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Questions? Contact the Managing Editor at editor@csrt.com.
Respiratory compromise needs a useful definition

Justin Sorge RRT FCSRT MPH

The new buzz phrase making the rounds in the respiratory therapy community is “respiratory compromise” (RC). This push to promote RC awareness seems to stem from a conference organized by the National Association for the Medical Direction of Respiratory Care (NAMDRC). There is now a Respiratory Compromise Institute, and the Canadian Society of Respiratory Therapists is on board, offering an online toolkit for practitioners to access a wealth of information. There is no dispute that there exists a period of time preceding respiratory failure in which timely intervention may prevent disease progression. However, this time period is difficult to quantify and, unfortunately, the definition of RC initially put forth by its architects is far too wide and vague to be of any clinical or research value. This presents a unique opportunity for interested clinicians and researchers to develop a pragmatic case definition that is evidence based.

In a monograph outlining the approaches to the different types of RC, Miller and colleagues [1] define RC as “a deterioration in respiratory function in which there is a high likelihood of decompensation into respiratory failure or death but for which timely specific interventions (enhanced monitoring or therapies) might prevent or mitigate decompensation.” Into this definition I could fit both birth and aspirating on dinner as equal states of RC.

From a clinical standpoint this definition does not provide adequate criteria for which practitioners may assess whether a patient is experiencing or at risk of developing RC. The authors of the monograph list a number of risk factors for the development of RC; however, the studies cited do not use RC as an endpoint. Indeed, throughout the paper the authors seem to conflate respiratory failure with RC, or they use mortality or intubation as evidence to support the conclusion that a certain risk factor is a predictor of RC, complicating our understanding. Table 1 of the monograph [1] provides early signs and monitoring options for the differing types of RC. The early warning signs are nonspecific and offer no objective indices for identification of RC. Similarly, the monitoring options do not provide objective clinical thresholds to stimulate intervention. As it stands, the recommendations presented do not offer anything new or novel from which practitioners will be able to identify, treat, or adjust patient plans in the context of RC [1].

The current definition is of little value in research as well. Researchers will have a difficult time dividing study subjects into case and control groups or establishing study endpoints based on the current definition. A brief search of the Medline database using the search term “respiratory compromise” (indicating the search term must be present in the title of the paper) and limiting results to years since the seminal NAMDRC conference identified that researchers are writing their own case definitions of RC, for research purposes. And, not unexpectedly, they differ.

Without a clear and standardized set of criteria to classify whether or not a person is experiencing RC, researchers have defined their own and these differences across studies make findings noncomparable. As an extension, without a useful case definition—one with which we are able to definitively classify or exclude occurrences—disease surveillance, the ongoing and continuous collection and analysis of health information useful for the purposes of planning and evaluating public health interventions, is not possible.

This brings up some important questions: Why the current RC campaign? Why now? Where is the study showing an increase in RC detection that stimulated the NAMDRC conference? If the campaign is evidence based, where is the evidence? The formulation of the RC campaign may very well have been instigated by clinicians drawing from their experiences and observations—again, there is no dispute that such a period exists—however, moving forward I encourage those interested in redefining RC do so in consultation of the literature.

The monograph [1] states that its authors hope to stimulate further research in the area of RC. Here is my wish list for researchers heeding this call: create an evidence-based, comprehensive, and objective case definition. The goal here should be to provide clinicians and researchers with a mutually exclusive, collectively exhaustive set of criteria to determine cases and noncases. As noted, there is a dearth of research on RC, specifically with cited reports focusing on events suggested to be a result of RC, so there will need to be an element of creativity. I would suggest reviewing the literature for significant and measurable predictors of measurable outcomes such as respiratory failure, mortality, intubation, etc. This may paint a clinical picture of the window preceding these outcome events, which I “think” is RC.

REFERENCE


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The use of high-frequency percussive ventilation after cardiac surgery significantly improves gas exchange without impairment of hemodynamics

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Objective: Respiratory failure represents a significant source of morbidity and mortality for surgical patients. High-frequency percussive ventilation (HFPV) is emerging as a potentially effective rescue therapy in patients failing conventional mechanical ventilation (CMV). Use of HFPV is often limited by concerns for potential effects on hemodynamics, which is particularly tenuous in patients immediately after cardiac surgery. In this manuscript we evaluated the effects of HFPV on gas exchange and cardiac hemodynamics in the immediate postoperative period after cardiac surgery, in comparison with CMV.

Methods: Twenty-four consecutive cardiac surgery patients were ventilated in immediate postoperative period with HFPV for two to four hours, then they switched to a CMV using the adaptive support ventilation mode for weaning. Arterial blood gases were performed during the first and second hour on HFPV, and at 45 minutes after initiation of CMV. Respiratory settings and invasive hemodynamic data (mixed venous oxygen saturation, central venous pressure, systemic and pulmonary blood pressure, cardiac output and index) were collected utilizing right heart pulmonary catheter and arterial lines during HFPV and CMV. Primary outcome was improvement in the ratio between partial pressure of oxygen to fraction of inspired oxygen (P/F ratio) and changes in hemodynamics.

Results: Analysis of data for 24 patients revealed a significantly better P/F ratio during the first and second hour on HFPV, compared with a P/F ratio on CMV (420.0 ± 158.8, 459.2 ± 138.5, and 260.2 ± 98.5 respectively, \(p < 0.05\), suggesting much better gas exchange on HFPV than on CMV. Hemodynamics were not affected by the mode of the ventilation.

Conclusions: Improvement in gas exchange, reflected in a significantly improved P/F ratio, wasn’t accompanied by worsening in hemodynamic parameters. The significant gains in the P/F ratio were lost when patients were switched to conventional ventilation. This data suggest that HFPV provides significantly better gas exchange compared with CMV and can be safely utilized in postoperative cardiac patients without any significant effect on hemodynamics.

