

Posture and nocturnal asthma

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ABSTRACT To investigate whether the supine posture caused sustained bronchoconstriction and could thus contribute to the development of nocturnal asthma, nine patients with nocturnal asthma were studied on two consecutive days, lying supine for four hours on one day and sitting upright for four hours on the other, the order of the two postures being randomised. Peak expiratory flow (PEF), forced expiratory volume in one second (FEV₁), and forced vital capacity (FVC) were measured immediately before and after the four hours and over the subsequent hour. There was no significant difference between the erect and supine posture for PEF (248 v 248 l/min), FEV₁ (1.31 v 1.22 l), or FVC (2.34 v 2.28 l) at the end of the four hours, nor did any significant change develop subsequently. Thus the supine posture is not associated with prolonged bronchoconstriction. As each patient had previously shown an average overnight fall in PEF of more than 20%, this study strongly suggests that the supine posture is not an important cause of overnight bronchoconstriction.

Introduction

Nocturnal wheeze is a common problem in many asthmatic patients.¹ The mechanism of nocturnal bronchoconstriction, however, remains obscure.² One theory is that the supine posture may result in substantial bronchoconstriction,³ but there is conflicting evidence about the importance of posture.³⁻⁵ We have therefore studied a group of patients with nocturnal bronchoconstriction to see whether maintaining the supine posture by day results in sustained bronchoconstriction.

Method

We studied nine asthmatic patients (four of them men) aged 19-67 years. All were taking inhaled beta₂ agonists, five oral theophyllines, seven inhaled corticosteroids, and two oral steroids (including one not having inhaled steroids). Each had recorded a mean overnight fall in peak expiratory flow of over 20% (range 21-52%) in the previous month. Each patient was stable at the time of study, having had no attacks of asthma or change in drug treatment for at least four weeks. All patients gave written informed consent.

Patients were studied at the same time of day on two consecutive days, on one of which they were supine for four hours and on the other sitting upright, the order of the postures being randomised.

Patients had their standard treatment at 7 am but no bronchodilators were allowed thereafter. They then

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attended the laboratory. Electroencephalogram, electromyogram, and electro-oculogram leads were attached by means of our standard electrode placement⁶ and recorded on a chart recorder (Neuroscribe, SLE, Croydon) running at 15 mm/s. This record was continuously monitored throughout the study and showed that no patient slept at any stage during either study. Peak expiratory flow (PEF) and forced vital capacity (FVC) measurements were made in triplicate, always in the sitting position, and the highest readings recorded. Baseline FEV₁ had to be within 0.3 l on the two days. At 1100 hours PEF and FVC measurements were recorded and blood was taken for determination of plasma theophylline concentrations. Patients then assumed the assigned posture for that day and remained in that posture for four hours. During this period they were not allowed to move or to have any bronchodilator treatment. At the end of the four hours PEF and FVC were immediately measured in the sitting position and further blood was taken for plasma theophylline assay. Measurements of PEF and FVC were repeated after a further 30 and 60 minutes while the patients remained seated.

Comparisons were made by Student's paired *t* test between the same time points on the two study days.

Results

One patient became wheezy between the four and five hour measurements on both study days and required treatment; her five hour measurements were not included in the analysis. Serum theophylline concentrations did not differ between the two days.

There were no significant differences between the mean (SD) baseline results on the two study days in

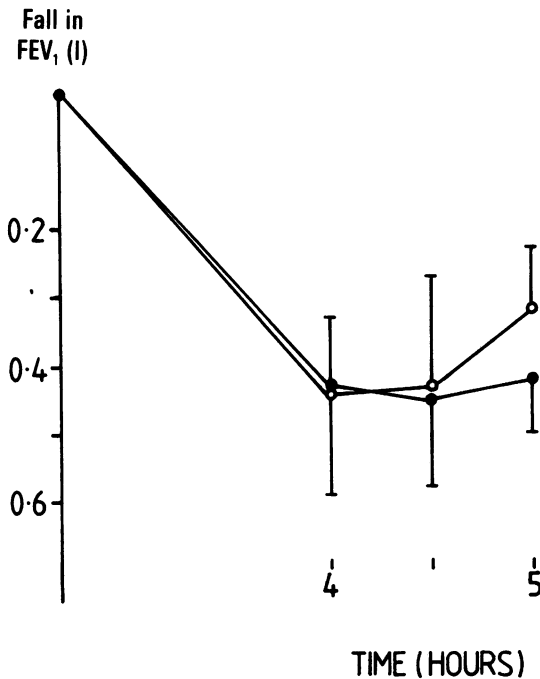


Fig 1 Decrease in FEV₁ (means with SEM) from baseline values after 4, 4.5, and 5 hours spent upright (●) and supine (○) in nine patients.

terms of PEF ("erect" day 317 (76), "supine" day 312 (60) l/min), FEV₁ (1.74 (0.6), 1.67 (0.55) l), or FVC. At the end of the four hours there was no significant difference ($p > 0.7$) between the two postures for PEF (erect 248 (28), supine 248 (30) l/min), FEV₁ (1.31 (0.16), 1.22 (0.14) l), or FVC (2.34 (0.28), 2.28 (0.37) l), nor did any significant change develop over the next hour ($p > 0.1$). The mean decrease in FEV₁ from time zero was similar ($p > 0.25$) at each time point (fig 1).

