medicine, Tohoku University School of Medicine, Sendai, Japan

ABSTRACT The panting manoeuvre may be used during the assessment of airway resistance and in asthmatic patients during bronchial provocation testing or spontaneous asthma. To study whether panting opens the larynx in patients with asthma, laryngeal resistance was examined in six patients with stable asthma before and after methacholine induced bronchoconstriction and in another six patients with spontaneous asthma. Subjects were asked to pant and then to hold their breath immediately afterwards. Laryngeal resistance after panting was compared to that during quiet tidal breathing. Change in laryngeal resistance was estimated by a method using low frequency sound and respiratory resistance by forced oscillation at 10 Hz. Mean baseline respiratory resistance during inspiration was 0.245 and 0.470 kPa/l.s before and after methacholine in the patients with stable asthma and 0.480 kPa/l.s in the patients with spontaneous asthma. In the patients with stable asthma mean laryngeal resistance was lower after panting than during the preceding quiet tidal breathing, both before and after methacholine induced bronchoconstriction (by 0.08 before and by 0.065 kPa/l.s after). In contrast, the patients with spontaneous asthma showed an increase in laryngeal resistance after panting of 0.089 kPa/l.s. The magnitude of change in laryngeal resistance after panting was similar to the change in respiratory resistance in the patients with spontaneous asthma and in the patients with stable asthma after methacholine, but was greater than the change in respiratory resistance in the patients with stable asthma before methacholine. These results suggest that panting may cause different effects on the laryngeal aperture in patients with stable and spontaneous asthma.

Introduction

Airway resistance is often measured during panting because this is thought to minimise the effect of breathing on the glottal aperture.¹ Higenbottam and Payne,² however, showed that glottal width was reduced in patients with chronic obstructive pulmonary disease and that it was not always greater during panting than during quiet breathing. We have also shown in normal subjects that the panting manoeuvre fails to open the larynx during histamine or methacholine induced bronchoconstriction.³ We have therefore investigated whether the panting manoeuvre opens the larynx in asthmatic subjects.

We have recently developed a technique for measuring change in laryngeal resistance with low frequency sound of 800 Hz.⁴ Voluntary closure of the larynx increases the sound pressure amplitude above the vocal cords and decreases that below the vocal cords. Changes in laryngeal resistance during voluntary

larynx narrowing correlate with changes in the difference between the sound pressure amplitude above and below the vocal cords. Thus if two microphones are attached to the anterior neck above and below the vocal cords laryngeal narrowing or widening may be estimated by forcing 800 Hz sound into the mouth of the subject.⁴

Goldstein and Mead⁵ examined respiratory impedance during breath holding immediately after panting. They suggested that the upper airways during breath holding immediately after panting were more fixed and open than during quiet tidal breathing and appeared to yield reproducible results with totally inexperienced subjects. To determine the effect of panting on laryngeal resistance in patients with asthma, we have examined laryngeal resistance during quiet breathing and during breath holding immediately after panting in asthmatic subjects in the control state, with methacholine induced bronchoconstriction, and during spontaneous asthma.

Methods

SUBJECTS

We studied 12 asthmatic subjects. The subjects were

Address for reprint requests: Dr T Takishima, First Department of Internal Medicine, Tohoku University School of Medicine, Sendai 980, Japan.

Accepted 16 June 1989

Laryngeal resistance immediately after panting in asthmatic subjects

laryngeal resistance). The estimates of change in laryngeal resistance are expressed as Δ sound (see legend to fig 1). The method differed from the original technique in that respiratory resistance was measured by forced oscillation at 10 Hz instead of 3 Hz. A frequency of 10 Hz enabled us to measure more rapid changes in respiratory resistance during breath holding immediately after panting than was possible with a frequency of 3 Hz. Change in laryngeal resistance calculated from change in respiratory resistance during voluntary closure of the larynx did not differ significantly between measurements with the forced oscillation technique made at 3 Hz and at 10 Hz.³

Laryngeal resistance decreased during inspiration and increased during expiration, change in laryngeal resistance being tightly coupled to ventilation. We took the midpoint of peak to peak values of laryngeal resistance during quiet tidal breathing as zero, and laryngeal resistances during inspiration and expiration were obtained at the mid tidal volume as the deviation from zero.³ Tidal volume and change in functional residual capacity were measured with a Krogh wedge spirometer (Chest Corporation, Tokyo). Mouth flow; respiratory resistance; and change in laryngeal resistance, tidal volume, and functional residual capacity were recorded with a pen recorder (Sanei, 8S, Tokyo).