Key Words: high-frequency percussive ventilation; cardiac surgery; gas exchange; hemodynamics

INTRODUCTION

Postoperative respiratory failure is a significant source of morbidity and mortality in postoperative patients. Postoperative pulmonary alterations after cardiac surgery include increased minute ventilation, breathing frequency, \(\text{CO}_2\) production and oxygen consumption, and decreased tidal volume [1]. Postoperative pulmonary complications in open-heart surgery also have an effect on gas exchange making it difficult to manage postoperative patients while on the mechanical ventilator and after extubation. Intrapulmonary shunting has been shown to be a large component of impaired gas exchange before, during, and after cardiac surgery [2]. Recruitment of lung tissue with increased lung volume may be beneficial in reducing intrapulmonary shunting and the resultant hypoxemia [3]. The methods employed for lung recruitment are widely varied, and there is considerable variance among methods in clinicians treating this patient population. Some of the recruitment maneuvers include high positive end expiratory pressure (PEEP), prone positioning, inhaled
The use of high-frequency percussive ventilation

vasodilators, high-frequency oscillatory ventilation, and high-frequency percussive ventilation (HFPV) [4]. Although, these maneuvers can be effective, they are not without significant practical difficulties when utilized in postoperative cardiac surgery patients. The often-fragile hemodynamic status of postoperative cardiac surgery patients requiring vasopressors and inotropes would benefit from methods that maximize lung recruitment and improve gas exchange without hemodynamic embarrassment. In this manuscript we compare gas exchange with hemodynamic performance of conventional mechanical ventilation (CMV) and HFPV.

HFPV was delivered via volumetric diffusive respirator (VDR-4; Percussionaire Corporation, Sandpoint Idaho USA). The VDR is classified as a pneumatically driven, time-cycled, high-frequency flow interrupter, intermittent mandatory ventilation [5]. It uses a sliding venturi to inject sub-tidal volumes at high frequencies. Settings include pulsatile flow rate, oscillatory continuous positive airway pressure (CPAP), PEEP, inspiratory time, expiratory time, pulse frequency, and pulse inspiratory and expiratory (I:E) ratio. The sliding venturi precisely stacks the injected sub tidal volumes at the high-frequency rate set, for the inspiratory time selected. The inspiratory time is time cycled off, allowing a drop to oscillatory CPAP/PEEP for the selected expiratory time. High-frequency rates may be varied from 80 to 1000 cycles per minute with the VDR-4.

HFPV has been used to improve gas exchange in patients with adult respiratory distress syndrome (ARDS) failing conventional ventilation, while increasing mean airway pressure and decreasing peak airway pressure without affecting hemodynamics [6, 7]. Previous studies have shown HFPV to be effective in reducing intracranial pressure in traumatic brain injury [8, 9], treating lung injury associated with burns and inhalational injury [10–13], reducing time on extra-corporeal membrane oxygenation [14, 15], recruiting lung volume and improving hypoxemia in pulmonary resection [16], and improving oxygenation and providing lung protective ventilation in pediatric acute respiratory failure [17]. HFPV was also used as salvage therapy in patients after cardiac surgery [18] and in morbidly obese patients [19]. To date, there have been no studies demonstrating improvement in oxygenation in the immediate postoperative period in cardiac surgery patients and its effects on hemodynamics.

METHODS
This study was approved by the New York Methodist Hospital Institutional Review Board for scientific and ethical merit. Twenty-four consecutive cardiac surgery patients undergoing elective cardiac surgery were recruited from February 2012 to April 2012. After arrival from the operating room the patients were transferred from the conventional ventilator used in the operating room to HFPV mode. HFPV mode was utilized for ventilation until initial hemodynamic and respiratory stability was achieved, followed by transitioning to CMV in two to four hours. Hemodynamic and respiratory stability was defined as stable vital signs, pressor, and inotrope requirements and stable acid base status of the patient.

Basic initial VDR settings include high-frequency rate of 500–600 percussions per minute, convective rate of 12 to 16 breaths per minute, oscillatory CPAP of 5 cm H₂O, the lowest pulsatile flow rate leading to the rise of the chest, fraction of inspired oxygen (FiO₂) of 70%, and I:E ratio of 1:1 on both convective and percussive rate (Figure 1). Humidification was provided by humidifier (MR 850, Fisher & Paykel, Auckland, New Zealand) set up at 37° C. Arterial blood gases (ABGs) were recorded at 15–30 minutes during the first and second hour HFPV, and 45 minutes after transition to CMV from HFPV. When hemodynamic and respiratory stability was achieved after two to four hours of HFPV, the patients were transferred to CMV for weaning and extubation (Hamilton G5, Hamilton Medical AG, Bonaduz, Switzerland). The mode used on the Hamilton G5 for weaning and extubation was adaptive support ventilation (ASV), which is classified as pressure-controlled intermittent mandatory ventilation.

ASV is a closed-loop mode of ventilation that provides ventilatory support based on the patient’s work of breathing. ASV uses Otis’ equation to predict a tidal volume and respiratory rate that minimizes the patient’s work of breathing [20]. The operator enters the patient height, gender, and percent minute volume (%MV), PEEP, FiO₂,
expiratory trigger sensitivity, trigger, ramp speed, and ASV pressure limit. The Hamilton G5 in ASV mode continuously monitors the work of breathing relative to the selected %MV, adjusting peak pressure, tidal volume, and breathing frequency accordingly. The range of %MV used in ASV mode was 130–160%, with FiO₂ of 0.70, and PEEP of 5 cm H₂O.

