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Measurement and regulation of nasal airflow resistance in man

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SUMMARY

A method for measuring human nasal airflow resistance (R_{naw}) is described. Air flows at constant pressure through both nasal cavities via a face mask and out through the mouth. Airflow is inversely related to R_{naw}. The method has several advantages over many other methods for measuring R_{naw}, in particular allowing aerodynamic separation of nose and lungs, and frequent measurements over long periods without discomfort to or intervention with subjects or patients. We have used this method to obtain standard values of R_{naw} in healthy subjects and in patients with asthma and/or rhinitis. Age has a negative correlation with R_{naw} but no sexual difference was seen. Cigarette smoking increases R_{naw} especially in young adults. Patients with rhinopathy have much higher resistances than healthy subjects, but those with asthma alone do not. R_{naw} is sensitive to changes in ventilation and lung volumes; deep inspiration and oral hyperventilation decrease R_{naw}, while deep expiration, nasal hyperventilation and breath-holding increase it. Hypoxia and hypercapnia locally applied in the nose increase R_{naw}. It is suggested that these changes are predominantly due to changes in control of the nasal vascular bed.

INTRODUCTION

There is an extensive literature on the physiological control of nasal airflow resistance (R_{naw}), some of which is discussed later.

We have developed a method for measuring human R_{naw} with two aims which are not always met by established methods. One is that there should be minimal invasion of the subject, if possible without tubes inserted into the nose which may be uncomfortable in patients, with use for measurements over long periods (10–30 min) without undue interference with the subject. Secondly, we wished to study the effects of gases and aerosols applied separately into the nose and into the lungs, and most current methods do not permit this. This paper describes the method, the baseline measurements on subjects and patients with respiratory disease, and the effects of some physiological interventions involving respiratory manoeuvres. Some of the results have been briefly reported elsewhere (Bundgaard et al., 1984).

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MATERIALS AND METHODS

The constant pressure method for measuring nasal airflow resistance (R_{naw})

The essence of the method is to blow air through the nose and out through the mouth at constant inflow pressure (usually 70 Pa or 7 mm H_2O) using as modified scuba nasal face mask, while measuring flow rate (Figure 1) (Bundgaard et al., 1983, 1984; Syabbalo, 1984). Nasal airflow is from a box pressurised by a fan. Changes in R_{naw} from normal (about 0.05–0.36 kPa · L^{-1} · s) to infinity did not appreciably alter the pressure in the box, which was continuously monitored. Box pressure changes only by about 4.2% when the mask is completely occluded ($R_{naw} = \infty$) compared with disconnected ($R_{naw} = 0$).

Flow, which if laminar is inversely proportional to R_{naw}, is measured with a Fleisch pneumotachograph and recorded on a chart recorder together with box pressure; they can also be stored on magnetic tape and/or fed to a microprocessor which computes R_{naw}.

Nasal airflow at constant input pressure is sensitive to pharyngeal pressure during breathing, since this would change the transnasal pressure producing flow. Measurements of R_{naw} are therefore, in practice, restricted to respiratory pauses (5–10 s at end-expiration) when the pharyngeal pressure is zero. However, it is possible to compensate for this effect by measuring the pressure at the back of the mouth, and this is done via the microprocessor. Otherwise the method measures

