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Correspondence

Effect of sleep deprivation on overnight bronchoconstriction in nocturnal asthma

SIR,—The paper by Dr JR Catterall and others (September 1986;**41**:676–80) on the overnight fall of FEV_1 in a group of 12 asthmatics on a night awake compared with a night asleep is an interesting contribution to a challenging field of research. The authors' conclusion that sleep is an important factor in determining overnight bronchoconstriction in nocturnal asthma seems, however, not quite justified since another potentially important factor was not controlled in the study: posture itself. It is apparent from the paper that the patients were spending a part of their time lying supine even on the night when they were kept awake. Thus there is a possibility that the greater fall in FEV_1 on the "asleep night" was due to a longer time in the lying position. The authors touch on this possibility in the discussion, but immediately dismiss it by reference to a work by Clark and Hetzel[1] in which asthmatics stated to be kept in bed over the day still developed a morning dip. This work has been widely quoted as evidence against posture having any importance in nocturnal asthma, but it is unclear how strictly controlled this experiment was. We obviously need more studies with such an approach.

Jönsson and Mossberg demonstrated that the supine position per se was a powerful stimulus for bronchoconstriction in 10 patients with chronic asthma.[2] On shifting from upright to supine position there was an immediate fall in peak expiratory flow (PEF), followed by a further progressive decrease as long as the patients remained in the supine position. After four hours PEF had decreased 25% on the average compared with initial presupine values. It is easy to imagine that such a progressive deterioration of airway patency may develop to frank asthma during a night in bed. On return to the upright position there was a rapid bronchodilatation but lung function still tended to be impaired compared with presupine values.

We have recently confirmed these results in another 13 asthmatics. Moreover, in these patients posture induced bronchoconstriction was completely prevented by inhalation of an anticholinergic drug, lending some support to the suggestion by Dr Catterall and coworkers that an increased vagal tone may be important in nocturnal asthma. It is often stated that there may be several different pathways leading to nocturnal asthma. This does not seem likely to us, given that nocturnal wheezing is such a common and typical feature of the disease. On the other hand, there are likely to be several links in the chain of events leading to increased obstruction at night—we think that there are strong reasons to regard posture itself as one such link.

BJÖRN MOSSBERG
KJELL LARSSON*
Department of Pulmonary Diseases
Södersjukhuset (South Hospital)
S–100 64 Stockholm
**National Board of Occupational Safety and Health*
S–171 84 Solna

1 Clark TJH, Hetzel MR. Diurnal variation of asthma. *Br J Dis Chest* 1977;**71**:87–92.
2 Jönsson E, Mossberg B. Impairment of ventilatory function by supine posture in asthma. *Eur J Respir Dis* 1984;**65**:496–503.

*** This letter was sent to the authors, who reply below.

SIR,—Drs Mossberg and Larsson are concerned that some of the differences in overnight bronchoconstriction between the night when the patients slept and the night when they were kept awake could be due to posture. We share this concern and indeed stated on page 678 that "we cannot therefore exclude the posture differences as contributing factors in the changes in peak flow." We also clearly stated why this posture difference was necessary in our protocol as "it was not feasible for the patient to be kept lying down throughout the night they were kept awake, as we would have been unable to keep these volunteer patients fully awake as defined by the EEG." Patients left lying down for long periods, especially at night, inevitably have micro-sleeps that would have affected our results.

Drs Mossberg and Larsson question the results of Clark and Hetzel,[1] which indicated that overnight bronchoconstriction was independent of posture. Before we published our study, Dr Hetzel kindly provided us with the raw data on which his conclusions were based, as published in his MD thesis,[2] and we are happy that his patients with nocturnal bronchoconstriction continued to have bronchoconstriction at night even when they were left lying in bed for the whole 24 hour period. The study of Jönsson and Mossberg[3] is a stimulating further attempt to examine the problems of posture. It is difficult, however, to draw conclusions from their results as presented. Firstly, peak flow rate is lower when measured in the supine posture than when measured in the standing posture and thus the only valid comparisons are those made in the same posture. Secondly, so far as we can gather from the methods section, the data were analysed by multiple *t* testing with reference to the control measurement, and there is no indication that any allowance was made for this multiple testing when the significance of changes was assessed. Thirdly, the most convincing analysis would have been direct comparison of peak flow, FEV_1, or FVC measurements made at four, four and a half, and five and a half hours after four hours lying down (fig 3, ref 3) with those made at the same time after four hours in the upright posture (fig 4, ref 3). This would have allowed for any circadian changes in airway calibre which might occur over the five and a half hour period of study.

This is a complex area in which further studies are indicated and we are grateful to Drs Mossberg and Larsson for emphasising these problems and look forward to the publication of their more recent studies.

JR CATTERALL
NJ DOUGLAS
Department of Respiratory Medicine
City Hospital
Edinburgh EH10 5SB

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