Ventilatory and hemodynamic parameters and values recorded were FiO₂, mean airway pressure (MAP), peak inspiratory pressures (PIP), respiratory rate, PEEP, tidal volume, I/E ratio, partial pressure of oxygen (PaO₂) to FiO₂ (P/F ratio), mixed venous oxygen saturation, central venous pressure, systemic and pulmonary blood pressure, cardiac output, and index.

ANALYSIS

Statistical analysis was performed using SPSS statistical package software. Continuous data were expressed as a mean ± standard deviation. A paired t-test was used in a side-by-side comparison of the mean of the first and second hour P/F ratios on HFPV with the corresponding values on ASV. Assessment of the effect of two ventilatory modes on hemodynamic parameters was made using an ANOVA test, to compare the first and second hours on HFPV to the first hour on ASV. All statistical tests were two sided and alpha was set at p < 0.05.

RESULTS

The mean age for these open-heart surgery patients was 61.2 ± 14.7 years (range 29–80 years). There were 12 males (50%), and 12 females (50%) included in this study analysis (Table 1). The mean P/F ratio was significantly higher when utilizing HFPV during the first and second hour as compared with ASV (420.0 ± 158.8, 459.2 ± 138.5, and 260.2 ± 98.5, p < 0.05, respectively). The mean MAP in HFPV group was similar to MAP in CMV group (11.0 ± 1.8 cm H₂O vs. 10.4 ± 2.1 cm H₂O). Additionally, there were no statistical differences in hemodynamic parameters observed between both modes of ventilation (Table 2).

### TABLE 1

Demographic and perioperative data of the cohort

<table>
<thead>
<tr>
<th>Procedure (n)</th>
<th>Male, n (%)</th>
<th>Age, years (range)</th>
<th>Procedure (n)</th>
<th>Valve (AVR, MV procedure)</th>
<th>Valve/CABG</th>
<th>Other (atrial myxoma, ASD repair)</th>
<th>FEV1 (%)</th>
<th>Preoperative LVEF (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CABG</td>
<td>12 (50%)</td>
<td>61.2 ± 14.7 (30–80)</td>
<td>Valve (AVR, MV procedure)</td>
<td>7</td>
<td>2</td>
<td>2</td>
<td>83 ± 16.6</td>
<td>49 ± 14</td>
</tr>
</tbody>
</table>

**CABG,** coronary artery bypass grafting; **AVR,** aortic valve replacement; **MV,** mitral valve; **ASD,** atrial septal defect; **FEV1,** forced expiratory volume at 1 second; **LVEF,** left ventricular ejection fraction. Continuous data are expressed as a mean ± standard deviation.

### TABLE 2

Cardiopulmonary data (mean ± standard deviation) during high-frequency percussive ventilation (HFPV) and conventional mechanical ventilation (CMV)

<table>
<thead>
<tr>
<th></th>
<th>First hour HFPV</th>
<th>Second hour HFPV</th>
<th>CMV</th>
</tr>
</thead>
<tbody>
<tr>
<td>FiO₂, %</td>
<td>69.6 ± 0.02</td>
<td>68.1 ± 0.04</td>
<td>68.3 ± 0.04</td>
</tr>
<tr>
<td>Mean airway pressure, cm H₂O</td>
<td>11.0 ± 1.8</td>
<td>10.8 ± 1.9</td>
<td>10.4 ± 2.1</td>
</tr>
<tr>
<td>pH</td>
<td>7.4 ± 0.1</td>
<td>7.4 ± 0.09</td>
<td>7.4 ± 0.07</td>
</tr>
<tr>
<td>PaCO₂ mm Hg</td>
<td>34.6 ± 8.2</td>
<td>34.1 ± 8.8</td>
<td>36.8 ± 6.4</td>
</tr>
<tr>
<td>PaO₂ mm Hg</td>
<td>293.3 ± 112.3*</td>
<td>313.2 ± 95.9*</td>
<td>178.5 ± 70.4*</td>
</tr>
<tr>
<td>P/F ratio</td>
<td>420.0 ± 158.8*</td>
<td>459.2 ± 138.5*</td>
<td>260.2 ± 98.5*</td>
</tr>
<tr>
<td>Cardiac output, L/min</td>
<td>5.1 ± 1.1</td>
<td>5.2 ± 1.0</td>
<td></td>
</tr>
<tr>
<td>Cardiac index, L/min/m²</td>
<td>2.6 ± 6.6</td>
<td>2.6 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>Mixed venous PaO₂, %</td>
<td>73.5 ± 6.2</td>
<td>73.9 ± 6.0</td>
<td></td>
</tr>
<tr>
<td>PAS, mm Hg</td>
<td>31.9 ± 7.1</td>
<td>31.6 ± 6.5</td>
<td></td>
</tr>
<tr>
<td>PAd, mm Hg</td>
<td>16.9 ± 4.7</td>
<td>16.7 ± 3.9</td>
<td></td>
</tr>
<tr>
<td>CVP, mm Hg</td>
<td>11.9 ± 3.7</td>
<td>12.1 ± 2.8</td>
<td></td>
</tr>
<tr>
<td>Mean Arterial Pressure, mm Hg</td>
<td>83.5 ± 10.8</td>
<td>84.5 ± 10.3</td>
<td></td>
</tr>
</tbody>
</table>

Data are expressed as a mean ± standard deviation. FiO₂, fraction of inspired oxygen; PaCO₂, arterial carbon dioxide tension; PaO₂, arterial oxygen tension; P/F ratio, ratio between PaO₂ and FiO₂; PAS, systolic pulmonary arterial pressure; PAd, diastolic pulmonary arterial pressure; CVP, central venous pressure.

*P < 0.05.

