Rhinology, 25, 245-248, 1987

The effect of alcohol ingestion upon nasal airway resistance

R. Eccles and N.S. Tolley, Cardiff, United Kingdom

SUMMARY

The effect of ingestion of moderate amounts of alcohol, on nasal airway resistance, was investigated in eight normal human subjects. Alcohol was found to significantly increase both inspiratory and expiratory nasal airway resistance, $P<0.01$. The implications of this finding are discussed, in terms of its relevance to the obstructive sleep apnoea syndrome.

Studies have shown that alcohol increases the incidence of snoring and sleep apnoea, (Issa and Sullivan, 1982). Approximately 20% of the adult population snore (Rice, 1986), and it has been shown that alcohol ingestion can lead to the appearance of frank obstructive sleep apnoea (OSA), in snorers (Scrima et al., 1982). Symptoms of nasal stuffiness associated with alcohol consumption are well documented in ENT practice.

There is now much evidence showing that nasal obstruction is associated with an increased tendency towards developing OSA (Wynne, 1983).

Previous studies by Robinson et al. (1985) have shown that alcohol can increase nasal resistance when adopting a sitting posture. It is well known that posture can exert a profound influence upon nasal airway resistance (Cole and Haight, 1984). The aim of this study was to assess the effects of alcohol consumption upon nasal airway resistance, when adopting a supine posture. Since by definition sleep apnoea occurs when lying down, we feel that possible changes in nasal resistance when adopting the supine posture, will be of more relevance to the sleep apnoea situation. Effects of alcohol upon nasal resistance when adopting a supine posture has to our knowledge, not been previously investigated.

METHODS

Seven subjects were chosen for the study (six male, one female, age range 18-20, mean age 18.5), drawn from the university student population. Subjects gave no past history of nasal problems, and none had suffered from a coryzal illness during the two weeks leading up to the study. Full rhinoscopic examination was performed upon all subjects.

Nasal resistance was measured by active anterior rhinomanometry, using a 150

Pascal sample pressure (Mercury Electronics, Glasgow, U.K.). Total nasal resistance was calculated from the formula R = P/V, and then with application of OHM's law for resistances in parallel.

All resistance measurements were performed with the subject in the supine position, as this would be the most appropriate posture to adopt, for comparison with nasal resistance changes in the sleep situation.

All subjects were instructed not to eat or drink for four hours prior to the study. Alcohol was given on a weight percentage basis (2.0 mls/kg body weight, maximum 160 mls of 40% (% volume)), in the form of vodka, with a small amount of orange juice added to improve palatability. None of the subjects were aware of the aims of the study. The protocol was as follows.

Subjects were instructed to blow their nose to remove extraneous secretions prior to the study. Subjects were then asked to lie supine, and a period of five minutes was allowed for postural changes in nasal resistance to stabilise. Recordings of inspiratory and expiratory resistance were then made. Sixteen separate recordings were made, in batches of four. If a range of greater than 0.1 $Pa/cm^3/s$ was found to occur in a given batch, these would be rejected, and measurements repeated. Total variability between the 16 measurements of resistance was therefore no greater than 0.1 $Pa/cm^3/s$.

After control measurements, subjects were taken to a separate room and instructed to drink their designated amount of alcohol within 30 minutes. A further period of 60 minutes was then allowed for absorption to take place. During this period subjects were told to sit quietly.

Resistance measurements were then repeated in a similar fashion to the control, and a blood sample was taken immediately after the last resistance recording, to provide an objective index of alcohol absorption.

Blood alcohol was measured by using the Lion laboratories, Head-Space Alcometer (AED3).

RESULTS

In six of the seven subjects ingestion of alcohol caused an increase in calculated total nasal resistance as measured in the supine position. The effects of alcohol on total inspiratory resistance in the seven subjects are illustrated in Figure 1. Similar changes in expiratory resistance were also found, but only the results for inspiratory resistance are presented.

