CASE STUDY

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

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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 expiratory 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 (P₁), 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 (P₁). According to the study protocol, the end inspiratory P1 was kept under 25 cmH20 and the end expiratory P1 between 0 and 10 cmH₂0. 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 P1, especially the end expiratory P_I , with different release times $(T_{I_{OW}})$.

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_{High}) that maintains alveolar recruitment. In addition, the expiratory pressure phase (P_{Low}) 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 cmH₂O to maintain oxygen saturation of 90%. However, because of high oxygen requirements (FiO₂), an esophageal balloon was inserted according to the manufacturer's guidelines (Hamilton Medical AG, Switzerland). The ventilator settings were adjusted to keep inspiratory P₁ below 20 cmH₂O and expiratory P₁ 0–5 cmH₂O (Figure 1).

The ventilator mode was changed to APRV as follows: P_{High} was set to 30 cmH₂O (same as inspiratory pressure on PCV), P_{Low} was set at 0 cmH₂O, 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_L , 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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FIGURE 1

Ventilator graphics display during pressure-controlled ventilation. On the X axis from top to bottom: airway pressure, esophogeal pressure, and transpulmonary pressure, all in cmH₂O. Y axis is time in seconds.



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 P₁ 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 $\mathrm{T}_{\mathrm{Low}}$, 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 dercruitment at end of expiration. In our 2012 review article [7], we described in detail the different methods of setting $T_{_{\rm Low}}$ 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 (τ) (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

FIGURE 2

Ventilator graphics display during APRV showing summary of airway pressure, esophogeal pressure, transpulmonary pressure, all in cmH₂O and flow in L/min, on X axis during different release times (0.1 – 0.7 s). Y axis is time in seconds.



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_{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_{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/cmH₂0; the chest wall compliance (C_{CW}), calculated as tidal volume divided by esophageal/pleural pressure (P_{PL}), was 29 mL/cmH₂O; and finally lung compliance (C_{L}), calculated as C_{RS} – C_{CW} was 21 mL/cmH₂O (Figure 3). The worst compliance of the lung would mean faster emptying and collapse compared with the higher chest

TABLE 1

Patient test results

	T _{Low} (s)							
	0.1	0.2	0.3	0.4	0.5	0.6	0.7	
Airway pressure (cmH ₂ O)	11	10	10	9	9	8	8	
Esophogeal pressure (cmH,O)	18	17	15	14	14	13	12	
Transpulmonary pressure (cmH,O)	-7	-7	-5	-5	-5	-5	-4	
% PEF	87	85	83	81	80	78	75	

% PEF, percentage of decay of peak expiratory flow at the end of the release time; $T_{\rm Low}$, 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, esophogeal pressure, and transpulmonary pressure all in cmH₂O. Y axis is time in seconds.

55 10 25.0 4.0	30 ^{Ppeak} cmH20 9.2 ^{ExpMinVol}	40 Paw 28 Ptrach 20 20 Minute 20 Min
1100 200	508 ^{mt}	40-Pest Paux) 40-Pest Paux) 40-CmH20
40 10	18 ^{fTotal}	30- 20- 10-
	22 ^{Pmean} cmH20	0 + + + + + + + + + + + + + + + + + + +
	18 fTotal brmin fSpont brmin	

wall compliance. Furthermore, an important issue not to be missed, as our patient τ was 0.2 s (calculated as compliance × resistance, i.e., 0.05 L/cmH₂O × 4 cmH₂O/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).

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_{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].



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 cmH₂O and flow in L/min. Y axis is time in seconds.



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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_{Low} may not be accurate.

Our case has some limitations in addition to the inherit limitation of the esophageal balloon pressure monitoring. (i) This is a single patient report with a short observation time. (ii) The $\rm P_{High}$ and the $\rm T_{Low}$ were kept constant and were not adjusted, so consequently the $\rm T_{High}$ got shorter each time the T_{Low} was increased. In retrospect we might have needed to change those variables to evaluate if that would have an effect on the expiratory P₁. (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_{Low} of 0 cmH_2O. 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_{I} is negative using the airway pressure at the $T_{I,ow}$, but it is positive at the end of the expiratory hold. In theory, calculating the $P_{\rm L}$ after the expiratory pause is the most appropriate way to set the T_{Low}. This dilemma needs to be further investigated. Hopefully new research and observations take into account these

limitations.

CONCLUSION

Setting APRV, especially the $T_{\mbox{\tiny Low}}$, with the aid of esophageal balloon to measure P_L is conceptually valuable and relatively feasible.

Setting T_{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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