PROTOCOL

Subjects sat in a pressure compensated volume displacement body plethysmograph, which was without amplitude or phase distortion up to 8 Hz. The subject, with noseclip in place, was instructed to breathe normally through a piece of flexible tubing held firmly in the mouth. Both buccal areas were compressed by the subject's hands. To avoid changes in skin tension and neck tissues, the head was kept vertical by adjusting the height of the chair from the mouthpiece. A constant bias flow of 0.4 l/s was applied by suction to minimise instrumental deadspace.

Six patients with stable asthma inhaled saline aerosols from a Vaponefrin nebuliser driven by a compressor (Nissho, Japan) for two minutes during quiet tidal breathing. The mean diameter of the particles produced by the nebuliser was 2.5 μ m (manufacturer's specification). There was no consistent difference in respiratory resistance or laryngeal resistance before and after inhalation of saline. After inhalation of saline subjects breathed quietly for two minutes while a forced oscillatory pressure of 10 Hz was applied in the mouth, and measurements of respiratory and laryngeal resistances were made. The applied forced oscillatory pressure was then stopped, and the panting manoeuvre at about 3 Hz was performed at functional residual capacity.³ The subject was asked to pant approximately in the resting tidal volume range. When functional residual capacity

changed over the tidal volume range the manoeuvre was repeated. After about three seconds the subject stopped panting and held his breath while a forced oscillatory pressure was applied to the mouth.⁵ As noise in the airway caused by the panting would interfere with the 800 Hz sound we analysed laryngeal resistance only in the interval after panting. After a five minute interval the same protocol was repeated. Five such manoeuvres were recorded.

Within a week of the first test the same six subjects were challenged with methacholine aerosols. The mean (SEM) inhalation time required to achieve a twofold increase in respiratory resistance was 4.5 (0.3) minutes. In preliminary experiments we found that the increase in respiratory resistance after methacholine inhalation with this method persisted with relatively little change for about 30 minutes. The subjects then performed the same procedure as on the first test day.

Six patients with spontaneous asthma were studied in the same way as the patients with stable asthma on the first test day. There was no consistent difference in respiratory resistance or laryngeal resistance between measurements before and after saline inhalation in any of the subjects.

ANALYSIS

The results are expressed as means with standard errors in parentheses. Statistical analysis was performed by means of a one way analysis of variance and Duncan's multiple range test. Significance was taken as $p < 0.05$.

Results

All subjects showed bronchial hyperresponsiveness to inhaled methacholine. There was no significant difference in the threshold concentrations of methacholine for the patients with stable asthma and the patients with spontaneous asthma ($p > 0.20$, table 1).

In the patients with stable asthma respiratory resistance during breath holding was lower after panting than during either the inspiratory or the expiratory phase of tidal breathing, both before and after methacholine. In the patients with spontaneous asthma, however, respiratory resistance was higher after panting than during quiet tidal breathing (fig 2). Although the patients with spontaneous asthma felt slight breathlessness and were inexperienced in the panting manoeuvre, the results obtained from each subject were consistent and reproducible.

METHACHOLINE CHALLENGE IN PATIENTS WITH STABLE ASTHMA

In the patients with stable asthma the mean (SEM) values for respiratory resistance during inspiration and expiration after saline inhalation before metha-