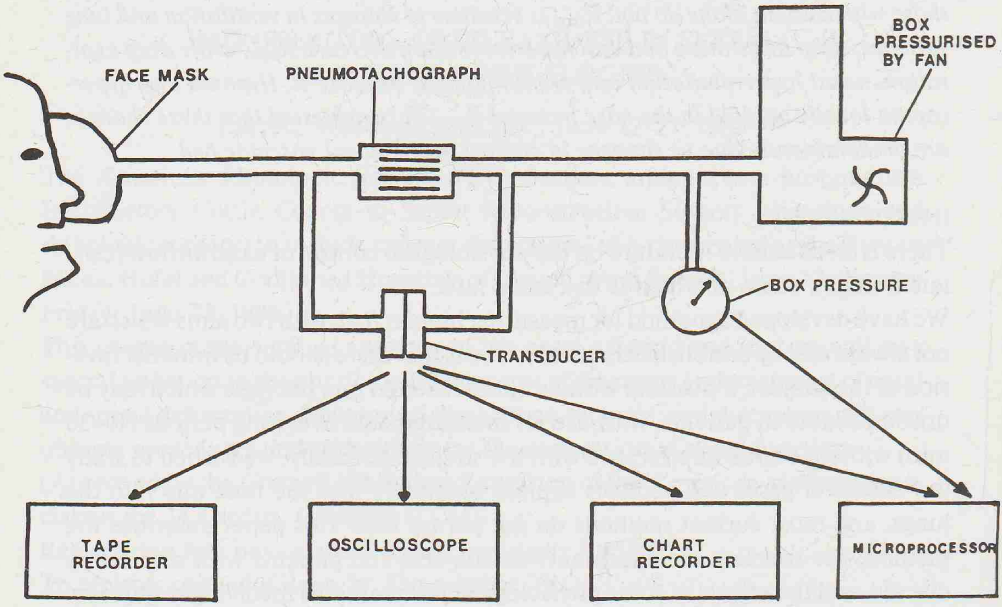

Figure 1. Diagram of method measuring nasal airflow resistance (R_{naw}). For description see text.

nose plus mouth resistance, but the latter is small if the mouth is widely open. The resistance of the tubing connecting the pressure box to the face mask was substracted from measured resistance to give R_{naw}. The system is calibrated by measuring flow with the mask disconnected ($R_{naw} = 0$) and with the mask occluded ($R_{naw} = \propto$).

The maximum flow generated by the pressure box (when $R_{naw} = 0$) can be varied by changing the fan and/or the dimensions of the box and its connections. The pressure boxes we have used generated maximum nasal flows in healthy subjects of 0.25–0.42 $L \cdot s^{-1}$, which is within the physiological range of flows through the nose.

Subjects

The following subjects were studied: (1) a group of 66 healthy subjects aged between 24 and 76 year, mean age 51.6 year. They consisted of subjects who had either come for routine assessment of cardiopulmonary function (found normal) at Rigshospitalet (Copenhagen) of were volunteers at St. George's Hospital Medical School (London); (2) a group of 14 well controlled asthmatic patients, age range 18–64 year, mean age 44.5 year; (3) a group of 13 asthmatic subjects who also had rhinopathy (usually allergic rhinitis), aged between 18 and 62 year; (4) a group of 12 subjects with rhinopathy, eg. common cold, allergic or non-allergic rhinitis, or nasal polyps, aged between 23 and 62 year. The mean weight of all subjects was 68.1 ± 2.1 kg (s.e.m.) and the mean height 165.7 ± 3.7 cm, there being no statistical correlation between weight or height and age, smoking habit or medical condition.

All the asthmatic subjects were withheld theophylline for 24 h and aerosol medication for 8 h before the investigation. The experiments were performed in modern, air-conditioned laboratories with near constant temperature and humidity, and all subjects gave verbal consent to the tests.

After 20 min acclimatisation to temperature and humidity in the laboratory the apparatus and experimental procedures were demonstrated to the subject. The modified scuba nasal face mask was worn by the subjects and control R_{naw} measurements (usually five or more) were obtained. All subjects were seated during the tests.

After the R_{naw} measurements, forced vital capacity (FVC) and forced expired volume in 1 s (FEV_1) were obtained using a spirometer, and sometimes peak expiratory flow rate (PEFR) was obtained using a Wright's flowmeter.

Changes in lung volume

Healthy subjects sat relaxed on a comfortable laboratory chair and were allowed 15–20 min to acclimatise to laboratory conditions. After control R_{naw} measurements at functional residual capacity (FRC), the effect of a deep inspiration to

total lung capacity (TLC), and the effect of a deep expiration down to residual volume (RV) were assessed. The subjects were instructed to pause at the end of the deep inspiration or at the end of the deep expiration. The lung volume changes were held for 5–10 s and R_{naw} was measured then and at intervals of 30 s up to 2 min afterwards at FRC. The experimental procedures were repeated randomly several times with intervals of 2 min between each deep inspiration or expiration, and the mean effects of deep inspiration and expiration were calculated.

Oral and nasal voluntary hyperventilation

After control R_{naw} measurements healthy subjects voluntarily hyperventilated at maximal ventilatory capacity (MVC) for 1 min either through the mouth or through the nose, as instructed. R_{naw} measurement at FRC were performed immediately, 30, 60 and 90 s after the manoeuvres.

Breath-holding

After control R_{naw} measurements at FRC healthy subjects held their breath at TLC to breaking point (range 35–150 s). R_{naw} was then measured at FRC immediately, 30, 60, 90 and 120 s after breath-holding.

