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# Nasal resistance in recumbency and sleep*

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SUMMARY

*Nasal resistances to respiratory airflow were measured by computer-assisted rhinomanometry in 21 adult males without major clinical nasal pathology. Measurements were obtained when seated and repeated on assumption of recumbency and during sleep. Resistance in Pa/cm$^3$/s of subjects (n=21) increased from a mean (±SD) of 0.14±0.07 in seated posture to 0.35±0.32 in recumbency. In the majority of subjects the increase was modest and was unaffected by sleep. It is suggested that unrecognized mucosal abnormality with resulting impairment of vascular tone or minor structural deviation of the nasal septum could account for the few cases of marked elevation of nasal resistance we observed in recumbency.*

*Key words: nasal resistance, nasal airflow, nasal obstruction*

## INTRODUCTION

Although it is not uncommon for patients to present with the complaint of nasal obstruction in recumbency, few healthy subjects are aware of changes in nasal airflow resistance, which have been shown to accompany alterations in posture.

In healthy noses, Haight and Cole (1989, 1984) have demonstrated marked airflow resistive responses of the separate nasal cavities to lateral recumbent postures, to pressures on the body surface, and also with the spontaneous nasal cycle. In each of these cases, Cole and Haight (1986) demonstrated that reciprocity of resistance between sides maintained a fairly constant resistance of the combined nasal cavities, which might partially explain the subject's lack of awareness of the alterations in resistance to respiratory airflow.

Reports by Hasegawa (1982) and Rundkrantz (1964) of nasal resistive responses indicate that infection and allergy can promote severe bilateral obstruction in recumbency, a response that is much less evident in healthy noses.

Our studies of airflow resistances in sleep have led to an accumulation of data concerned with nasal resistance to respiratory airflow in seated, recumbent and sleeping subjects. Results in subjects with noses unremarkable to clinical examination are reported and discussed in this communication.

## METHODS

Twenty-one symptomless adult male volunteers aged 22 to 76 years (mean(±SD): 40±13 years), whose nasal cavities were unremarkable to rhinoscopic examination were subjects of the investigation.

Nasal airflow resistances were measured with each subject in a seated posture and then repeatedly in dorsal recumbency, awake, and asleep.

In the seated position, rhinomanometry was performed using the head-out body plethysmograph described by Cole and Havas (1987). During recumbency, awake, and asleep nasal airway resistances were measured by carefully placing a modified CPAP mask as pneumotachograph. Sleep monitoring was done according to standard procedure for this laboratory as detailed by Hoffstein et al. (1991). The polysomnographic chart together with pressure-flow curves from the nasal airway were observed continuously by the investigators and a sleep technologist. Subjects were allowed 30-min acclimatization to laboratory conditions and changes in body position before recordings were done. During sleep airway data were collected when polysomnography showed that subjects were stable within a sleep stage. Data were collected every 5 min or whenever changes in nasal properties or sleep stage took place. For every recording, the value of nasal resistance was a mean of three consecutive measurements with a coefficient of variation <5%. The complete experimental set-up is described elsewhere in a communication by Miljeteig et al. (1993). Prior to the study, validation of recordings from the mask was done by performing 27 paired concurrent measurements with the head-out plethysmographic box. In four subjects, nasal resistance was varied by inserting moist cotton in the nares. Each pair of measurements was averaged from three consecutive readings with a coefficient of variation <5%. Statistical analysis revealed a high degree of correlation between the two systems, close to the line of identity (correlation coefficient 0.994; $r^2$=0.998).

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Changes in sleep stage occurred very quickly. Acclimatization could not be allowed, thus airway data were collected when we were able to identify sleep and stability within a sleep stage.

## RESULTS

In this group of subjects we found the average nasal resistance in the seated position to be 0.14±0.07 Pa/cm$^3$/s. In the recumbent posture nasal airflow resistance increased significantly to 0.35±0.32 Pa/cm$^3$/s (t-test, $p < 0.005$). Too few observations in different sleep stages unabled us to calculate inter-stage differences. One subject was not able to fall asleep and is not included in the statistical calculations. Figure 1 shows the distribution of nasal resistance in all 21 subjects. It is noted that all but two subjects had an increase in nasal resistance on assumption of recumbency, although in most of them the increase was modest and resistance remained within normal limits.