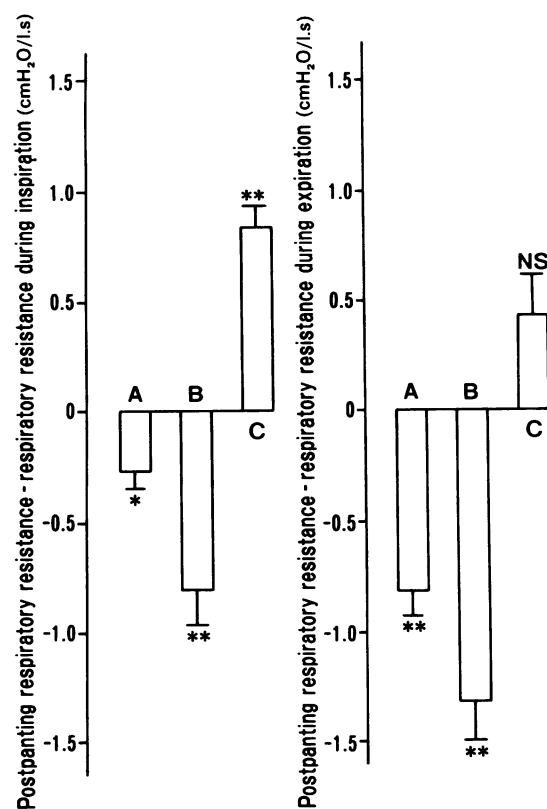


Fig 2 Differences between respiratory resistance during breath holding immediately after panting (postpanting) and respiratory resistance during inspiration and between postpanting respiratory resistance and respiratory resistance during expiration in the six patients with stable asthma before (A) and after methacholine challenge (B) and in the six patients with spontaneous asthma (C) (mean (SEM) values). Significant differences between postpanting respiratory resistance and respiratory resistance during inspiration or expiration are indicated by * ($p < 0.02$) and ** ($p < 0.01$).

choline challenge were 0.245 (0.029) and 0.294 (0.029) kPa/l.s. These did not differ significantly from the inspiratory or expiratory resistance (0.255 (0.029) and 0.304 (0.029) kPa/l.s) on the control day in the same patients. After methacholine there was an increase in respiratory resistance, from 0.245 (0.029) to 0.470 (0.029) kPa/l.s during inspiration and from 0.294 (0.029) to 0.519 (0.029) kPa/l.s during expiration. Percentage changes in inspiratory and expiratory Δ sound (see legend to fig 1) from the control values, however, (-3.6 (2.4) and -2.9 (2.3)) are not significant ($p > 0.20$).

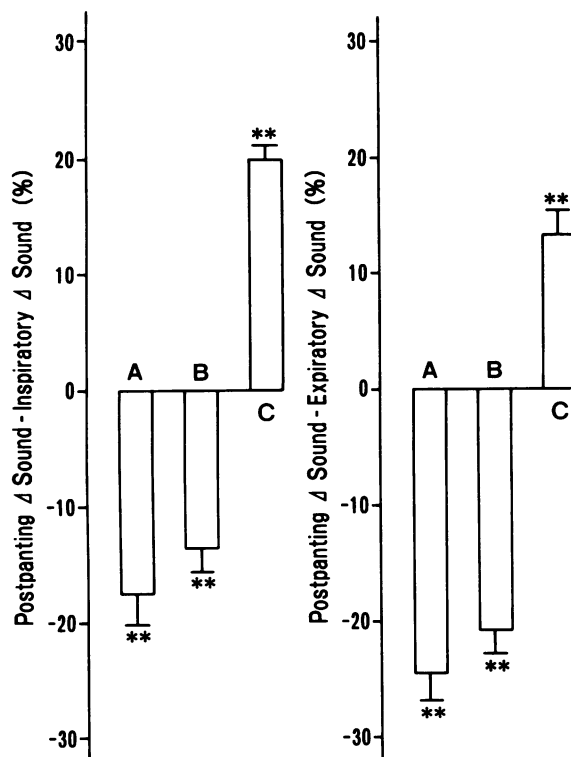


Fig 3 Differences between postpanting and inspiratory Δ sound and between postpanting and expiratory Δ sound in the six patients with stable asthma before (A) and after methacholine challenge (B) and in the six patients with spontaneous asthma (C). Results are reported as mean (SEM) values. Significant differences between postpanting Δ sound and inspiratory or expiratory Δ sound are indicated by ** ($p < 0.01$).