**DISCUSSION**

This study is the first descriptive analysis of the applicability of HFPV in comparison with CMV in cardiac surgery patients with close monitoring of their hemodynamics. The mean P/F ratio was significantly higher when utilizing HFPV during the first and second hour as compared with ASV (420.0 ± 158.8, 459.2 ± 138.5 and 260.2 ± 98.5, p < 0.05, respectively). The mechanism by which HFPV carries out gas exchange is suggested by various theories, including direct bulk flow, longitudinal dispersion of gas molecules at terminal airways and alveoli, Pendelluft air flow between neighboring lung regions thereby increasing dead space ventilation, laminar flow, cardiogenic mixing, and molecular diffusion.

With the use of HFPV, we noticed significant improvement in the P/F ratio without any effect on hemodynamics. HFPV was shown to improve gas exchange at lower PIP, yet higher MAP [11, 12, 17]. Higher MAP theoretically can compromise cardiac function, especially in the setting right after cardiac surgery, therefore raising concerns for possible worsening of hemodynamics with HFPV. Several previous reports didn’t detect any major effects of HFPV on hemodynamics in burn and adult respiratory distress syndrome patients [23, 24]. Reper and his colleagues [23] compared hemodynamics, blood oxygenation, and ventilatory parameters in eight stable ICU burn patients. Hemodynamic data were not significantly affected, PIPs were significantly lower with HFPV, but MAPs were unchanged. Oxygenation and CO₂ removal were significantly better. Gallagher and his colleagues [24] compared HFPV with CMV. PaO₂ on HFPV improved significantly (p < 0.01) compared with PaO₂ on CMV at the same level of FiO₂. Cardiac output was unaffected by the change to HFPV.

This study is unique in that it directly compares oxygenation in two vastly different ventilation modes after cardiac surgery within the same patient. Henceforth, HFPV has only been used and studied as a salvage therapy for patients failing CMV. In our study, the striking difference in the P/F ratio in the first and second hour on HFPV, compared with conventional ventilation, at a comparable MAP (Table 2) may represent a more effective method of augmenting lung recruitment and improving ventilation/perfusion (V/Q) matching. The potential benefits of HFPV in postoperative open-heart cardiac surgery patients are promising. Further studies comparing HFPV to conventional ventilation in the immediately postoperative cardiac surgery patient are therefore warranted.
The use of high-frequency percussive ventilation

In this population of postoperative open-heart cardiac surgery patients, HFPV was able to significantly improve gas exchange as reflected by a better P/F ratio without hemodynamic consequence.

CONCLUSIONS

REFERENCES

DISCLOSURE

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CASE STUDY

Esophageal pressure balloon and transpulmonary pressure monitoring in airway pressure release ventilation: a different approach

Ehab G. Daoud, MD, FACP, FCCP1,2,3, Kimiyo H. Yamasaki, RRT4, Keith Nakamoto, RRT4, Denise Wheatley, RRT4


This is a case of Acute Respiratory Distress Syndrome managed using esophageal balloon catheter to adjust inspiratory pressure and positive end expiratory pressure according to the inspiratory and expiratory transpulmonary pressures. There are no studies that examine the transpulmonary pressures in airway pressure release ventilation (APRV). We aimed to test the feasibility of using the esophageal balloon in the nonconventional mode of APRV. All pressures were observed when switching the mode from a pressure-controlled mode to APRV using the same inspiratory pressure and using various incremental release times (T_{low}) to calculate the transpulmonary pressure. At all T_{low} levels the transpulmonary pressure at end exhalation was in the negative value indicating alveolar collapse. A larger study is needed to confirm our findings and to help guide setting APRV.

Key Words: esophageal balloon; transpulmonary pressure; APRV; PEEP

INTRODUCTION

The use of esophageal pressure monitoring as a surrogate for pleural pressure and, hence, the transpulmonary pressure (P_{A}-P_{E}), which is the distending pressure of the lungs, was described in the mid-20th century. This technology has many benefits in different clinical situations during mechanical ventilation, for example to assess patient’s effort when the respiratory muscles are active, to monitor the patient-ventilator interactions, and to facilitate the weaning process from mechanical ventilation [1, 2]. However, its use has been mostly limited to clinical research [1]. The recent international Safe Lung study [3] revealed that such technology is rarely used in Acute Respiratory Distress Syndrome (ARDS) patients. Increased interest in using such technology arose after a 2008 study [4] used esophageal pressure monitoring to set inspiratory pressures and positive end expiratory pressure (PEEP) in ARDS using the transpulmonary pressure (P_{A}-P_{E}). According to the study protocol, the end inspiratory P_{E} was kept under 25 cmH_{2}O and the end expiratory P_{E} between 0 and 10 cmH_{2}O. Using such a strategy resulted in improved oxygenation, compliance, and trend towards improved mortality. Our understanding of ventilator-induced lung injury (VILI) has markedly advanced over the last decade [5]. The use of the esophageal balloon monitoring during mechanical ventilation is most appealing and has a physiologically sound base to avoid lung stress and strain, thus reducing VILI. Given those benefits, many ventilator manufacturers have incorporated esophageal pressure monitoring in their products. To our knowledge, this strategy has not been duplicated in any of the nonconventional modes of ventilation such as airway pressure release ventilation (APRV). We aimed to test the esophageal balloon in APRV and the resultant P_{E} especially the end expiratory P_{E} with different release times (T_{low}).

Introduced in the mid-1980s, APRV is considered by many as a nonconventional mode of ventilation [6]. APRV is an inverse ratio, pressure controlled, intermittent mandatory ventilation with unrestricted spontaneous breathing [7] that is mainly used as an alternative mode of ventilation in the difficult to oxygenate patient. APRV has many potential benefits described elsewhere [5] and beyond the scope of this paper. Briefly, it is the use of a long inspiratory pressure phase (P_{E}) that maintains alveolar recruitment. In addition, the expiratory pressure phase (P_{E}) and the release time (T_{low}) are kept very short to create auto-PEEP and to prevent end expiratory volume loss and alveolar derecruitment [8]. However, setting APRV has been a subject of much debate [7], especially the lack of consensus regarding its settings [9].

