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Correspondence

High-frequency percussive ventilation as rescue treatment in severe hypoxemic respiratory failure in term neonates[☆]

To the Editor:

We read with interest the article by Rizkalla [1] about efficacy of high-frequency percussive ventilation (HFPV) in children with acute respiratory failure failing conventional ventilation (CV) [1]. The authors reported improvements in oxygenation index (OI), oxygen saturation (SaO₂) index, PaO₂/fraction of inspired oxygen (FiO₂), and SaO₂ as measured by pulse oximetry/FiO₂ as early as 12 hours, which continued to 48 hours after transition. Improved oxygenation occurred without an increase in mean airway pressures (MAPs). As commented by the authors, these results are consistent with those published in adults with acute respiratory distress syndrome failing CV, in whom PaO₂/FiO₂ improved after only 1 hour after switching to HFPV, at the same [2] or even lower ventilator pressures [3]. Surprisingly, the authors did not mention the use of HFPV in neonates. Even if the evidence on the use of HFPV in neonates is limited and dates back to the late 80s, these studies confirmed an improvement in oxygenation both in preterm newborns with respiratory distress syndrome in the presurfactant era and in term newborns with acute respiratory distress syndrome, failing CV [4].

Moreover, we observed similar results of Rizkalla [1] in 6 term neonates (gestational age 40.5 [±0.6] weeks, birth weight 3080 [±260] g) with severe hypoxemic respiratory failure and persistent pulmonary hypertension of the newborn (PPHN) (defined as pulmonary mean blood pressure (MBP), estimated from transtricuspidal regurgitation flow, more than 70% systemic MBP on echocardiography), who failed CV and/or high-frequency oscillatory ventilation (HFOV) and maximal medical therapy (inhaled nitric oxid (iNO) and sildenafil) and who were switched to HFPV. Oxygen saturation, PaCO₂, PaO₂, pH, peak inspiratory pressure (PIP), positive end-expiration pressure, MAP, respiratory rate, FiO₂, OI (= MAP × FiO₂/PaO₂), PaO₂/FiO₂, and MBP were recorded before and during HFPV. High-frequency percussive ventilation (delivered by a VDR-4 ventilator; Percussionaire, Sandpoint, ID) was started at a high-frequency percussive rate of 500 to 700 cycles per minute, superimposed on a convectional rate of 20 to 30 breaths per minute, a PIP and MAP matching those used during CV/HFOV, positive end-expiration pressure 6 to 8 cm H₂O, and an inspiratory/expiratory ratio of 1:1. A Wilcoxon test for paired data was used for comparisons. Data are reported as median (interquartile range). Analyses were performed using MedCalc software rel. 9.3.9.0 (MariaKerke, Belgium).

As Rizkalla [1], we observed a marked oxygenation improvement after rescue by HFPV: OI significantly decreased (mean difference: -19.3 ± 9.1), and preductal SaO₂ as measured by pulse oximetry/FiO₂ significantly increased in the 2 hours after the switch to HFPV (Fig. 1), and these improvements were sustained during the next 48 hours. Of note, the decrease in OI was even more pronounced than the one reported by the authors. In agreement with Rizkalla [1], improved oxygenation occurred without an increase in airway pressures (MAP: 17 [12–19] cm H₂O before switch vs 14 [12–19] cm H₂O 2 hours after switch to HFPV, $P = .8$; PIP: 40 [35–41] cm H₂O before switch vs 40 [36–44] cm H₂O 2 hours after switch to HFPV, $P = 1$) and without any change in hemodynamic parameters. This latter result is particularly important in the context of PPHN that might be associated with a severe hemodynamic impairment. Differently from the authors, we did not observe any change in Pco₂ values (Paco₂ 43.0 [39.6–47.6] mm Hg before switch vs 41.4 [35.8–45.9] mm Hg 2 hours after switch to HFPV), probably because our patients were hypoxemic but not hypercapnic before switching to HFPV. Eventually, one infant died, whereas the other infants were discharged in good conditions after 14 (±4) days of mechanical ventilation and 25 (±10) days of supplemental oxygen. Our results strengthen the effectiveness of HFPV in improving oxygenation in a lung protective manner. Moreover, as the use of HFPV in the specific subset of hypoxemic respiratory failure associated with PPHN has never been reported to date, our experience highlights the potential role of this ventilation also in the subset of neonatal hypoxemic respiratory failure unresponsive to CV/HFOV and medications, particularly where extracorporeal membrane oxygenation (ECMO) is not available.

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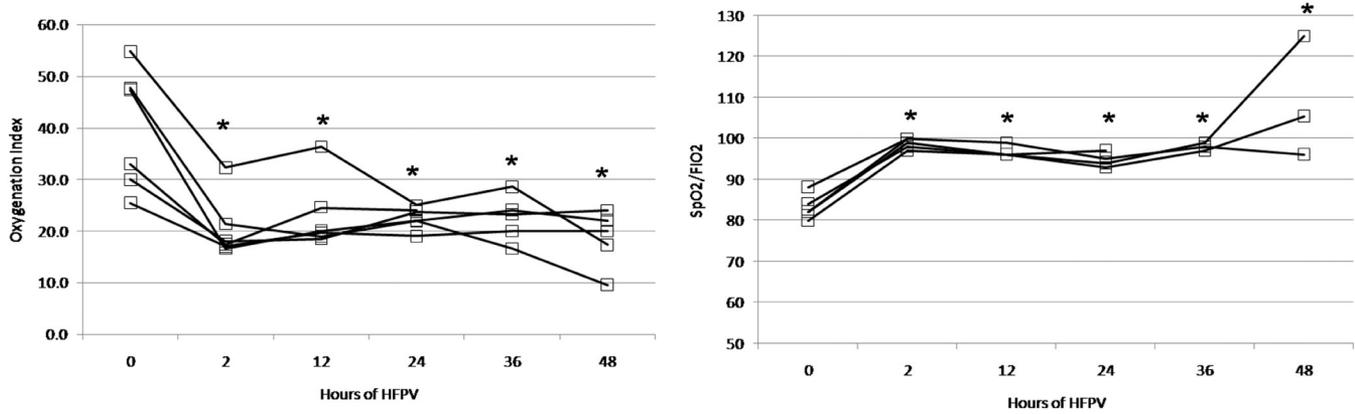


Fig. 1. Changes in OI and preductal SaO₂ as measured by pulse oximetry/FiO₂ in 6 neonates with hypoxemic respiratory failure failing CV or high-frequency mechanical ventilation and medical therapy after switch to HFPV. Individual patient data are reported. **P* < .05, refers to comparison of variables at 2, 12, 24, and 48 hours of HFPV to preswitch (0 hours) values (Wilcoxon test for paired data).

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