CHANGES IN LARYNGEAL AND RESPIRATORY RESISTANCE WITH PANTING

In the patients with stable asthma respiratory resistance and Δ sound during breath holding after panting were significantly lower than inspiratory or expiratory respiratory resistance and Δ sound during tidal breathing, both before and after methacholine. In the patients with spontaneous asthma, however, respiratory resistance and Δ sound during breath holding after panting was higher than inspiratory or expiratory resistance and Δ sound during tidal breathing (fig 3).

The changes in respiratory resistance and estimated changes in laryngeal resistance after panting are shown for all experiments in table 2. The changes in respiratory and laryngeal resistances were similar in the patients with stable asthma after methacholine and in those with spontaneous asthma ($p > 0.20$). In the patients with stable asthma before methacholine,

Table 2 Baseline respiratory resistance during inspiration, changes in respiratory resistance, and estimated changes in laryngeal resistance during breath holding immediately after panting from corresponding respiratory resistance and laryngeal resistance during inspiration (mean (SEM) values)

	<i>Stable asthma</i>		<i>Spontaneous asthma</i>
	<i>Methacholine</i>		
	<i>Before</i>	<i>After</i>	
Baseline respiratory resistance during inspiration (kPa/l.s)	0.245 (0.029)	0.470 (0.029)	0.480 (0.049)
Changes in respiratory resistance (kPa/l.s)	− 0.029 (0.008)	− 0.077 (0.014)	0.084 (0.011)
Estimated changes in laryngeal resistance (kPa/l.s)	− 0.080 (0.012)**	− 0.065 (0.010)	0.089 (0.006)

**p < 0.01 for difference between respiratory and laryngeal resistance.

however, the decrease in laryngeal resistance was larger than that in respiratory resistance.

Discussion

Stanescu *et al*⁷ photographed the glottis and simultaneously measured airway resistance during both panting and continuous expiration. They found the glottal aperture to be larger and airway resistance to be smaller at any lung volume during panting than during continuous expiration. Several other studies in normal subjects in the control state have also suggested that the laryngeal aperture is wider during panting than during quiet tidal breathing.^{1,3,9} In our previous study panting constricted the larynx in normal subjects after methacholine and histamine induced bronchoconstriction.³ In the present study laryngeal resistance after panting in the patients with stable asthma was lower before and after methacholine induced bronchoconstriction than during the inspiratory phase of tidal breathing, suggesting that panting opened the larynx in these subjects irrespective of induced bronchoconstriction. Although methacholine and histamine cause laryngeal constriction in association with bronchoconstriction in normal subjects,^{3,8,10,11} methacholine inhalation increased respiratory resistance without changing laryngeal resistance in the present patients with stable asthma. Whether methacholine inhalation gives rise to upper airway narrowing by stimulation of upper airway receptors or by eliciting reflexes secondary to bronchoconstriction is not clear. We previously reported an increase in laryngeal resistance after methacholine challenge in normal but not in asthmatic subjects, despite the greater mag-

nitude of bronchoconstriction in the asthmatic subjects.⁷ These observations suggest that methacholine induces changes in laryngeal resistance in normal subjects by some action on the upper airways. The absence of an increase in laryngeal resistance in asthmatic subjects could be due to failure of the lower concentrations of methacholine inhaled by these subjects to activate the relatively less sensitive upper airway receptors. The lack of laryngeal constriction during quiet tidal breathing in patients with stable asthma suggests that the larynx behaves much the same as in normal subjects.

Laryngeal resistance increased after panting in the patients with spontaneous asthma. Previous reports have suggested that the laryngeal aperture area decreases during spontaneous attack of asthma.^{7,12,13} Thus, in contrast with the patients with stable asthma, the larynx might be narrowed before the panting manoeuvre and the panting manoeuvre might further constrict the larynx in these patients.