Different gas tensions in the nose

The following gas mixtures (BOC) were used in healthy subjects: (a) air; (b) 10% O_2 and 90% N_2; and (c) 5% CO_2, 20% O_2 and 75% N_2. The gas mixtures were kept in thin meteorological bags with low internal resistance and which did not appreciably affect the constant pressure in the pressure box which was modified into a double box to allow the bags to be fitted at the rear. The gas mixtures were kept at room temperature.

Control R_{naw} measurements were obtained over a period of 2 min; the bag containing the gas mixture was then connected and measurements of R_{naw} were obtained over a period of 2 min, and for another 2 min after the bag had been removed. All measurements were at FRC. All the gas mixtures were randomised and none of the subjects was aware which gas mixture was being applied.

Results are analysed statistically for groups of subjects by Student's paired t-test. Significances of responses in individual subjects is indicated where appropriate. $P < 0.05$ is taken as statistically significant.

RESULTS

Factors affecting control R_{naw} in healthy subjects

The mean control R_{naw} in 66 healthy subjects was 0.26 ± 0.020 kPa · L^{-1} · s. There was a statistically significant inverse relationship between age and R_{naw}, which decreased with age in both males and females (Table 1, Figure 2). There was also a statistically significant decrease with age in PEFR and FEV_1/FVC (Table 1).

Table 1. Effects of age on nasal resistance of smokers and non-smokers.

age group (years)	n	S/NS	R_{naw} (kPa · L^{-1} · s)	FEV_1/FVC (%)	PEFR (L · min^{-1})
20–40	10	S	0.44 ± 0.063	84.2 ± 1.8	533 ± 37
	12	NS	0.22 ± 0.034**	82.8 ± 2.9	522 ± 27
	22	S + NS	0.32 ± 0.041	83.4 ± 2.9	528 ± 38
40–60	17	S	0.28 ± 0.068****	75.3 ± 2.0***	383 ± 25****
	9	NS	0.23 ± 0.037	83.7 ± 1.7*	440 ± 39***
	26	S+NS	0.26 ± 0.027	77.7 ± 1.7	400 ± 22****
over 60	11	S	0.18 ± 0.041****	71.3 ± 2.7****	393 ± 33****
	7	NS	0.18 ± 0.046	74.4 ± 4,1***	329 ± 19***
	18	S + NS	0.18 ± 0.030****	72.6 ± 2.3****	366 ± 23****

S = smokers, NS = non-smokers
Values are means ± s.e.m.
* $p < 0.05$
** $p < 0.01$ for non-smokers compared with smokers
*** $p < 0.05$
**** $p < 0.01$ for higher age groups compared with 20–40 years.

Although R_{naw} was higher in males than in females (0.29 ± 0.026 compared with 0.23 ± 0.028 kPa · L^{-1} · s, n = 28 and 38 respectively), the difference was not statistically significant, despite the greater weight of the males (79.1 ± 3.3 compared with 61.4 ± 1.6 kg); larger body size might be expected to correspond to smaller R_{naw}. Women also had significantly lower PEFRs (352 ± 15.0 against 488 ± 24.1 L · min^{-1}), but there was no significant difference between FEV_1/FVCs for the two sexes.

There was no significant correlation between body weight or height and R_{naw}.

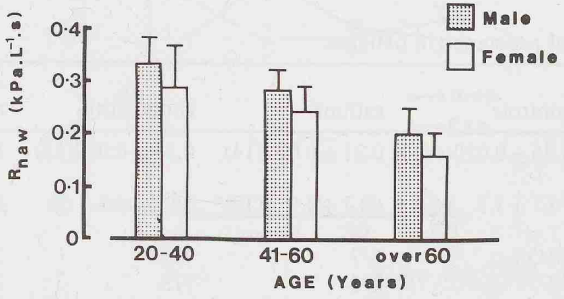

Figure 2. Effect of age on nasal airflow resistance (R_{naw}). Healthy subjects were divided into three age groups. R_{naw} decreased with age. Values are means and s.e.m.s. There were no statistically significant differences between males and females, but the over-60 group had significantly ($p < 0.05$) lower R_{naw} than the 20–40 year old group.

