Safety of Sputum Induction

To the Editor:

We have read with great interest the article by Castagnaro et al (October 1999), reporting a significant drop in oxygen saturation in the absence of an equivalent drop in FEV1 during sputum induction. This, however, has not been our experience. Although we agree that FEV1 may fall in asthmatics, COPD patients, and normal volunteers, we have not seen significant changes in oxygen saturation. We have found induced sputum to be a safe procedure in normal subjects, stable and exacerbated COPD patients, as well as in cystic fibrosis patients, using both low- and high-output nebulizers with no changes in oxygen saturation of this magnitude. Our mean fall in oxygen saturation was only 0.6%, even in COPD patients with very low FEV1.

Although, as the authors point out, there have been no studies specifically examining changes in oxygen saturation during sputum induction, one article found only a small (0.63%), although statistically significant, fall in oxygen saturation. The authors describe a recent editorial by Pavord et al, suggesting that induced sputum may be an inflammatory stimulus and that total saline solution load may be one mechanism for this. It is likely that this explains the change in inflammatory cells seen in induced sputum 24 h after sputum induction. However, it is unlikely that hypertonic saline solution could cause clinically significant bronchoconstriction via such a pathway, as this occurs in some subjects within minutes. It is more likely that hypertonic saline solution acts as a direct bronchial irritant, thus provoking bronchoconstriction.

In conclusion, we feel this article highlights important issues regarding the safety of sputum induction and the possible proinflammatory nature of hypertonic saline solution. However, the changes in oxygen saturation are hard to understand and are not consistent with our experience or other published studies.

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References


To the Editor:

We appreciate the comment by Kelly et al on our article about oxygen desaturation during sputum induction in asthmatics, smokers, and healthy subjects. These authors pointed out that our data are not in agreement with those of Bhowmik et al, who reported small changes in oxygen saturation during sputum induction in COPD patients. Our study, however, is not comparable to the Bhowmik study with regards to several points. Firstly, because in our study, hypertonic saline solution inhalation was prolonged for a maximum of 30 min (the mean duration of the overall sputum induction procedure was 24 min), whereas in the Bhowmik study, duration of overall sputum induction procedure did not exceed 14 min. Secondly, we continuously measured arterial oxygen saturation from 2 min before the start of sputum induction until the procedure was completed, and we considered all oxygen saturation data. Bhowmik et al measured only oxygen saturation at three different points: prior to starting the procedure, following 7 min of inhalation, and at the end of the procedure.

Additionally, our data are consistent with other studies, in which sputum induction and oxygen saturation monitoring were performed following the same methodology we used. In this regard, Miller et al and Leigh et al found that sputum production induced by hypertonic saline solution caused a marked oxygen desaturation in HIV patients. Also, in these studies, healthy subjects also had a significant fall in oxygen saturation during sputum induction (although less pronounced than that of HIV patients).

Lastly, our recent findings show that during sputum induction of COPD patients with mild to moderate airway obstruction, oxygen saturation falls as it does for asthmatics, smokers, and healthy subjects during the same procedure.

Based on these data, we recommend that in subjects who are hypoxemic before sputum induction, oxygen saturation monitoring should be performed during the procedure.

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References