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CORRESPONDENCE

Flow-volume indices in snorers with and without-OSAS

To the Editor:

In a paper on obstructive sleep apnoea recently published in the Journal, BOGAARD *et al.* [1] found correlations between maximal sleep O_2 desaturation on the one hand and peak inspiratory flow and forced inspiratory volume % predicted on the other.

In their figures 2 and 3, they illustrated this by means of linear regression lines. However, for several reasons the regressions are not linear. One reason is that flow-volume indices above a certain value will not influence the arterial oxygen tension. The other reason is the curvilinear relationship between oxygen tension and oxygen saturation. Therefore, I believe that it is misleading to draw these straight regression lines, even though it is convenient (too convenient) to have the computer perform this manoeuvre.

Reference

1. Bogaard JM, v.d. Meché FGA, Poublon RML, *et al.* Indices from flow-volume curves in relation to cephalometric, ENT and sleep O_2 saturation variables in snorers with and without obstructive sleep apnoea. *Eur Respir J* 1995; 8: 801–806.

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REPLY

From the Authors:

Dr Berglund poses two questions, a statistical one and physiological one. Firstly, concerning the statistical question, the type of regression is not dependent on underlying physiology. In my opinion, using the eye as the most simple statistical instrument, our experimental data clearly show a decrease of, *e.g.* peak inspiratory flow (PIF) with increasing nadir of nocturnal oxygen saturation.

The second step in our analysis, for which one needs a computer, is to find the model which minimizes the residual variance and gives the highest coefficient of determination (r^2). A linear model, exponential model and a power function gave correlation coefficients of 0.48, 0.50 and 0.50, respectively, whereas the Spearman Rank correlation coefficient was 0.48. From this, we concluded a linear relationship to be a valid description, other models giving no appreciable larger reduction of residual variance.

The third step concerns the physiological interpretation of the data. We do not interpret the relationship between arterial oxygen tension and flow-volume indices but have put forward a hypothesis about maximal degree of nocturnal desaturation and awake upper airway resistance as (qualitatively) represented by the inspiratory indices. We realize that the strength of inspiratory muscles also plays a role.

Crucial points in our hypothesis are the widening effect on upper airway aperture by a compensating mechanism protecting the upper airways from collapse, as described previously. Superimposed on this mechanism, which may explain the high mean PIF and forced inspiratory volume in one second (FIV_1), is a narrowing, which increases with increasing degree of nocturnal desaturation. Our hypothesis, which is based on previously described model analyses of the obstructive sleep apnoea syndrome (OSAS), we admit to be speculative.

Consequences of the pattern of the O_2-dissociation curve are discussed in the last sentence of page 804. The spread in the data is too large to separate this effect from the overall effect that we describe.

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