Patient and Methods

No ethics review committee approval was needed given the nature of the case report. The patient signed an approval to publish the case, and no personal information or photographs of the patient were included.

A 61-year-old obese male was admitted to the hospital with bilateral severe community-acquired pneumonia leading to acute respiratory failure and severe ARDS with PaO_{2}/FiO_{2} of 75. He was managed with pressure-targeted controlled mechanical ventilation (PCV–CMV), to target a tidal volume (VT) 6 mL/kg ideal body weight, and PEEP was adjusted to 15 cmH_{2}O to maintain oxygen saturation of 90%. However, because of high oxygen requirements (FiO_{2}), an esophageal balloon was inserted according to the manufacturer’s guidelines (Hamilton Medical AG, Switzerland). The ventilator settings were adjusted to keep inspiratory P_{E} below 20 cmH_{2}O and expiratory P_{E} 0–5 cmH_{2}O (Figure 1).

The ventilator mode was changed to APRV as follows: P_{E} was set to 30 cmH_{2}O (same as inspiratory pressure on PCV), P_{E} was set at 0 cmH_{2}O, T_{low} started at 0.1 s and increased by increments of 0.1 to 0.7 s, the release number was 10 s, with each cycle 6 s. T_{high} was variable from 5.9 to 5.3 s relative to the incremental increased T_{low}. We measured and recorded the airway pressure, esophageal pressure, inspiratory and expiratory P_{E}, VT, expiratory flow, and percentage decay of expiratory flow from peak expiratory flow (PEF) at the end of the T_{low}. Each setting was recorded for 2 min.

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RESULTS

The results are summarized in Figure 2 and Table 1. At all levels of $T_{Low}$ from 0.1 to 0.7 s, the expiratory $P_L$ was constantly a negative value indicating alveolar collapse. The expiratory flow at the end of the releases ranged from 75% to 87%.

DISCUSSION

The concept of using an esophageal pressure monitoring to guide setting mechanical ventilation, especially PEEP, has been on the rise and gaining momentum over the last decade. Research has shown that setting PEEP during conventional mechanical ventilation to maintain a positive $P_L$ at end of expiration improves oxygenation, respiratory compliance, and the trend towards improved mortality [4]. No such research was duplicated with the use of APRV. APRV setting, especially $T_{Low}$, has been the subject of much debate and confusion. The concept of $T_{Low}$ is to create a short release time creating auto-PEEP to avoid volume loss and alveolar derecruitment at end of expiration. In our 2012 review article [7], we described in detail the different methods of setting $T_{Low}$ in APRV with the relative advantages and disadvantages of each method. Briefly, it has been suggested to set $T_{Low}$ empirically in a range of 0.2–0.8 s [8, 10], but some have advocated to set it to achieve 50% – 75% of PEF [8]. Another study suggested to set it according to a certain time constant ($\tau$) (calculated as the product of the static respiratory compliance and resistance) and to adjust it for a certain tidal volume per release [11]. DiRocco et al. [12] suggested that alveolar
derecruitment still occurs despite short release time in APRV in an animal model of lung injury.

We previously found that the expiratory flow decay and, hence, auto-PEEP created during APRV is variable depending on the ventilator manufacturer and thus may not be reliable [13]. Similarly, another bench study that compared three different methods of setting $T_{\text{Low}}$ described the challenges and the unpredictability of auto-PEEP in APRV [14].

In a recently published APRV review [15], we called for research using innovative ways to set APRV, including the use of esophageal pressure monitoring or measuring the functional residual capacity. To our knowledge this is the first attempt to investigate the $P_L$ in APRV. Kollisch-Singule et al. [16] recently used esophageal balloon monitoring in APRV to monitor respiratory mechanics in an animal model of extrapulmonary lung injury with no mention of $P_L$ during the $T_{\text{Low}}$.

Our case is just “food for thought” and hopefully will encourage more research into this controversial and critical aspect of setting APRV. An intriguing observation in our case is that the esophageal pressure change during the release did not parallel the airway pressure (Figure 2) though as expected the drop increased steadily from 0.1 to 0.7 s because of the different compliances and resulting time constants between the lung and the chest wall. Our patient’s total respiratory system compliance ($C_{RS}$), calculated as the tidal volume divided by plateau pressure (obtained during brief inspiratory pause) – total PEEP, was 50 mL/cmH$_2$O; the chest wall compliance ($C_{CW}$), calculated as tidal volume divided by esophageal/pleural pressure ($P_{PL}$), was 29 mL/cmH$_2$O; and finally lung compliance ($C_L$), calculated as $C_{RS} - C_{CW}$, was 21 mL/cmH$_2$O (Figure 3). The worst compliance of the lung would mean faster emptying and collapse compared with the higher chest wall compliance. Furthermore, an important issue not to be missed, as our patient $\tau$ was 0.2 s (calculated as compliance × resistance, i.e., 0.05 L/cmH$_2$O × 4 cmH$_2$O/L/s), the expiratory flow did not decay by 63.2% each 0.2 s to almost reach zero at 4 time constants of 0.8 s as expected per a mathematical method previously published [7] (Figure 4).

