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Research Article

Efficiency of the Optimization of Pressure-Support Ventilation Based on Analysis of the Diaphragmatic Electrical Activity

Maxime Grigoli, Laure Crognier, Fanny Vardon-Bounes, Stéphanie Ruiz, Antoine Rouget, Vincent Minville, Olivier Fourcade, Jean Marie Conil and Bernard Georges*

Service de Réanimation Polyvalente, CHU Rangueil, 1 Avenue Jean Poulhès, Pôle d'Anesthésie et Réanimation, Toulouse Cedex, France

***Corresponding Author:** Bernard Georges, Department of Anaesthesiology and Intensive Care Units, University Hospital of Toulouse, Toulouse Cedex, France.

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Abstract

Purpose: The main goal of our study was to demonstrate a decrease in the total number of patient-ventilator asynchronies in pressure-support ventilation by using the EAdi signal in comparison with ventilator pressure and flow curves single analysis.

Materials and Methods: This was a prospective, sequential, non-randomized study. We compared the number of asynchronies in PSV during two phases in patients at risk for weaning difficulties using pressure and flow curves of the ventilator or using the analysis of the electromyographic signal of the diaphragm (EAdi).

Results: Thirty-one patients were included. We found a decrease in the number of asynchronies per minute (1.8 vs 2.6/minute, p = 0.0062) and the index of asynchrony (5.7 vs 8.6%, p = 0.0037) during the optimization phase based on the analysis of EAdi signal compared to the first phase. The pressure-support was significantly lower during the second phase (11 vs 8 cmH2O, p = 0.001) and expiratory trigger was earlier (50% vs 40%, p = 0.0023).

Conclusion: The analysis of the electromyographic signal of the diaphragm associated with the respirator curve analysis allows a reduction in patient-ventilator asynchrony. Optimizing pressure support level and expiratory cycling could allow a limitation of over-assistance, but further studies are necessary in order to determine the clinical impact of this improved synchrony.

Keywords: *Electromyographic Signal; Patient Ventilator Asynchrony*

Abbreviations

AI: Asynchrony Index; ARDS: Acute Respiratory Distress Syndrome; COPD: Chronic Obstructive Pulmonary Disease; Eadi: Electromyographic Signal of the Diaphragm; ICU: Intensive Care Unit; iPEEP: Intrinsic Positive End-Expiratory Pressure; NAVA: Neurally Adjusted Ventilatory Assist; PBW: Predicted Body Weight; PEEP: Positive End-Expiratory Pressure; PSV: Pressure-Support Ventilation; RASS: Richmond Agitation Sedation Scale; SAPS: Simplified Acute Physiology Score; VT: Tidal Volume

Introduction

Prolonged weaning from mechanical ventilation is defined by continued ventilation more than 7 days after first spontaneous breathing trial and weaning failure is defined as the failure to pass a spontaneous-breathing trial or the need for reintubation within 48 hours following extubation [1]. This is associated with an increased in-hospital mortality. Almost 25% of mechanically ventilated patients experience prolonged weaning in Intensive Care Unit (ICU) [1]. For these patients, the time spent in weaning can reach half of the total duration of invasive ventilation [2,3]. To limit difficult weaning from ventilator support, the setting of the ventilator may to allow the

patient to generate spontaneous efforts and to reduce the patient's work [4,5]. So, physicians should theoretically improve patient-ventilator synchronization so as to reduce ventilator-associated diaphragmatic dysfunction. Pressure Support Ventilation (PSV) with positive end-expiratory pressure (PEEP) is commonly used, as recommended by the International Consensus Conference on Intensive Care Medicine [1,6].

Major asynchronies are present in more than 25% of the mechanically ventilated patients [7]. Asynchronies increase the duration of mechanical ventilation and both ICU and hospital lengths of stay, which are potentially a cause of morbidity and mortality [7-9]. Major asynchronies are easy to detect on the respiratory curves and are represented by ineffective effort, auto-triggering and double-triggering [7,10]. But there are also others asynchronies, which are more difficult to detect and which are inspiratory delay and late cycling. These asynchronies are explained by an incorrect delay between diaphragmatic signal and ventilator response [11]. They are represented by too much delay between diaphragmatic contraction (start of inspiration) and respiratory insufflation, or a prolonged inspiratory cycle while the patient is already in expiration time with diaphragmatic relaxation [12-14]. Prolonged inspiration time favors other asynchronies and are potentially responsible for dynamic hyperinflation and increased work of breathing [15].

