Unheated or No Humidification Bubble for Long-Term Nasal Low-Flow Oxygen

A Matter of Nasal Mucosa Response or Disease Progression

To the Editor:

We have read with great interest the paper by Franchini et al[1] published in *CHEST* (August 2016), which reported on a randomized clinical trial comparing the effects of dry nasal low-flow oxygen (NLFO) and cold bubble humidified NLFO on nasal mucociliary clearance (MCC), mucus properties, inflammation, and symptoms in subjects with chronic hypoxemia requiring long-term domiciliary oxygen therapy. A few issues come to mind.

First, this study showed that cold bubble humidified NLFO and dry nasal NLFO had similar effects on MCC, mucus hydration, and pulmonary function. Furthermore, NLFO therapy significantly improved the symptoms of coughing and sleep disturbance without statistical differences between the humidified and dry nasal NLFO groups.

Second, in this study, neither of the NLFO treatments reached the recommended levels of temperature and humidity (100% relative humidity and 44 mg/L absolute humidity [AH]).[1] Humidified NLFO obtained higher AH (21 mg/L) compared with dry nasal NLFO (9 mg/L). The optimal conditions of AH and relative humidity help to keep stable the rheological characteristics and volume of airway secretions, increase the mucociliary clearance, and prevent the inflammatory reactions to thermal injury or fluid imbalance. The heated humidification during NLFO provides a better temperature and humidity of inspired gas, preserving the MCC and pulmonary function. The authors declared that the evaluation of the clinical impact of heated humidification during NLFO was beyond the scope of this study. This choice may be questionable. We think that the inclusion of a third group with heated humidified NLFO would have added more value to this study, since it has considerable effects on the considered end points.

Third, this study included patients with chronic obstructive disease. Mucus hypersecretion and chronic productive cough are particular features of chronic bronchitis. The primary mechanisms responsible for excessive mucus production in COPD are the overproduction and hypersecretion by goblet cells, inflammatory cell activation, and mucin gene transcription.[2] Furthermore, the progression of COPD is also associated with pathologic deterioration of mucociliary clearance and lung function.[3]

According to the Global Initiative on Obstructive Lung Disease (GOLD) classification of severity of airflow limitation in COPD, at the beginning of the study the included patients were in GOLD 3 but they worsened after 24 months reaching GOLD 4.[4] Since the chronic respiratory disease exacerbated over time in this study, it is hard to say whether the worsening of nasal mucosa functional status is due to the chronic illness progression or to the use of NLFO. We think that further prospective studies should investigate this topic.

Maria Vargas, MD
Naples, Italy
Antonio M. Esquinas, MD, PhD, FCCP
Murcia, Spain

AFFILIATIONS: From University of Naples "Federico II" (Dr Vargas) and Hospital Morales Meseguer (Dr Esquinas).

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CORRESPONDENCE TO: Maria Vargas, MD, Department of Neurosciences, Reproductive and Odonthostomatological Sciences, University of Naples "Federico II", Naples, Italy; e-mail: vargas.maria82@gmail.com

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Response

To the Editor:

We thank Drs Vargas and Esquinas for their interest in our manuscript.[1] We fully agree that a third study arm including heated humidification would have provided a worthy comparison. Indeed, we have previously published that in infants receiving chronic