---

**TABLE 1**

<table>
<thead>
<tr>
<th>$T_{\text{Low}}$ (s)</th>
<th>0.1</th>
<th>0.2</th>
<th>0.3</th>
<th>0.4</th>
<th>0.5</th>
<th>0.6</th>
<th>0.7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway pressure (cmH$_2$O)</td>
<td>11</td>
<td>10</td>
<td>10</td>
<td>9</td>
<td>9</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Esophageal pressure (cmH$_2$O)</td>
<td>18</td>
<td>17</td>
<td>15</td>
<td>14</td>
<td>14</td>
<td>13</td>
<td>12</td>
</tr>
<tr>
<td>Transpulmonary pressure (cmH$_2$O)</td>
<td>−7</td>
<td>−7</td>
<td>−5</td>
<td>−5</td>
<td>−5</td>
<td>−5</td>
<td>−4</td>
</tr>
<tr>
<td>% PEF</td>
<td>87</td>
<td>85</td>
<td>83</td>
<td>81</td>
<td>80</td>
<td>78</td>
<td>75</td>
</tr>
</tbody>
</table>

% PEF, percentage of decay of peak expiratory flow at the end of the release time; $T_{\text{Low}}$, time low or release time in seconds.

---

**FIGURE 3**

Ventilator graphics display during volume-controlled ventilation with an inspiratory hold maneuver to calculate the static compliance of respiratory system and airway resistance. On the X axis from top to bottom: airway pressure, esophageal pressure, and transpulmonary pressure all in cmH$_2$O. Y axis is time in seconds.

---

**FIGURE 4**

Lung simulator diagram of airway pressure release ventilation: volume (yellow), lung pressure (white), and flow (orange)/time curve. Time constant (TC) was known and the $T_{\text{Low}}$ was set to more than 4 TCs. The blue vertical lines represent each TC. Intrinsic PEEP at each TC would be equal to the point intersecting with the pressure curve, or it can be calculated as the end expiratory lung volume divided by respiratory compliance. Notice that at each TC the flow curve did not decay to 36.2% from its previous value as expected per the mathematical model. Reproduced with permission from Respiratory Care [5].

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**FIGURE 5**

Ventilator graphic display during APRV with an expiratory hold maneuver to calculate auto-PEEP, total PEEP, and end expiratory transpulmonary pressure at the end of the release time. On the X axis from top to bottom: airway pressure, esophageal pressure, and transpulmonary pressure all in cmH$_2$O and flow in L/min. Y axis is time in seconds.
This finding confirms our previous observation that the flow decay differs with the ventilator model and from the mathematical theory to the bedside [13]; therefore, using the time constant to set \( T_{\text{Low}} \) may not be accurate.

Our case has some limitations in addition to the inherent limitation of the esophageal balloon pressure monitoring. (i) This is a single patient report with a short observation time. (ii) The \( P_{\text{High}} \) and the \( T_{\text{Low}} \) were kept constant and were not adjusted, so consequently the \( T_{\text{High}} \) got shorter each time the \( T_{\text{Low}} \) was increased. In retrospect we might have needed to change those variables to evaluate if that would have an effect on the expiratory \( P_{E} \). (iii) The exact amount of auto-PEEP in APRV is hard to obtain or calculate at the bedside, and doing an expiratory hold maneuver at the end of the release was never documented as the accepted way to measure total PEEP or the auto-PEEP if using \( P_{\text{Low}} \) of 0 cmH\(_{2}\)O. Consequently, we did not apply an expiratory hold at the end of the release. Figure 5 shows an expiratory hold maneuver in a different patient on APRV. The expiratory \( P_{E} \) is negative using the airway pressure at the \( T_{\text{Low}} \), but it is positive at the end of the expiratory hold. In theory, calculating the \( P_{E} \) after the expiratory pause is the most appropriate way to set the \( T_{\text{Low}} \). This dilemma needs to be further investigated.

Hopefully new research and observations take into account these limitations.

**CONCLUSION**

Setting APRV, especially the \( T_{\text{Low}} \), with the aid of esophageal balloon to measure \( P_{E} \) is conceptually valuable and relatively feasible.

Setting \( T_{\text{Low}} \) in APRV according to the percentage of PEF might not be a valid method of avoiding alveolar collapse. More studies are needed to confirm these findings.

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CASE STUDY

Considerations for chest clearance and cough augmentation in severe bulbar dysfunction: a case study

Jodi Elizabeth Allen, MMedSci, MRes, Ellen Louise O’Leary, BSc


This case study describes a 21-year-old male with congenital myotonic dystrophy referred to respiratory physiotherapy with a weak cough and upper respiratory tract secretions. Mechanical insufflation–exsufflation (MI–E) was prescribed. Post initiation, the patient described a worsening of secretions and increased attendances to hospital with suspected chest infection. He also described difficulties with speaking after use of MI–E. Multidisciplinary assessment of cough as well as bulbar and swallow function resulted in a primary diagnosis of oro-pharyngeal dysphagia as well as weak cough. An alternative prophylactic therapy programme including active cycle of breathing, chest wall percussions, and manually assisted cough, was prescribed to facilitate clearance of upper airway secretions and patient comfort. The case highlights some of the risks associated with cough augmentation techniques derived from single-discipline intervention in the neuromuscular patient population. Comprehensive multidisciplinary assessment and management were key to redefining this patient’s diagnosis, allowing effective and individualised treatment.

Key Words: Neuromuscular; Cough; Mechanical insufflation–exsufflation; Bulbar; Dysphagia; Multidisciplinary team

INTRODUCTION

Myotonic dystrophy type 1 (DM1) is the most prevalent form of neuromuscular disease in adults, affecting 1 in every 8000 people worldwide [1]. It is a progressive multisystem genetic disorder caused by unstable trinucleotide (CTG) repeated expansions in untranslated DNA. There is a moderate correlation between longer CTG repeat expansions, an earlier age of onset, and more severe disease [2].

Congenital DM1 is a distinct clinical phenotype of DM1, which includes the clinical features of severe generalised weakness, hypotonia, and respiratory compromise. Weakness progresses slowly in early adulthood with severe cardiopulmonary complications manifesting into the third and fourth decade of life. Adults also present with bulbar weakness and oro-pharyngeal dysphagia (henceforth referred to as dysphagia) [3]. The average age of death is 45 years [2, 4].