When individual data were examined, no patient showed substantially greater bronchoconstriction after lying down (fig 2) than after sitting.

Discussion

This study shows that the assumption of the supine posture for four hours is not associated with prolonged bronchoconstriction in patients who had previously shown substantial bronchoconstriction after overnight sleep. The study suggests that the supine posture is not a major cause of such overnight bronchoconstriction.

A previous study had claimed that asthmatic patients developed bronchoconstriction when lying down.³ This result may have reflected, at least in part, the mechanical disadvantage in performing forced expiratory manoeuvres in the supine posture. The

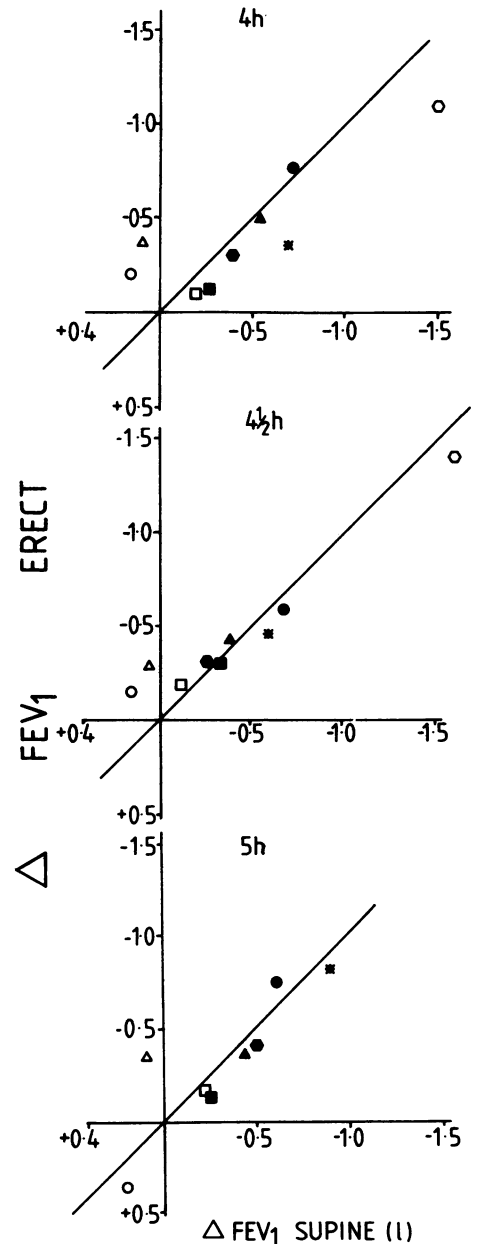


Fig 2 Comparison of FEV₁ change in the supine and erect postures between baseline values and those obtained at 4, 4.5, and 5 hours in individual patients.

conclusion drawn from that study, that the supine posture results in bronchoconstriction, is also open to statistical criticism.⁷ The current study, which avoids these problems, shows that patients who have prolonged bronchoconstriction after lying in bed over-

night do not have prolonged bronchoconstriction after lying in bed during the daytime.

Clearly, a study of nine subjects cannot exclude the supine posture as a cause of nocturnal asthma in occasional patients. The current study, however, had a 90% power to detect a 60 l/min difference in PEF after four hours in the two postures at the 5% level. As these patients developed bronchoconstriction overnight consistently with an average PEF decrement of 120 l/min, clearly other causes of nocturnal bronchoconstriction must predominate.

All our patients developed bronchoconstriction during the period of study, but this was uninfluenced by posture. It is likely to have developed because the 7 am dose of inhaled beta agonists taken by all patients had worn off. We considered it necessary to study patients with clear nocturnal bronchoconstriction and in these patients it was not possible to withhold their 7 am beta agonist dose. As the dose was the same on both study days, however, this was not a complicating factor in the analysis of the results.

Our results support those of Clark and Hetzel,⁴ who in a study that was not fully documented kept five asthmatic patients in bed for 24 hours and reported that their nocturnal bronchoconstriction persisted even though they remained supine all day. This study shows that lying down is not a major cause of nocturnal asthma but does not clarify the mechanism of nocturnal bronchoconstriction. The most likely current explanation is that this is caused by sleep synchronised⁸ circadian variations in autonomic function.⁹⁻¹¹

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