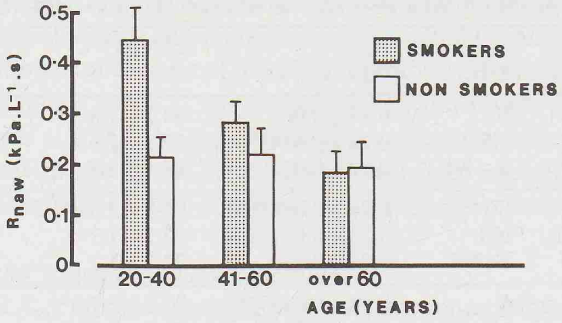

Figure 3. Effect of smoking on nasal airflow resistance (R_{naw}) for different age groups of healthy subjects. Only in the lowest age group was there any significant difference, smokers having a higher R_{naw} than non-smokers. For statistical analysis see Table 2.

There was a weak ($r = 0.336$) but significant ($P < 0.05$) correlation between PEFR and $R_{naw} = 0.0049/PEFR + 0.58$) but there was no but significant correlation between FEV_1/FVC and R_{naw}.

Smokers had a significantly higher R_{naw} than did non-smokers (Figure 3), the effect being greater in the young age group (20–40 year). In the oldest age group (over 60 year) the effect seemed to be absent. Smokers had a significantly lower FEV_1/FVC compared with non-smokers (74.9 ± 1.64 and $80.1 \pm 2.2\%$ respectively), especially in the middle age group, but PEFRs were not significantly different. Table 1 analyses results.

As expected, patients with rhinopathy had a higher R_{naw} than controls (Table 2). Their FVC, FEV_1 and FEV_1/FVC were normal. R_{naw} was highest in subjects with acute nasal symptoms, eg. a recent "cold".

The man R_{naw} in asthmatic patients was lower than that of healthy subjects, but there was no significant difference between these two values (Table 2). Only two

Table 2. Nasal resistance in patients.

	controls	asthma	rhinopathy	asthma and rhinopathy
R_{naw} (kPa · L^{-1} · s)	0.26 ± 0.020(66)	0.21 ± 0.027(14)	0.54 ± 0.082(12)	1.01 ± 0.028(13)**
FEV_1/FVC (%)	76.7 ± 1.3 (49)	60.7 ± 4.9 (10)*	78.9 ± 4.5 (8)	45.3 ± 4.0 (6)**
male/female	28/38	7/7	7/5	2/13
smokers/ non-smokers	38/28	2/14	7/5	2/13

Values are mean $\pm$ s.e.m.; n-values in parentheses;
*$p < 0.001$ for control compared to asthmatics
**$p < 0.05$ for asthmatics compared with asthma and rhinopathy patients.

of the asthmatics smoked and there was almost no difference in R_{naw} in asthmatics compared with non-smokers (0.206 and 0.211 kPa · L^{-1} · s respectively). FEV_1/FVC was significantly lower in asthmatics than in healthy subjects.
Asthmatic patients with rhinopathy had high R_{naw}s (Table 2). Some subjects in this group had nasal polyps. This group of subjects also had significantly lower FEV_1/FVC values compared with the patients with asthma alone.

Day-to-day variation in R_{naw}
There was day-to-day variation in R_{naw}, the variation being more marked in patients with asthma and/or rhinitis than in healthy subjects. The mean percentage standard deviations (and their s.e.m.'s) of the four groups are: healthy $12.2 \pm 0.51\%$; asthmatics $22.2 \pm 4.8\%$; asthmatics with rhinitis $62.5 \pm 16.7\%$; rhinopathy alone $78.8 \pm 45.5\%$.

Respiratory manoeuvres and R_{naw}
Deep inspiration up to TLC in healthy subjects significantly decreased R_{naw} by $15.1 \pm 4.2\%$ (Figure 4). Thirty-two tests were done on six subjects, and in four the mean changes were statistically significant. One subject with rhinitis showed a fall in R_{naw} on deep inspiration. Forced expiration down to RV had the opposite effect, increasing R_{naw} by $40.3 \pm 13.2\%$ (Figure 4). Fourteen tests were done on seven subjects, and in all the increases were statistically significant. Both the effects were shortlived, R_{naw} returning to its pre-test value within 30–60 s. The decrease in R_{naw} during maximal inspiration was positively related to the initial R_{naw}

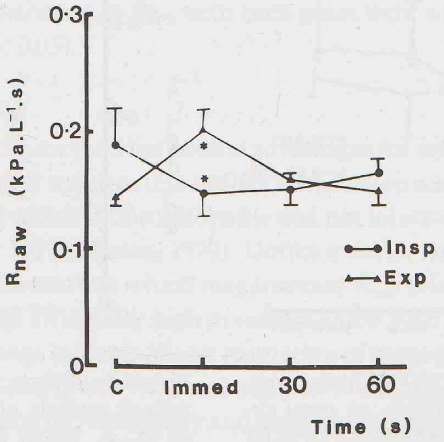

Figure 4. Effects of deep inspiration and deep expiration on nasal airflow resistance (R_{naw}). Values are given for controls before intervention, R_{naw} immediately after, and 30 and 60 s after deep inspirations and expirations, the immediate values being obtained at the inspiratory and expiratory lung volumes. Asterisks indicate significant ($p < 0.05$) changes of the groups of subjects from control.