Respiratory insufficiency is the leading cause of morbidity and mortality in the neuromuscular disease population [5]. Decline in respiratory muscle function results in ventilatory failure and impaired cough effectiveness leading to secretion accumulation, recurrent chest infections, and possible death [6]. Bulbar impairment (most commonly in the form of weakness) also predisposes individuals to weak cough, aspiration, and difficulty clearing oral secretions [5].

Peak cough flow (PCF) is a measure of cough strength. A PCF above 250–260 L/min may be sufficient to prevent pneumonia in people with neuromuscular disease [7, 8]. Physiotherapists assess PCF and tailor treatment to the component(s) of the cough cycle affected: inspiratory muscle strength, glottic closure, and expiratory muscle strength [9]. Bulbar impairment results in altered range, strength, or sequencing of glottic movement, which reduces PCF even in the absence of respiratory muscle weakness [10, 11]. Speech and Language Therapists (SLT) assess bulbar function via direct testing of muscle strength and range and rate of movement, plus they assess voice, speech, and swallowing. Bulbar impairment also causes dysphagia resulting in poor clearance of food, fluids, and/or saliva from the mouth and pharynx with risk of material being aspirated into the airway.

Cough augmentation is helpful for clearing pulmonary secretions and is useful in the prevention and treatment of pneumonias [5, 6, 8]. Mechanical insufflation–exsufflation (MI–E), a form of cough augmentation, is used to maintain respiratory health in advanced neuromuscular disease [10]. Positive and negative pressure is sequentially applied to the airways to facilitate upward clearance of pulmonary secretions [12]. For those with bulbar impairment, the suitability of MI–E is currently unclear. MI–E can provoke significant laryngeal addiction during inspiratory and expiratory stages in patients with bulbar impairment secondary to amyotrophic lateral sclerosis [11, 13]. Whilst pressures should be titrated to suit the needs of the individual [11–14]; those with severe bulbar involvement are often excluded from studies evaluating the efficacy of MI–E [10, 15]. This lack of consensus leaves clinicians to assess the risk versus benefit of MI–E on an individual patient basis. This case study considers the relevance of comprehensive bulbar assessment to inform prophylactic cough augmentation in neuromuscular disease and suitability of MI–E in the presence of severe bulbar impairment.

CASE PRESENTATION

A 21-year-old male with a diagnosis of genetically confirmed congenital DM1 was electively admitted to adult neuromuscular services in October 2016 for review of his MI–E settings. MI–E had previously been provided by the respiratory physiotherapist upon request of the treating medical team because of weak cough and difficulty clearing secretions. Following initiation of MI–E, the patient had three hospital admissions, all for suspected chest infections. The patient’s mother also described an accumulation of secretions and difficulty speaking for up to an hour after use of MI–E. A referral was made to physiotherapy to adjust MI–E settings.
Relevant medical history included right hemiplegic cerebral palsy due to a neonatal intracerebral haemorrhage, adenotonsillectomy, sialorrhea, and tongue reduction surgery. A diagnosis of respiratory failure was made at 18 years of age when non-invasive ventilation was prescribed.

Informed consent was obtained from the patient after the nature of the procedure had been fully explained.

EXAMINATION

The individual had marked scoliosis (cobb angle > 90° ) with an asymmetrical posture in his powered wheelchair. Weight was reported to be stable at 51.2 kg. Peak cough flow was 140 L/min as measured by Wrights Mini Peak Flow Meter with a facemask. Maximum insufflation capacity was not completed owing to an inability to breath stack, suggestive of dysfunction at the level of the glottis. Clinical signs of pooled oral and pharyngeal secretions were evident despite active prescription of hyoscine. The physiotherapist initiated a referral to SLT to establish the influence of bulbar muscle weakness on the individual’s sialorrhea, weak cough, and poor outcomes of MI–E.

SLT assessment identified: macroglossia (large tongue), mandibular prognathism (protrusion of the mandible beyond the top teeth), bilateral weakness of the face and tongue, pooled saliva in the oral cavity, moderate–severe dysarthria, and intermittent wet vocal quality suggestive of a severe underlying dysphagia secondary to weakness.

We hypothesised that bulbar weakness was the primary cause of the patient’s weak cough. This same impairment was likely to be affecting swallowing function resulting in unmanaged saliva and pooled food and fluids in the pharynx.

INVESTIGATIONS

Investigations aimed to (i) examine the bulbar impairment and its impact on cough and swallowing and (ii) understand the influence of the bulbar impairment on the suitability of use of MI–E.

Assessment via fibreoptic endoscopic evaluation of swallowing (FEES) revealed gross pooling of secretions throughout the pharynx at rest which were breathed into and out of the airway. This indicated a diffuse pharyngeal motor and sensory impairment (Figure 1). On provision of food and drink, material remained in the pharynx entering the laryngeal vestibule with eventual aspiration (Figure 2). Volitional coughing was strong enough to eject some material from the laryngeal vestibule but not strong enough to propel material from the pharynx into the mouth (Figure 3). Secretions, food, and fluid continued to spill into the laryngeal vestibule without clearance with more to eat and drink.

To support decisions regarding ongoing use of MI–E, nasendoscopic images were recorded before and after its use (insufflation pressure +10 cmH₂O, exsufflation pressure –40 cmH₂O manual). After use of MI–E, secretions were immediately expectorated from the oral cavity and the nasendoscopic view of the pharynx and upper airway was visually clearer (Figure 4).

