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Reducing reintubation and postextubation respiratory failure: improving high-flow oxygen support performance

Laura Colinas[1], Alfonso Canabal[1,2], Gonzalo Hernández[1]

[1]Hospital Virgen de la Salud, Toledo, Spain; [2]Francisco de Vitoria University, Madrid, Spain

Correspondence to: Gonzalo Hernández, MD, PhD. Critical Care Medicine, Hospital Virgen de la Salud, Mezquite n 12, 6 A, 28045, Madrid, Spain. Email: ghernandezm@telefonica.net.

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We would like to thank Dr. Chen and his colleagues for their interest in our study. We are grateful for the insightful comments of our manuscript and we are happy to respond to them.

Extubation failure is an independent predictor of mortality. The development of therapies for preventing this has been focused on specific causes of reintubation [corticoid for larynx edema and noninvasive ventilation (NIV) for the hypercapnic respiratory failure in patients with pulmonary chronic diseases] and in patients with risk factors associated with extubation failure. Usually, extubated patients receive conventional oxygentherapy for correcting the oxygenation impairment. This system provides low-flow and doesn't guarantee the fraction of inspired oxygen (FiO_2).

As discussed in detail in your paper, the high-flow nasal cannula (HFNC) generates several physiological effects. This mode now allows constant FiO_2 during all the respiratory cycle, it confers a low level of continuous positive airway pressure proportional to administrated flow which reduces work of breathing and increased end-expiratory lung volume, partly through intrinsic positive end-expiration pressure compensation and dead-space washout. The effect of HFNC on carbon dioxide clearance is uncertain in chronic hypercapnic patients, but has been described in ear, nose and throat surgical patients under apneic conditions, suggesting that dead space washout must be accompanied by some degree of alveolar ventilation, as apneic time rises up to 35 minutes (1). In addition, HFNC provided heated and humidified oxygen, which improves comfort and possibly reduces airway inflammation and facilitates the drainage of respiratory secretions. Gas conditioning has demonstrated in experimental research a potentially protective effect on respiratory mucosa. In addition, clinical trials report an improvement in the ability to clear respiratory secretions (2,3). Regarding our methodology of the study, HFNC was applied before the extubation to block the entrance of dry and cold air in the patients' native airway from the beginning. Although this is speculative, it could play a major role in the early benefit that we found in the lower rate of upper airway obstruction (laryngeal edema requiring reintubation which wasn't described in the HFNC group).

In our study (3), we aimed to evaluate whether high-flow oxygen therapy after planned extubation would reduce the need for reintubation compared with standard oxygen therapy in patients at low risk for reintubation. The All-cause reintubation within 72 hours was lower in the high-flow group (4.9% *vs.* 12.2%). This difference was mainly attributable to a lower incidence of respiratory-related reintubation in the high-flow group (1.5% *vs.* 8.7%). The main benefit was observed on reducing reintubation secondary to hypoxemia and inability to clear secretions (3). These results agree with those obtained by Maggiore *et al.* (2).

We classified patients according to the criteria for risk of reintubation. We didn't use the type of the respiratory failure considering that we searched for the HFNC preventive function and not the treatment once that the failure was present. Today it's unclear how identify at-risk patients for extubation failure. Previous trials, like Thille *et al.*'s study (4), have tried to set up the underlying risks of extubation outcome. In keeping with these studies, we included ten risk factors. In our opinion, our strict criteria clearly select a low-risk population.

Simultaneously, we compared HFNC with NIV in

patients at high risk for reintubation in a non-inferiority trial (5). While studies have suggested that prophylactic NIV could prevent postextubation respiratory failure, they appear inconsistent with regard to reintubation. However, Thille *et al*. (6) adds new data supporting the benefit of NIV for this indication. In a general population of critical patients it hasn't been proved (7), for that reason in the low-risk group it was compared to conventional oxygen therapy.

Regarding the study protocol, patients who tolerated the spontaneous breathing trial were reconnected with the previous ventilator settings for rest (8) and clinical evaluation of the airway patency. Flow was initially set at 10 L/min and titrated upward in 5 L/min steps until patients experienced discomfort. Twelve hours after extubation a steady sate is usually obtained. In our study we observed a tolerated main gas-flow of 31 L/min, a moderate flow, suggesting that under these conditions the flow tolerated is a marker of severity. After 24 hours, high-flow therapy was stopped and, if necessary, patients received conventional oxygen therapy. In our preliminary study (9), gas-flow >35 L/min 12 hours after extubation predicted reintubation; unfortunately, this results was not reproduced in the trial. Results in the high-risk group, could elucidate the role of the tolerated flow according with the risk of failure.

Our study is similar to a pragmatic study, considering that we try to evaluate the HNFC efficacy under real and no ideal circumstances. Therefore, HFNC was applied 24 hours after extubation because 24 hours is the standard monitoring time before ICU discharge after a planned extubation. The 24-hour time limit can restrict HFNC effects to this time period (2). Nevertheless, the results confirmed that 24 hours was enough to reduce the rate of reintubation. Our 24-hour limit probably helped physicians appreciate undertreated respiratory distress at an early stage and not delay the reintubation. This could be possible because HFNC increases comfort, oxygenation and being able to disguise respiratory distress. In fact, as suggested by Kang *et al*. (10), applying HFNC in patients with respiratory failure according to clinical response could lead to delayed intubation. This could be associated with a worse outcome, as has been shown with NIV. HFNC doesn't delay the reintubation under the conditions of our protocol. We attribute this result to the preventive intention, the fixed period and to the predefined reintubation criteria. We consider that this is our study's main safety variable. Even so, we are waiting to see if this safety is confirmed in the high-risk group.

We agree with Dr. Chen and colleagues that further trials are needed to evaluate the pathophysiological mechanism of extubation failure and the HFNC physiological effects in the etiology of respiratory failure at intubation.

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Footnote

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Conflicts of Interest: Dr. Hernández reported travel expenses and personal fees from Fisher & Paykel. The other authors have no conflicts of interest to declare.

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