Because estimated changes in laryngeal resistance in the interval after panting were roughly similar to changes in respiratory resistance in the patients with stable asthma after methacholine and in the patients with spontaneous asthma, an increase or decrease in laryngeal aperture area would explain the changes in respiratory resistance. In the patients with stable asthma before methacholine, however, the estimated decrease in laryngeal resistance was larger than the decrease in respiratory resistance in the interval after panting. Respiratory resistance measured by the forced oscillation technique includes not only the resistance of the airway but also the tissue resistance of the lung and chest wall. Respiratory muscle contraction might therefore occur during breath holding after panting, resulting in an underestimation of the decrease in respiratory resistance in the patients with stable asthma. This explanation of our findings is unlikely because we observed no discrepancy in the changes in laryngeal and respiratory resistance during breath holding immediately after panting in the patients with spontaneous asthma and in those with stable asthma who had methacholine, who should have required a greater respiratory effort than patients with stable asthma without methacholine. Alternatively, panting might have increased airway resistance below the larynx.

In a previous study⁷ we measured upper airway resistance directly by intratracheal lateral pressure and mouth flow, and examined the relation between the increase in Δ sound and the increase in upper airway resistance during methacholine and histamine inhalation in 10 normal subjects. When respiratory resistance increased approximately twofold upper airway resistance increased in a way that nearly followed the relation observed during voluntary glottal closure.⁴

Furthermore, changes in Δ sound fairly well reflected changes in upper airway resistance during the slow vital capacity manoeuvre in normal and asthmatic subjects.⁷ Changes in functional residual capacity after methacholine provocation and during an attack of asthma probably do not influence the relation between Δ sound and change in upper airway resistance.

The present observations suggest that subglottal intrapulmonary airway obstruction may be overestimated with the panting manoeuvre in patients with spontaneous asthma. It is important to consider the contribution of the larynx in bronchoconstriction when subglottal intrapulmonary airway obstruction is being assessed from measurements of airway resistance in a body plethysmograph.

References

- Spann RW, Hyatt RE. Factors affecting upper airway resistance in conscious man. *J Appl Physiol* 1971;**31**:708–12.
- Higenbottam T, Payne J. Glottis narrowing in lung disease. *Am Rev Respir Dis* 1982;**125**:746–50.
- Sekizawa K, Sasaki H, Takishima T. Laryngeal resistance immediately after panting in control and constricted airways. *J Appl Physiol* 1985;**58**:1164–9.
- Sekizawa K, Shindoh C, Hida W, Suzuki S, Akaizawa Y, Shimizu Y, Sasaki H, Takishima T. Noninvasive method for detecting laryngeal narrowing with low-frequency sound. *J Appl Physiol* 1983;**55**:591–7.
- Goldstein D, Mead J. Total respiratory impedance immediately after panting. *J Appl Physiol* 1980;**48**:1024–8.
- Takishima T, Hida W, Sasaki H, Suzuki S, Sasaki T. Direct-writing recorder of the dose-response curves of the airway to methacholine. Clinical application. *Chest* 1981;**80**:600–6.
- Stanescu DC, Pattijn J, Clement J, van de Woestijne KP. Glottis opening and airway resistance. *J Appl Physiol* 1972;**32**:460–6.
- Shindoh C, Sekizawa K, Hida W, Sasaki H, Takishima T. Upper airway response during bronchoprovocation and asthma attack. *Am Rev Respir Dis* 1985;**132**:671–8.
- Brancatisano TP, Dodd DS, Engel LA. Respiratory activity of posterior cricoarytenoid muscle and vocal cords in humans. *J Appl Physiol* 1984;**57**:1143–9.
- England SJ, Ho V, Zamel N. Laryngeal constriction in normal humans during experimentally induced bronchoconstriction. *J Appl Physiol* 1985;**58**:352–6.
- Higenbottam T. Narrowing of glottis opening in humans associated with experimentally induced bronchoconstriction. *J Appl Physiol* 1980;**49**:403–7.
- Collett PW, Brancatisano T, Engel LA. Changes in the glottic aperture during bronchial asthma. *Am Rev Respir Dis* 1983;**128**:719–23.
- Lisboa C, Jardim J, Angus E, Macklem PT. Is extra-thoracic airway obstruction important in asthma? *Am Rev Respir Dis* 1980;**122**:115–21.
- Cotes JE. *Lung function: principles and application in medicine*. 3rd ed. Oxford: Blackwell, 1979:369.