Patient-ventilator asynchrony detection using flow and pressure-time curves has a poor sensitivity and detecting patient-ventilator asynchronies at the bedside is sometimes difficult even for experienced clinicians [16]. To detect these asynchronies, neural diaphragmatic electrical activity (Eadi) recorded using specialized nasogastric tubes equipped with electrodes could be used to optimize the ventilator settings to improve the matching between the patient and the ventilator [17].

The main objective of our study was to demonstrate a decrease in the total number of patient-ventilator asynchronies and in asynchrony index [7] using Eadi signal in comparison with ventilator pressure and flow curves single analysis. The secondary objectives were to identify which types of asynchronies were decreased and to assess the optimization in pressure level support.

Materials and Methods

Study design

This was a prospective, sequential, non-randomized, single-center study conducted in the medical/surgical ICU of Toulouse Rangueil University Hospital, France, from May 2013 to January 2014. The study was approved by the Ethics Research Committee of our institution (No. 25- 0413, April 17, 2013). Only oral information was given to the patient or the family before inclusion as requested by the Ethics Research Committee, but the patient or his relative could decline to participate.

Patients

Patients admitted to the ICU, ventilated using PSV with an established prolonged weaning or with suspected predictive criteria for prolonged weaning were eligible for inclusion. The predictive criteria for prolonged weaning were defined by duration of mechanical ventilation more than 7 days after first spontaneous breathing trial, or reintubation within 48 hours following extubation. Respiratory diseases (Chronic Obstructive Pulmonary Disease (COPD), restrictive diseases), chronic heart diseases (left ventricular failure, coronary artery disease), recent complicated abdominal or thoracic surgery were also taken into account as predictive criteria of difficult weaning. Patients RASS (Richmond Agitation Sedation Scale) score should be equal to zero, without sedation for more than 24 hours. Exclusion criteria were patients under 18 years old, pregnancy, contraindication to Eadi nasogastric tube placement (recent esophageal or gastric surgery, esophageal varices), absence of the Eadi catheter at the time of inclusion and placement for study reasons, progressive infectious process (temperature above 38.5°C), hemodynamic failure (mean arterial pressure less than 65 mmHg or catecholamine treatment), decision to withhold life-sustaining treatment, poor short-term prognosis and presence of a guardianship.

Study protocol

Before weaning from ventilator support, patients were ventilated in one of the two pressure level modes used in our department such as PSV or Neurally adjusted ventilatory assist (NAVA) using Eadi catheter (Maquet Critical Care, Solna, Sweden). When the patient presented weaning criteria (cessation of any sedation, inotropic or vasopressor treatment, $FiO_2 \leq 50\%$ and PEEP ≤ 5 cmH_2O), in accordance with our protocol of daily screening for weaning criteria, a weaning trial was performed. During the first phase where patients were ventilated in PSV, ventilation parameters were adjusted by an expert physician in ventilation, separately for each patient, in accordance with recommendations of optimizing the absence of asynchronies without the help of Eadi signal. The Eadi curve was not accessible when adjusting the first phase. The level support was set to obtain a tidal volume (VT) about 6 ml/kg of predicted body weight (PBW). The inspiratory trigger in-flow was adjusted as low as possible without auto-triggering. External PEEP and expiratory cycling were also optimized with fan flow curves such as expiratory flow returned to zero, unless if the patient had expiratory muscle activity. Maquet trigger range from 1 to 10 corresponds to 3 to 1 L/min. Because this study was a non-interventional study, we didn't measure intrinsic PEEP. Expiratory pause, as practiced in controlled ventilation, was not suitable here. In pressure support ventilation, the assessment of esophageal pressure decrease in regard of the intragastric pressure was not possible because we didn't use esophageal or gastric pressure sensor. As flow curve didn't return to zero, we deduced the presence of an intrinsic PEEP.