Videofluoroscopy (VFS) was conducted to examine the impact of MI–E on the lower airway, anatomy that cannot be visualised via FEES. Prior to MI–E, swallowing strategies were provided to clear the pharynx as much as possible and continued until residues failed to clear any further. Figure 5 shows pre- and post-MI–E VFS images. Material not visible prior to MI–E was subsequently seen in the lower airway suggesting that although MI–E is useful to propel upper airway material from the pharynx to mouth with low inspiratory pressures, it may have also propelled pooled material into the lower airway.
**FIGURE 3**
Fibreoptic endoscopic evaluation of swallowing images after volitional coughing: clearance from the airway but not the pharynx.

- Cough not strong enough to propel food and drink material from the pharynx back into the mouth
- Majority of food and drink material ejected from the upper airway after volitional coughing

**FIGURE 4**
Fibreoptic endoscopic evaluation of swallowing images pre- (left) and post- (right) mechanical insufflation–exsufflation.

- Upper airway houses more food and drink pre MI-E (L) than post MI-E (R)
- Pharynx houses more food and drink pre MI-E (L) than post MI-E (R)

**FIGURE 5**
Videofluoroscopy images pre (left) and post (right) mechanical insufflation–exsufflation (MI–E). Arrow on right highlights material in trachea not evident pre MI-E.
A diagnosis of severe bulbar impairment affecting both cough and swallow was confirmed. Poor glottic function contributed to poor cough strength. Poor swallowing function resulted in pharyngeal residues of food, fluid, and pooled saliva. Using MI–E in the presence of pharyngeal residues is likely to have provoked aspiration.

**MANAGEMENT**

Given the severity of the dysphagia, a recommendation was made for enteral feeding to eliminate risk of aspiration from oral food and fluids. Pharmacological review was requested to optimise anticholinergic management of saliva. Given the likely provocation of aspiration from MI–E, the history of patient discomfort following its use, and the multiple hospital attendances since its prescription, a recommendation was made to cease MI–E until dysphagia and secretion management had been optimised.

Active cycle of breathing exercises and manual therapeutic techniques including chest wall percussions and manually assisted cough were prescribed as alternative chest clearance techniques. Continued use of regular oral suction to clear excess saliva was advised. Recommendations were made that MI–E should still be considered in the event of an acute chest infection where the necessity of airway clearance for optimal ventilation may likely outweigh chronic risks of infection secondary to provoked aspiration.

**OUTCOMES**

After balancing the risks and benefits of oral and enteral feeding, the patient elected to continue eating and drinking in the least restrictive manner unless he became critically unwell with aspiration pneumonia. In June 2017, eight months after an alternative chest clearance programme had been implemented, the individual reported a reduction in upper respiratory tract secretions and denied any further hospital admissions. He remains under the care of the local SLT team for monitoring of his symptoms and continues to have regular discussions regarding enteral feeding.

**DISCUSSION**

Respiratory insufficiency secondary to neuromuscular disease can lead to ventilatory and cough failure resulting in recurrent pneumonias [6]. Dysphagia and bulbar weakness provoke symptoms similar to those associated with respiratory weakness, namely upper respiratory secretions, chest infections, and weak cough. In this case, respiratory symptoms were indicative of both dysphagia and cough failure. We argue that implementation of MI–E without having explored the significance of the dysphagia and bulbar impairment may have resulted in recurrent aspiration and persistence of these respiratory symptoms. Consideration of dysphagia should therefore be integral to routine neuromuscular respiratory assessment.

There is a growing literature regarding implementation of MI–E in patients with neuromuscular disease and bulbar impairment [11, 13, 16, 17]. The need for individual titration of MI–E in patients with bulbar impairment to minimise laryngeal adduction during insufflation and exsufflation is well documented [11, 13]. Bulbar impairment can however lead to dysphagia. Studies have not explored the influence of this dysphagia, which may constitute a contraindication for MI–E.

Retained oral and pharyngeal residues are well documented in DM1 and other neuromuscular disease groups [20]. These residues are the primary concern for patients requiring cough augmentation, regardless of primary aetiology. Although the additional diagnoses of cerebral palsy and scoliosis may also have affected swallowing and respiratory function in this case, examining this influence is beyond the scope of this report.

MI–E settings of insufflation (+10 cmH₂O) and exsufflation (–40 cmH₂O) with a low flow were tailored to bias upward movement of secretions in this case. In the presence of pharyngeal secretions caused by dysphagia, positive pressure is likely to have provoked aspiration as well as support some upward clearance to the mouth. Daily provoked aspiration as a consequence of a chest clearance plan undermines the very purpose for which it has been implemented; hence, the decision to cease rather than titrate MI–E. Nasendoscopy was a useful tool in establishing the presence of pharyngeal secretions and the need for dysphagia management prior to provision of positive pressure adjuncts.

We advocate for collaborative working between physiotherapy and speech and language therapy in the context of patient-centred prophylactic neuromuscular respiratory management.

**CONCLUSIONS**

Careful evaluation of the physiological benefits and consequences of cough augmentation is required when assessing people with bulbar impairment. Clinicians should challenge the habits of prophylactic MI–E provision without considering the potential impact of providing positive pressure adjuncts in the presence of pooled secretions secondary to an underlying dysphagia. Multidisciplinary collaboration is essential in providing individually tailored care in this complex patient group.

**DISCLOSURES**

All authors contributed to the concept or design of the work, the acquisition, analysis, or interpretation of the data. All authors were involved in drafting and commenting on the paper and have approved the final version. This study did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. The authors declare no financial relationships with any organizations that might have an interest in the submitted work in the previous three years and no other relationships or activities that could appear to have influenced the submitted work.

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CORRIGENDUM

Medical Air: What you don’t know won’t hurt you, but what about your patients?

Paul Edwards¹ and Patricia-Ann Therriault², RRT, FCSRT, INH., FSCRT


The hyperlinks in the reference list have been updated. The correct reference list is presented below.

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* Clinical significance unknown.


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