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High-frequency percussive ventilation improves oxygenation and ventilation in pediatric patients with acute respiratory failure☆

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ABSTRACT

Purpose: High-frequency percussive ventilation (HFPV) in pediatrics has been described predominantly in burned patients. We aimed to describe its effectiveness and safety in noninhalational pediatric acute respiratory failure (ARF).

Methods: We conducted an observational study in a tertiary care pediatric intensive care unit on 31 patients with ARF failing conventional ventilation transitioned to HFPV. Demographics, ventilator settings, oxygenation index, oxygen saturation index, oxygen saturation as measured by pulse oximetry/fraction of inspired oxygen (FIO_2), and PaO_2/FIO_2 were recorded before and during HFPV.

Results: Initiation of HFPV was associated with improvements in oxygenation index, oxygen saturation index, PaO_2/FIO_2, and oxygen saturation as measured by pulse oximetry/FIO_2 as early as 12 hours ($P < .05$), which continued through 48 hours after transition. Improved oxygenation occurred without an increase in mean airway pressures. Reductions in $PaCO_2$ occurred 6 hours after initiation of HFPV and continued through 48 hours ($P < .01$). Improved gas exchange was accompanied by reduced peak-inflating pressures at all time intervals after initiation of HPFV ($P < .01$). Vasopressor scores were similar before and after initiation of HFPV in patients requiring vasoactive support. Twenty-six (83.9%) of 31 patients survived to hospital discharge.

Conclusions: In a heterogeneous population of pediatric ARF failing conventional ventilation, HFPV efficiently improves gas exchange in a lung-protective manner.

1. Introduction

Acute respiratory failure (ARF) is a leading cause of morbidity and mortality in pediatric intensive care units (PICUs), and mechanical ventilation remains the mainstay of therapy. Ventilator-induced lung injury has been a well-documented consequence of mechanical ventilation [1,2], prompting use of lung-protective strategies and the development of alternative nonconventional modes of ventilation [1-5].

High-frequency percussive ventilation (HFPV) is a unique mode that attempts to combine the beneficial effects of conventional and high-frequency ventilation [6]. It stacks successive subtidal volume breaths at a rapid rate superimposed upon conventional cyclic rates, allowing for progressive stepwise inflation of the lung to a set peak pressure, and a passive exhalation to a predetermined lower pressure. Continuous pneumatic compressions also allow for a mobilization of retained airway secretions [7].

High-frequency percussive ventilation was initially described in burned patients with inhalational injury, where it efficiently mobilized retained soot compared with conventional ventilation [8-10]. More recently, HFPV has been described in adult patients without burn injury but with ARF, primarily as a rescue mode for patients unable to meet oxygenation and ventilation goals with conventional ventilation [11-14]. High-frequency percussive ventilation is consistently reported to improve oxygenation at lower pressures than those used for conventional ventilation, despite a lack of reduction of mortality or ventilator days [11,14,15].

In pediatric burned patients [9,10,16-18], retrospective studies have also suggested lower inflation pressures and improved oxygenation. A prospective trial comparing conventional ventilation with HFPV in burned children demonstrated lower inflation pressures and marginally improved oxygenation with HFPV, but showed no significant outcome differences [16]. The single published report of HFPV use in nonburn pediatric ARF was as a salvage mode in an infant with hydrocarbon aspiration [19]. Despite use of this mode of ventilation for more than 3 decades, the use and efficacy of HFPV as

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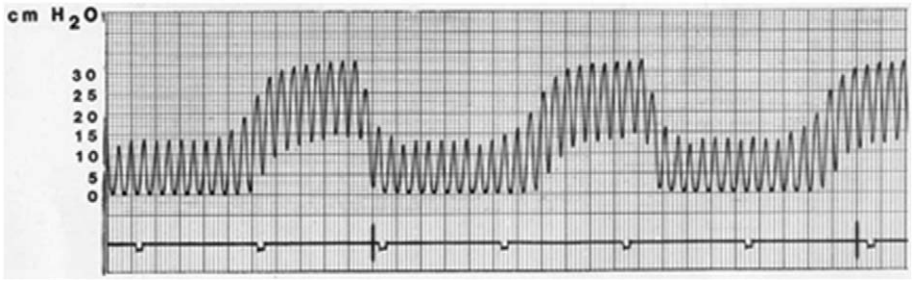

Fig. 1. Time-pressure tracing of the HFPV ventilatory cycle on the VDR-4. The ventilator delivers pneumatically driven, subtidal volume breaths at a set percussive rate (shown as 500 breaths/min) successively to a high pressure (peak inspiratory pressure or, alternatively, the pulsatile flow rate) for a predetermined inspiratory time. Exhalation to a preset low pressure (end-expiratory pressure analogous to positive end-expiratory pressure) is passive and kept there for a preset expiratory time. Reproduced with permission from Percussionaire.

a primary ventilator strategy or rescue mode in nonburn pediatric respiratory failure is unknown.

In this study, we describe our initial experiences with HFPV in pediatric patients with ARF. We aimed to evaluate the changes in respiratory and hemodynamic function in patients with ARF in whom HFPV was initiated after failure of conventional ventilation. We hypothesized that there would be a significant and sustained improvement in oxygenation and a reduction in peak-inflating pressures after transition from conventional ventilation to HFPV.