Just before respiratory curves recording, the electromyographic sensor was connected to the Servo-I® ventilator and both respiratory curves and Eadi signals were registered. In the second phase, respiratory settings, level support, inspiratory trigger in-flow, external PEEP and expiratory cycling were optimized by the same expert physician while taking into account the Eadi analyses so as to minimize asynchronies highlighted by the Eadi signal. To adjust the expiratory cycling, we considered that the end of neural inspiratory effort was reached after 70% Eadi signal decrease [11]. Then, respiratory curves and electromyographic signal were recorded. The second phase was carried out rapidly after the first in order to be certain regarding patient stability. Investigators were not involved in setting the ventilator.

Data acquisition

To collect data, we used respiratory curves recovery software *(Servo-I software CPR, Maquet Critical Care).* During each 75 minutes phase, pressure, flow and volume curves and Eadi signal were recorded. Then, we proceeded manually by analyzing 8 periods of one minute every ten minutes, ie an analysis period of 8 minutes per phase. We also noted VT, expiratory minute volume (Vmin), maximum Eadi signal (Eadimax) and respiratory rate.

Asynchronies analysis

Analysis of respiratory curves was manually performed together by two independent physicians, a third one being solicited in case of disagreement.

Five types of asynchronies were analyzed, that we can define as 3 major asynchronies or macro asynchronies, and 2 others as micro asynchronies:

- Ineffective triggering defined by the existence of a diaphragmatic signal (flow decrease) not followed by a ventilator cycle during the ventilator expiratory phase (Figure 1).
- Auto-triggering defined by the existence of a ventilator cycle without diaphragmatic signal (Figure 2A).
- Double triggering defined according to Thille [7], by the presence of two successive inspiratory cycles without intermediate expiration or separated by an expiratory time less than one-half the mean inspiratory time (Figure 2B).
- Inspiratory delay (micro-asynchrony) defined by a delay of over 200 milliseconds between diaphragmatic contraction and the beginning of insufflation. In the absence of consensual definition, we used the delay of 150 milliseconds that represents the conscious perception threshold and that could be source of discomfort [18]. Given the precision of the curves analysis software to measure the delays, we added a 50 milliseconds safety margin to be sure not to overestimate these micro-asynchronies (Figure 3A).

- Late cycling (micro-asynchrony) defined by excessive inspiratory time of more than 200 milliseconds between the end of diaphragmatic contraction and the opening of expiratory valve [11]. The time delay of 200 milliseconds has been selected for the same reason (Figure 3B).

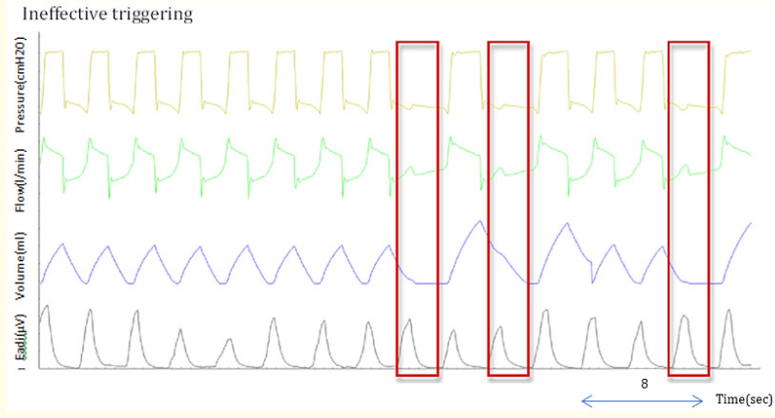

Figure 1: *Ineffective triggering.*

Yellow line: Pressure (cmH_2O); Green line: Flow (l/min); Blue line: volume (ml); Grey line: Eadi (µV).

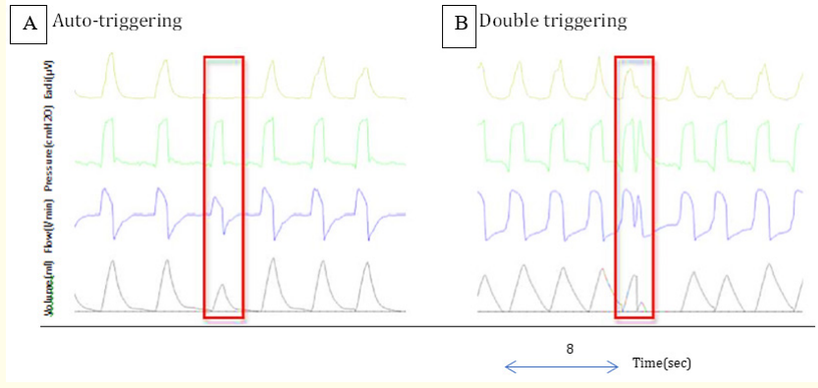

Figure 2: *Auto-triggering and double triggering.*

Yellow line: Eadi (µV); Green line: Pressure (cmH_2O); Blue line: Flow (l/min); Grey line: volume (ml).