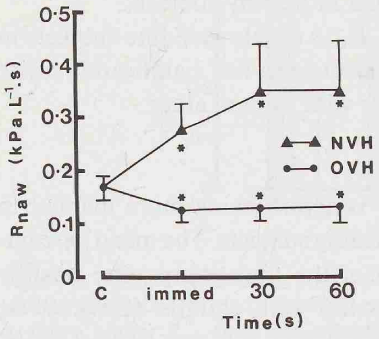

Figure 5. Effect of oral hyperventilation (OHV) and nasal hyperventilation (NHV) on nasal airflow resistance (R_{naw}). Values are controls before hyperventilation, and immediately after 2 min hyperventilation and at 30 and 60 s later. Oral hyperventilation significantly decreased and nasal hyperventilation significantly increased R_{naw}.

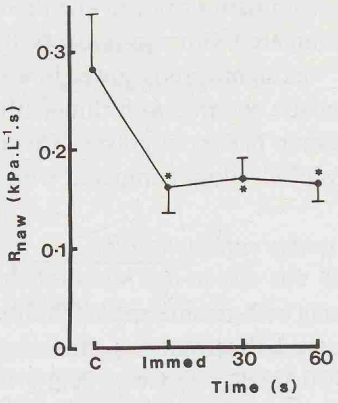

Figure 6. Effect of breath-holding to breaking point on nasal airflow resistance (R_{naw}). Breath-holding was at total lung capacity. Measurements given are the control before breath-holding, and the values immediately after and at 30 and 60 s after, all values being at FRC. There was a significant and maintained decrease in R_{naw} after breath-holding.

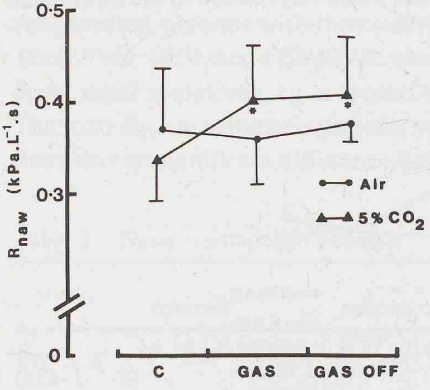

Figure 7. Effects of 5% CO_2 in the nose, compared with air controls, on nasal airflow resistance (R_{naw}). Values are given for average controls before, at the end of 2 min exposure to the gas, and at the end of 2 min after removal of the gas. Compared with air controls, 5% CO_2 caused a significant and maintained increase in R_{naw}.

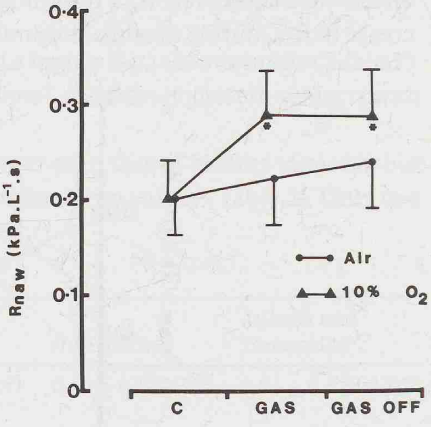

Figure 8. Effects of 10% O_2, compared with air controls, on nasal airflow resistance (R_{naw}). Values are given for controls, maximum response at the end of 2 min exposure to hypoxia, and response at the end of 2 min after removal of the gas. Compared with air controls, 10% O_2 caused a significant increase in R_{naw}.