2. Methods

2.1. Patient selection and design

We conducted a retrospective observational study in patients receiving HFPV for failure of conventional ventilation at the Children's Hospital of Philadelphia, a 55-bed, tertiary care PICU. All patients were identified from a database of HFPV use. The study was approved by the hospital institutional review board, and the requirement for informed consent was waived. All consecutive patients receiving HFPV between October 1, 2010, and January 31, 2012, were eligible for inclusion, which totaled 40 patients. Patients were excluded if HFPV was initiated for reasons other than failure of conventional ventilation, which removed 9 patients from our consecutive cohort: in 4 patients, HFPV was empirically initiated for smoke inhalation; in another 4 patients, HFPV was used during extracorporeal membrane oxygenation; and in 1 patient, a diagnosis of unrepaired cardiac disease prompted transfer out of our PICU. This left 31 patients available for analysis.

2.2. Conventional ventilation strategy

Determination of failure of conventional ventilation and decision to use alternate modes were left to the discretion of the attending physician. Despite the lack of a formal protocol, our institutional practice for respiratory failure is to initiate conventional ventilation with a minimum of 5 cm H_2O of end-expiratory pressure and 6 to 8 mL/kg of tidal volume and to attempt to wean fraction of inspired oxygen (F_{IO_2}) to 0.60 or less. Inability to wean F_{IO_2} prompts escalation of end-expiratory pressures and subsequent repeat efforts to wean F_{IO_2}, with the goal to maintain peak inspiratory pressures of 32 cm H_2O or less. Persistently elevated peak pressures ($\geq$32 cm H_2O), ongoing hypercarbia ($P_{aCO_2} \geq 80$ or pH < 7.30), or oxygenation difficulties (inability to wean $F_{IO_2} \leq 0.60$ despite increasing end-expiratory pressure) prompt reevaluation of the ventilatory strategy and a change in the mode of ventilation. All patients were ventilated with a decelerating flow waveform on conventional ventilation, justifying our use of peak-inflating pressures as a risk factor for alveolar distension.

2.3. High-frequency percussive ventilation strategy

Our institution uses the VDR-4 (Pecussionaire, Sandpoint, Idaho). Typical HFPV starting settings used were a high-frequency percussive rate of 500 to 600 breaths/min (lower rates for hypercarbia)

Table 1
Characteristics of patient population and HFPV use

Variable[a]	(n = 31)
Age (y)	1.6 (0.6, 6.8)
Weight (kg)	10.0 (7.1, 25.2)
Sex (male), n (%)	15 (48.4)
Race, n (%)	
Asian/Pacific Islander	1 (3.2)
Black/African American	10 (32.2)
Hispanic	3 (9.7)
White	17 (54.8)
PRISM III at 12 h	6.5 (1, 11)
Immunocompromised, n (%)	6 (19.4)
Length of mechanical ventilation before transition to HFPV (d)	1.0 (0.0, 4.0)
Vasopressors, n (%)	18 (58.1)
Vasopressor score[b] before transition to HFPV (n = 18)	5.0 (2.0, 12.0)
Vasopressor score after transition to HFPV (n = 18)	7.5 (5.0, 14.0)
Ancillary therapy used before HFPV[c], n (%)	
Neuromuscular blockade	16 (51.6)
Inhaled nitric oxide	9 (29.0)
HFOV	3 (9.7)
Corticosteroids	5 (16.1)
Prone positioning	2 (6.5)
Exogenous surfactant	1 (3.2)
Severity of oxygenation impairment, n (%)	
PF ratio < 200 (n = 16[d])	15 (93.8)
SF ratio < 264[e] (n = 31)	25 (80.6)
SF ratio < 221[e] (n = 31)	19 (61.3)
Barotrauma, n (%)	
Before transition to HFPV	3 (9.7)
After transition to HFPV	4 (12.1)
Reason for stopping HFPV, n (%)	
Significant improvement in respiratory failure	23 (74.2)
Death or withdrawal of life support	5 (16.1)
Inadequate improvement, dyssynchrony, or poor tolerance	3 (9.7)
Total HFPV days	4.0 (2.3, 6.0)
Total ventilator days	16.0 (10.0, 22.8)
Total PICU length of stay (d)	22.0 (17.0, 34.8)
Mortality, n (%)	5 (16.1)

[a] Continuous data are in the form of median (25th, 75th percentiles), and categorical data are in the form of n (%).

[b] Vasopressor score = dopamine dose ($\mu g\,kg^{-1}\,min^{-1}$) × 1 + dobutamine ($\mu g\,kg^{-1}\,min^{-1}$) × 1 + epinephrine ($\mu g\,kg^{-1}\,min^{-1}$) × 100 + norepinephrine ($\mu g\,kg^{-1}\,min^{-1}$) × 100 + phenylephrine ($\mu g\,kg^{-1}\,min^{-1}$) × 100 + milrinone ($\mu g\,kg^{-1}\,min^{-1}$) × 10. Vasopressor score medians (interquartile range) reflect only the 18 patients ever exposed to these medications.

[c] More than 1 category was possible.

[d] Arterial blood gas data available for 16 patients.

[e] Cutoff values for mild and moderate/severe ARDS using noninvasive, S_{PO_2}-based measures of oxygenation impairment.