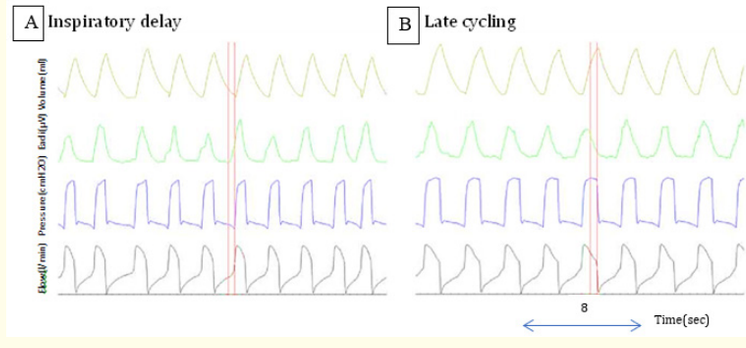

Figure 3: *Inspiratory delay and late cycling*

Yellow line: volume (ml); Green line: Eadi (µV); Blue line: Pressure (cmH_2O); Grey line: Flow (l/min).

The total number of asynchronies was then calculated for each phase. The asynchrony index (AI) was calculated. This index corresponded to the total asynchronies divided by the number of diaphragmatic cycles (ventilator cycles + wasted efforts) multiplied by 100. For each type of asynchronies, we also calculated the percentage of asynchronies which was the number of asynchronies divided by the number of total respiratory cycle for the period x 100. Furthermore, we calculated the VT in ml/kg of PBW, corresponding to the different recordings periods.

Statstical analysis

Characteristics of the population and analyzed variables were described with median and 95% confidence interval (95% CI) because of the non-homogeneity of the groups and the non-Gaussian distribution of the majority of the variables.

The different respiratory phases were compared using non-parametric tests:

- Wilcoxon test (paired samples) for continuous variables;
- Cochran's Q test for qualitative variables.

The study was carried out using MedCalc® statistical software version 15 (Mariakerke, Belgique). A $p< 0.05$ was considered to be statistically significant.

The main objective was to demonstrate a decrease in total number patient-ventilator asynchronies, and in asynchrony index. Power analysis indicated that a sample size of 28 was sufficient to demonstrate a 20% reduction in the number of asynchronies between the two settings of PSV modes, with α and β risks of 0.05 and 0.20 respectively.

Results

Population

The study included 31 patients. The main characteristics at inclusion are summarized in table 1. Median severity score (SAPS II) was equal to 58. Sixteen patients (52%) presented with several criteria of prolonged weaning. The median duration of weaning from ventilatory support was 8 days for an overall ventilation time of 15 days. Failed extubation was experienced by 10 patients (32%) before inclusion in the study.

Gender male/female	23/8
Age (years)	65 (53.6-72.7)
BMI (Kg/m^2)	28 (24.7-30.2)
SAPS II	58 (45-66)
Diseases predicting a difficult weaning	
COPD (%)	19 (61)
Heart failure (%)	8 (26)
Coronary artery disease (%)	9 (29)
Gastrointestinal surgery (%)	7 (23)
Cardio-Thoracic surgery (%)	9 (29)
Neuromuscular disease (%)	1 (3)
Length of ventilator weaning (days)	8 (6 - 8.8)
Length of mechanical ventilation (days)	15 (11.6 - 20)
ICU length of stay (days)	20 (17 - 25.4)
Failure at least one time extubation (n (%))	10 (32)
Mortality (n (%))	6 (19)

Table 1: *Characteristics of the population (n = 31).*

Data are expressed as median (confidence interval 95%) or counts (percentage).