($p < 0.05$). In four subjects both deep inspirations and deep expirations were tried, and each of them showed decreases and increases in R_{naw} respectively.
Oral voluntary hyperventilation for 1 min decreased R_{naw} by $26.6 \pm 8.3\%$ (Figure 5). Six healthy subjects were studied and all showed a fall in R_{naw}. Nasal voluntary hyperventilation had the opposite effect, causing an increase of $61.5 \pm 22.2\%$ (Figure 6). Six of seven subjects showed this effect. The responses lasted more than 1 min.
Breath-holding at TLC to breaking point (35 s to 2.5 min) significantly decreased R_{naw} by $37.7 \pm 8.9\%$ (Figure 7). This was done in six healthy and four rhinitis subjects, and all but one rhinitis subject decreased R_{naw}. The effect lasted for over 1 min. The decrease in R_{naw} following breath-holding was positively related to the initial R_{naw} ($p < 0.05$).

Nasal gas mixtures and R_{naw}
Local application to the nose of 5% CO_2, 20% O_2 and 75% N_2 significantly increased R_{naw} by $20.49 \pm 5.81\%$, while air controls caused a decrease of $2.66 \pm 7.23\%$ (Figure 7). The test was done 12 times in seven subjects, five of whom had rhinitis, and all subjects showed an increase in R_{naw} with CO_2. The responses persisted 2 min after the CO_2 was discontinued.
10% O_2 significantly increased R_{naw} by $39.17 \pm 4.28\%$, whereas air controls caused an increase of $7.23 \pm 5.23\%$ (Figure 8). The test was done 13 times in eight subjects, three of whom had rhinitis, and all of them showed an increase in R_{naw} greater than for air controls. The responses persisted 2 min after the hypoxia was discontinued. The effect of hypoxia in the nose was significantly larger than that of hypercapnia. The increases in R_{naw} with both gases were significantly related to the initial R_{naw} ($p < 0.05$).

DISCUSSION

The constant pressure method has several advantages for measuring R_{naw}. Unlike posterior rhinometry it requires less subject cooperation and does not require an oropharyngeal tube which is uncomfortable and not tolerated by 50–60% of subjects (Kortekangas, 1972; Masing, 1979). Unlike anterior rhinometry it does not insolve nasal instrumentation which may increase R_{naw} (Haight and Cole, 1984) and does not require artificially high pressures when both nostrils are blocked. A particular advantage is that it allows restriction of gases and aerosols either to the nose or to the lungs, with minimal entry to the other site.
In common with posterior rhinometry and most forms of anterior rhinometry, the measured R_{naw} includes the resistance of the nares, the nasal cavities and the nasopalatine orifice. It can be argued that this total resistance is what the respiratory apparatus has to overcome in breathing, but of course the control mechanisms for different parts of the total nasal flow-channel are different. Some of the

manoeuvres we have tested, for example deep inspiration and deep expiration, undoubtedly modify the position of the palate during the manoeuvre, but there is no evidence that nasopalatine resistance is appreciably changed afterwards.
Although our method would allow measurement of resistance through each nasal cavity separately, we have not extended it to this application. Therefore our results can give no information about the nasal cycle. Again, although it seems probable that most of the physiological responses we have described are due to changes in the nasal vasculature, what is happening in individual components of this complex system is not clear, and discussion of this topic would be speculative.
The range of R_{naw} in healthy subjects, quoted in the extensive literature with many methods, is between 0.05 and 0.40 kPa · L^{-1} · s with means from 0.12 to 0.37 kPa · L^{-1} · s. Our results fit well into this range. Three of the most recent studies are by Broms (1980) who found 0.36 kPa · L^{-1} · s in 37 healthy subjects, by Forsyth et al. (1983) who found 0.25 kPa · L^{-1} · s in 20 healthy subjects, and by Ghaem and Martineaud (1985) who found 0.19 and 0.096 kPa · L^{-1} · s at a flow of 0.5 L · s^{-1} in 32 subjects with posterior and anterior rhinometry respectively.
As would be expected, R_{naw} is highest in newborn babies (Polgar and Kong, 1965; Stocks and Godfrey, 1978) due to the anatomy and the reduced dimensions of the nose and nasopharynx. R_{naw} almost reaches the adult value at 16 year (Saito and Hishihata, 1981). In adults our results show that it significantly decreases from 20 to 60 year and presumably this is due to mucosal atrophy.
We were unable to show any significant sexual difference in R_{naw}, although the mean value was lower for females despite smaller body size. This confirms other studies (Konno et al., 1982; Saito and Nishihata, 1981), although Ghaem and Martineaud (1985) found a higher R_{naw} in females than in males. We found no correlation between body weight and R_{naw}, as is also true in infants and children (Saito and Nishihata, 1981; Stocks, 1980), or between height and R_{naw}, also confirming other studies (Broms, 1980; Konno et al., 1981; Saito and Nishihata, 1981; Stocks and Godfrey, 1978). There was, however, a weak but significant correlation between PEFR and R_{naw}, although not between FEV_1/FVC and R_{naw}.
Our results show that smokers have a higher R_{naw} especially for young adults. In the elderly there was no significant difference in R_{naw} between smokers and non-smokers; indeed R_{naw} was slightly lower in smokers, indicating that cigarette smoking may accelerate atrophy of the nasal mucosa. The acute effect of cigarette smoking may be a decrease or increase in nasal blood flow depending on the nicotine level of the cigarette (Friedell, 1953). We did not study enough subjects to allow a distinction between current smokers and ex-smokers, but our results suggest that this distinction would only be important in the younger age groups.
In 1972, Nolte reported an increased R_{naw} in patients with chronic bronchitis and silicosis. As there is a strong association between chronic bronchitis and cigarette