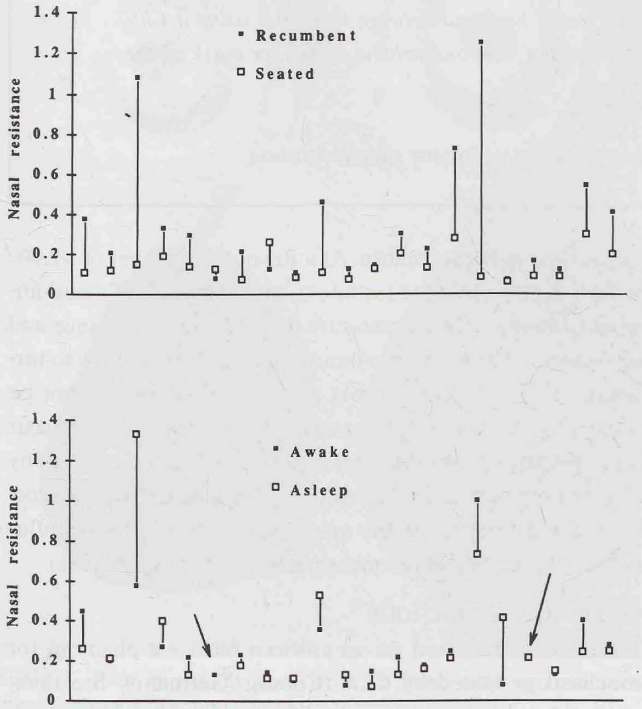


Figure 1. Awake, seated *versus* recumbent (upper panel) and recumbent, awake *versus* asleep (lower panel) nasal resistances (Pa/cm$^3$/s) in 21 subjects. Short arrow indicates subject that not was able to fall asleep. Large arrow indicates subject with identical values for wakefulness and sleep.

## DISCUSSION

Our clinical rhinomanometric experience with many thousands of subjects over the past 10 years indicate that computer-averaged nasal resistance to respiratory airflow of seated healthy adults is usually <0.2 Pa/cm$^3$/s and rarely exceeds 0.25 Pa/cm$^3$/s. Nasal resistances of seated subjects reported here averaged 0.14±0.07 Pa/cm$^3$/s (n=21). Most subjects demonstrated an increase in airflow resistance on assumption of a recumbent posture, but in most cases the increase was modest (Figure 1) and no statistical significant difference was found between wakefulness and sleep. Too few observations in each sleep stage unabled us to compare interstage differences statistically.

Since it has been shown that nasal mucosal diseases can promote nasal obstruction in recumbency it seems reasonable to assume that the nasal mucosa of the few subjects who demonstrated marked elevation of resistance was not entirely normal. Indeed, rhinitis is very common and early stages of onset, late stages of recovery or a mild attack might be unrecognized on clinical examination and yet cause paresis of capacitance vessels, as noted by Rundkrantz (1964), and this may lead to hydrostatic postural change in blood content with resulting elevations of resistance in recumbency. It is also known that mucosal activity decreases by age. Some of the huge intersubject variations may be explained by this. Indeed, the older subjects showed less response when assuming recumbent posture.

Clinically significant structural abnormalities were not detected in our subjects, but insignificant structural airway narrowing, especially at restricted sites (e.g., the valve region) can cause ipsilateral and in some cases bilateral obstruction as a result of even moderately increased congestion depending upon posture or the nasal cycle as described by Cole and Haight (1986).

The importance of partial nasal obstruction in relation to snoring and sleep-disordered breathing is described in communications by Miljeteig et al. (1992) and Metes et al. (1992), but has not yet been conclusively established. Our investigations suggest that in the majority of healthy noses marked elevation of respiratory airflow resistance does not result from recumbency and sleep.

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