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CORRESPONDENCE

Flow-limitation and upper airways

To the Editor:

We read with interest in the February 1995 issue of The European Respiratory Journal the article by KOULOURIS *et al.* [1] "A simple method to detect expiratory flow-limitation during spontaneous breathing".

The authors applied negative pressure at the mouth (-5 cmH_2O) during a tidal expiration (NEP) to 22 patients with chronic obstructive lung disease. They conclude that this provides a simple, noninvasive method for the detection of expiratory, intrathoracic, flow-limitation.

Their reasoning is as follows. Application of NEP during expiration increases the pressure gradient between the alveoli and the airway opening. In non-flow-limited subjects, the expiratory flow should increase with NEP. By contrast, in flow-limited patients, application of NEP should not change the expiratory flow.

The readers would like to check the hypothesis of the authors, by comparing the flow rate before and after negative pressure is applied at the mouth. However, no mention of the flow rate, before and after NEP, can be found in the results section or elsewhere. To demonstrate the correctness of their approach, the authors present instead two figures, as "representative" examples.

The reasoning of the authors would be correct in the absence of extrathoracic upper airways. It would also be correct if upper extrathoracic airways are rigid structures, uninfluenced by transmural pressure changes; or, if these airways are bypassed (see below). Upper airways are in fact compliant structures. A negative pressure applied at the mouth induces a negative intraluminal pressure in these airways. Since outside pressure is atmospheric pressure, transmural pressure would become negative. A negative transmural pressure will narrow the upper airways, thus creating a flow-limiting segment. In other words, flow-limitation would be located at the extrathoracic, and not intrathoracic level.

KOULOURIS *et al.* [1] are probably aware of the interference of the upper airways upon their results, since they emphasize that none of their patients had a history of obstructive sleep apnoea or any evidence of upper airway obstruction. Furthermore, they state that the negative pressure that they applied, *i.e.* -5 cmH_2O, is considerably less than the negative pressure applied by SURATT *et al.* [2], who have shown that in supine subjects, "collapse" of the pharyngeal airway did not occur at negative pressures between -11 and -40 cmH_2O. This is true except that SURATT *et al.* [2] were speaking about "closure" of upper airways, and therefore zero flow and not flow-limitation. A much lower negative transmural pressure is needed to narrow than to close these airways.

In a previous study, published recently by the same authors [3], the same method was used to demonstrate flow-limitation in patients during mechanical ventilation. However, in that study, a cuffed endotracheal tube was used to intubate the patients, thus bypassing the upper airways. This was not the case in their study [1] published recently in The Journal.

We have recently shown [4] in supine, relaxed, healthy volunteers, that negative pressure at the mouth, during expiration, induces flow-limitation of the upper airways. Since these airways are unstable structures, a negative pressure of only -2 cmH_2O was enough to induce flow-limitation. When subjects contracted their upper airway muscles, "to resist" the applied pressure, there was no flow-limitation. By measuring supraglottic pressure and visualizing the oropharynx, we have demonstrated that flow-limitation was produced by narrowing of the upper airways.

References

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REPLY

From the authors:

We welcome the comments of S. Kostianev, Cl. Veriter and D. Stânescu concerning our article "A simple method to detect expiratory flow-limitation during spontaneous breathing" [1].

Our analysis essentially consists in comparing the expiratory flow-volume curve obtained during a control breath with that obtained during the subsequent expiration in which negative expiratory pressure (NEP) is applied. Subjects in whom application of NEP does not elicit an increase in flow over part or all of the control tidal volume range are considered to be flow-limited

(FL). By contrast, subjects in whom flow increases with NEP throughout the control tidal volume range are considered to be not flow-limited (NFL). In our chronic obstructive lung disease (COPD) patients, when present, flow-limitation encompassed 40–82% of the latter part of tidal expiration. In one of our "representative" examples, expiratory flow-limitation was absent as flow increased throughout the control tidal volume range, and in the other flow-limitation encompassed 82% of the control tidal volume range, *i.e.* the flow-volume curves without and with NEP were superimposed over this volume range. Thus, as clearly stated in our paper, the changes in flow with NEP are nil in FL subjects over at least part of the control tidal volume range, whereas in NFL patients flow with NEP is higher throughout expiration.

We are well aware that NEP may induce flow-limitation within the upper airways, as postulated by SANNA *et al*. [2]. Indeed, it is possible that in our NFL patients the flows achieved during application of NEP were indeed limited by such a mechanism. This, however, is irrelevant in terms of our analysis, since an increase in flow with NEP signifies that there is no flow-limitation under control conditions. In our opinion, it is unlikely that flow-limitation within the upper airways could explain the results obtained in our FL patients, because it is highly improbable that, under these conditions, the flow-volume curves with and without NEP would be superimposed. By contrast, such superimposition is predictable in the case of intrathoracic flow-limitation. In this connection, it should be noted that NEP did not cause a decrease in flow below control values in any of the COPD patients of our study.

In their elegant study, SANNA *et al*. [2] found that in relaxed supine normal volunteers who were ventilated with negative pressure applied at the mouth (and hence tidal breathing presumably occurred below functional residual capacity (FRC)) a negative pressure of only -2 cmH_2O was enough to induce extrathoracic flow-limitation when the subject were instructed to relax the upper airway muscles. Our subjects were COPD patients who were breathing spontaneously, and hence their results are not comparable with those of SANNA *et al*. [2]. Nevertheless, the main point made by these authors is in line with our current studies, namely that the NEP technique can be used not only to detect intrathoracic flow-limitation but also to elicit extrathoracic flow-limitation, especially in patients with obstructive sleep apnoeas.

References

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