smoking, the basic cause was not clear. In our study there was no significant difference in R_{naw} between asthmatics and healthy controls (smokers and/or non-smokers). Few of the asthmatics smoked. Cohen (1969) found that patients who responded to bronchodilator drugs had higher R_{naw}s than normal. Cohen's patients actually had chronic bronchitis and obstructive emphysema and were similar to those of Nolte (1972). The high R_{naw} in these patients may have been due to cigarette smoking or to nasal inhalation of industrial pollutants.

R_{naw} was high in subjects with rhinopathy, obviously as expected, whether or not they also had asthma. This finding confirms that of Ogura (1970) who showed a correlation between chronic nasal obstruction and lower airways obstruction. However, Table 2 shows that asthma with a low FEV_1/FVC can coexist with normal R_{naw}, and rhinopathy can coexist with normal FEV_1/FVC .

Repeated daily measurements of R_{naw} showed far greater lability in patients with rhinitis than in those with asthma alone, and in the latter compared with healthy subjects. Similar findings have been described by other workers (Butler, 1960; Cole et al., 1980). However, no attempt was made to standardise time of day or day of week.

Respiratory manoeuvres and R_{naw}

In four of six subjects in our study, deep inspiration up to TLC decreased R_{naw}. (A similar response was seen in one patient with rhinitis). The effect lasted less than 30–60 s. The most likely mechanism is an increase in nervous activity ('inspiratory drive') to the nasal vasculature, although the involvement of nasal muscles (nares and palatine) was not eliminated. During inspiration there is an increase in the activity of the cervical sympathetic nerve (Adrian et al., 1932; Cohen, 1979; Eccles and Lee, 1981; Joels and Samueloff, 1956) concurrent with nasal vasoconstriction (Eccles and Lee, 1981). Another possibility is decongestion of nasal sinusoids due to a decrease in jugular venous pressure as a result of a fall in intrathoracic pressure.

Deep expiration down to residual volume increased R_{naw} in all our subjects, the effect lasting less than 60 s. The mechanisms may be a decrease in sympathetic nervous "inspiratory drive" during expiration, or an increase in intrathoracic pressure, decreasing jugular venous return and causing congestion of nasal sinusoids, as has been described elsewhere (Mink, 1920). It is unlikely that this effect is due to an action of alae nasi muscles or upward movement of the soft palate because during expiration the nares and nasopharynx dilate (Bridger and Proctor, 1970; Kortekangas, 1972).

Breath-holding to breaking point decreases R_{naw} (Dallimore and Eccles, 1977, Hasegawa and Kern, 1978; Tatum, 1923), and we have confirmed this observation. The mechanism may be hypercapnia and hypoxia which stimulate central and peripheral chemoreceptors respectively and produce reflex sympathetic nasal

vasoconstriction. The response is abolished by cervical sympathectomy (McCaffrey and Kern, 1979; Tatum, 1923).

Tatum et al. (1923) found that "over-ventilation" produced a very distinct sense of increased resistance to the nasal airway passages. Dallimore and Eccles (1977) showed that maximal oral and nasal hyperventilation in three subjects for 3 min, the maximum time which could be tolerated without too much discomfort or syncope, caused an increase in R_{naw}. The effect was more marked with oral hyperventilation, possibly because high flow rates were difficult with nasal hyperventilation. They proposed that the increase in R_{naw} was due to a fall in PCO_2 but not regulated by the sympathetic nervous system because the response was not abolished by cervical sympathectomy (Tatum, 1923).