The results were pooled for statistical analysis and it was found that the mean inspiratory total nasal resistance increased from 0.31 $Pa/cm^3/s$ +/− 0.02 (+/− s.e.m.) to 0.46 $Pa/cm^3/s$ +/− 0.05 (+/− s.e.m.), 90 minutes after ingestion of alcohol. The difference between the control and post alcohol resistance values was significant ($P < 0.01$), when analysed by application of the paired Student t-test. The individual blood alcohol levels are illustrated in Figure 1, and the mean

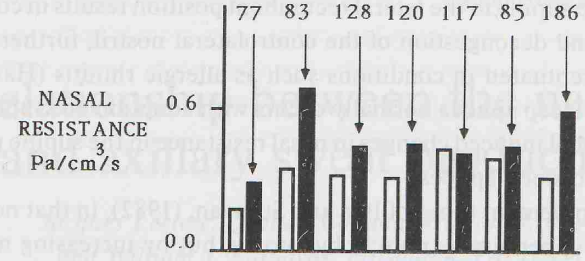

Figure 1. The effects of alcohol on inspiratory nasal resistance in supine subjects. Nasal resistance values are shown prior to ingestion of alcohol (unshaded columns) and 90 min after ingestion of alcohol (shaded columns). Each resistance value is the mean value of sixteen measurements. The blood levels of alcohol (mg/%) for each subject 90 min after ingestion are shown above each post alcohol resistance value.

alcohol blood level was 113 mg% +/− 15.5 (+/− s.e.m. n=7.) 90 minutes after ingestion of alcohol.

No correlation was found between the increase in nasal resistance and blood alcohol levels.

DISCUSSION

The results clearly show that ingestion of alcohol causes an increase in nasal airway resistance in supine subjects. This finding is of particular importance in subjects who are prone to sleep apnoea, as an increase in nasal resistance may increase the tendency towards developing sleep apnoea by two mechanisms. Firstly, higher negative pharyngeal pressures will ensue, and this will generate greater collapsing forces upon the airway. Secondly, nasal obstruction will diminish the contribution of upper airway afferents, to the control and regulation of upper airway muscles.

Alcohol may increase nasal airway resistance by several mechanisms. Firstly by its central depressant actions, it may diminish sympathetic tone to the sinusoidal capacitance vessels of the nose. Secondly, it may exert a peripheral vasodilator effect, leading to swelling of the nasal mucosa and increased nasal airway resistance. It may also act via other mechanisms, such as interference with catecholamine release at sympathetic nerve endings.

The possibility that alcohol may exert its effect upon the nose via several mechanisms, may help explain why we could find no correlation between blood alcohol level and increase in nasal airway resistance. Our findings in this respect were in agreement with those of Robinson et al. (1985).

They however measured nasal resistance in the sitting as opposed to the supine position, as performed in this study. Previous studies have shown that posture can exert profound effects upon nasal airway resistance (Cole and Haight, 1984).

Moving from the supine to the lateral recumbent position results in congestion of the ipsilateral and decongestion of the controlateral nostril, furthermore these changes are accentuated in conditions such as allergic rhinitis (Hasegawa and Saito, 1979). As sleep apnoea normally occurs with adoption of a supine posture, we feel that alcohol induced changes in nasal resistance in the supine posture, are more relevant to sleep apnoea.

Our findings supplement those of Issa and Sullivan, (1982), in that not only may alcohol lead to upper airway muscle hypotonia, but by increasing nasal airway resistance, alcohol will further accentuate the tendency towards upper airway collapse.

Advice regarding alcohol consumption in subjects susceptible to sleep apnoea, should therefore be clear.

ACKNOWLEDGEMENTS

We would like to acknowledge assistance in the study given by Miss Julie Lambourne from the South Glamorgan Institute of Higher Education, and also from Mr Clive Selwood and Mr Ken Tomlinson, from the Department of Biochemistry, Cardiff Royal Infirmary, for performing the blood alcohol assays.

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R. Eccles, Ph.D.
Dept. of Physiology
University College Cardiff
Wales CF1 1XL
United Kingdom