Hasegawa and Kern (1978) also reported that hyperventilation increased R_{naw} in 50–60% of subjects and either no change or a decrease in the rest. Hyatt and Wilcox (1961) reported a decrease in upper airway resistance during hyperventilation, which presumably might involved a decrease in R_{naw}. Most of these studies do not indicate if the hyperventilation was nasal or oral or both.

We found that oral hyperventilation decreased R_{naw}, an effect which is similar to that seen with physical effort (Dallimore and Eccles, 1977; Forsyth and Cole, 1983; Hasegawa and Kern, 1978; Konno et al., 1981; Richerson and Seebohm, 1968; Syabbalo, 1984; Syabballo et al., 1985). On the other hand nasal hyperventilation increased R_{naw}, a similar effect to that seen by most other authors. There are several possible mechanisms. Oral hyperventilation, like exercise or deep inspiration (see above), may be associated with an increase in "inspiratory activity" via the cervical sympathetic nerve to the nasal vessels causing vasoconstriction and a decrease in R_{naw}. It is unlikely that the hypocapnia and hyperoxia associated with hyperventilation would cause the decrease in R_{naw}, since hypercapnia and hypoxia decrease R_{naw} in animals and man (Dallimore and Eccles, 1977; McCaffrey and Kern, 1979a, b). It is also unlikely that the increase in R_{naw} is due to a local effect of hypocapnia in the nose, as suggested by others (Dallimore and Eccles, 1977; Hasegawa and Kern, 1978) since we found that local hypercapnia increased R_{naw}.

Although oral hyperventilation decreased R_{naw}, nasal hyperventilation increased it. The latter response could be due to a dominant action from nasal airflow nervous receptors, an effect similar to sniffing which is known to increase R_{naw} (Zanjanian, 1975). Cooling the nose by evaporation may also increase R_{naw} (Cole, 1982), possibly by reflex pathways. The same nerve endings could cause a reflex increase in mucus secretion which would increase R_{naw}. Such mechanisms could explain the difference between oral and nasal hyperventilation in our results.

Local nasal gas tensions and R_{naw}

The local effect of hypoxia and hypercapnia in the nose on R_{naw} does not seem to

have been investigated previously, although there are many reports of the effects of systemic hypoxia and hypercapnia (see above) which lower R_{naw}. Our results are consistent with a local vasodilator action of hypercapnia and hypoxia in the nose, as for most other vascular beds. We cannot say which particular blood vessels are involved, although presumably there is mucosal hyperaemia. This vasodilatation may presumably be overpowered by the vasoconstrictor sympathetic reflex from the chemoreceptors during systemic hypoxia and hypercapnia. Our method would allow a small proportion of the gas mixture applied to the nose to enter the lungs and exert systemic actions but, as indicated above, these would be in the opposite direction to those observed. Most studies on the effect of blood gas tensions on R_{naw} have been with oral breathing of gas mixtures, and this would prevent or minimise any direct action of the gases on the nose.

RÉSUMÉ

Une méthode de mesure de la résistance nasale à l'écoulement de l'air (R_{naw}) est décrite. L'écoulement de l'air dans les deux cavités nasales est causé par une pression constante dans un masque couvrant la face. L'air sort par la bouche. Le débit d'air est inversement lié à R_{naw}. Cette méthode a plusieurs avantages par rapport à beaucoup d'autres méthodes de mesure; en particulier elle permet la séparation de l'aérodynamique du nez et des poumons et des mesures fréquentes pendant de longues périodes sans inconfort pour les sujets ou les malades et sans intervention sur eux. Il y a une corrélation négative entre l'âge et R_{naw} mais il n'y a pas de différence liée au sexe. Fumer la cigarette cause l'augmentation de R_{naw} surtout chez de jeunes adultes. R_{naw} est beaucoup plus grande chez les malades atteints de rhinopathie que chez les sujets bien portants mais ne l'est pas chez les malades ayant un asthme isolé. R_{naw} est affecté par les modifications de la ventilation et des volumes pulmonaires; l'inspiration profonde et l'hyperventilation par la bouche font baisser R_{naw}, tandis que l'expiration profonde, l'hyperventilation par le nez et l'apnée la font augmenter. L'hypoxie et l'hypercapnie localisées au nez font augmenter R_{naw}. Notre suggestion est que des modifications du contrôle au lit vasculaire du nez sont la cause prédominante de ces modifications.

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