Changes in ventilator settings

Ventilation settings are shown in table 2. The median of pressure support level was 11 cmH_2O (9.2 - 12) in the first phase (PSV) and 8 cmH_2O (6.6 - 10) in the second phase (PSV optimized with Eadi signal). Pressure support level was lowered in 14 patients (45%). It was not modified in 7 patients (23%) and was increased in 10 patients (32%) in phase 2. The difference in pressure support was significant as well as the expiratory trigger setting (p = 0.01), as a percentage of peak flow, which was earlier in phase 2 (40% *vs*. 50%, p = 0.023). Median inspiratory flow trigger level was 7 (6 - 8) in the first phase and 8 (5 - 8) in the second phase.

	Phase 1	Phase 2	*p*
Pressure support (cm H_2O)	11(9.2 - 12)	8 (6.6 - 10)	0.001*
PEEP (cm H_2O)	6 (5 - 6)	6 (5 - 6.4)	0.8552
Slope (sec)	0.05 (0.05 - 0.05)	0.05 (0 - 0.05)	0.0654
Sensibility of inspiratory trigger in flow (1-10)	7 (6 - 8)	8 (5 -8)	0.4037
Expiratory cycling (% of peak flow)	40 (35 - 40)	50 (40 - 52)	0.0023*
FiO_2 (%)	35 (30.9 - 40)	35 (30 - 35)	0.0039*
PaO_2/FiO_2	271 (240 to 372)	303 (271 to 372)	0,0021*
Ventilation rate (breaths/min)	29 (26 - 30)	29 (26 - 32)	0.6480
Expiratory minute volume (L/min)	12.3 (11 - 13.2)	11.2 (9.9 - 13)	0.0745
Expiratory volume (mL)	422 (382 - 454.7)	393 (369.6 - 442.4)	0.0304*
Tidal volume (mL/Kg of PBW)	6.5 (5.8 - 6.9)	6.2 (5.7 - 6.5)	0.0324*
Maximal Eadi (µV)	14.8 (11.2 - 16.1)	17 (12.9 - 20.9)	0,0859

Table 2: *Ventilator settings.*

*Data are expressed as median (95 % confidence interval) * ($p < 0.05$).*

PEEP level was lowered in 8 patients (26%), was identical in 16 patients (52%) and was increased in 7 patients (23%) but the difference was not significant.

Ventilation frequency was not different between the two phases, but there was a decrease in tidal volume with a median of 6.5 ml/kg of PBW in phase 1, against 6.2 ml/kg of PBW in Phase 2 (p = 0.0324). We noted a significant decrease in FiO_2 and improvement of PaO_2/FiO_2 in phase 2.

Effect of PSV optimization on asynchronies

During the second phase with PSV optimization using Eadi signal, the number of asynchronies each minute was lower than in standard PSV (1.8 *vs*. 2.6/min, p = 0.0062). Asynchrony index (AI) was reduced in phase 2 (5.7 *vs* 8.6%; p=0.0037) as shown in figure 4. AI higher than 10% was found in 14 cases (45%) in phase 1 and 9 cases (29%) in phase 2, but difference was not significant.

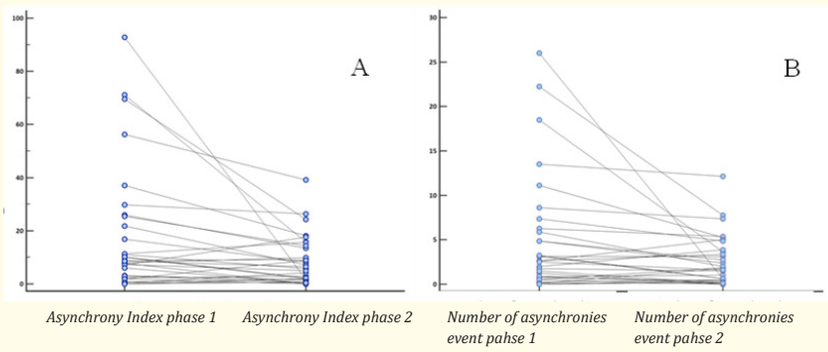

Figure 4: *Asynchrony index (A) and number of asynchronies (B) in phase 1 and phase 2.*

This reduction of overall asynchronies was due to a reduction of micro-asynchronies. Indeed, the percentage of ineffective triggering, auto-triggering and double triggering was not different between the two phases. However, the percentage of inspiratory delay and late cycling was slightly lower in phase 2 than in phase 1. These results are shown in table 3.

	Phase 1	Phase 2	*p*
Asynchronies			
Number (events/min)	2.6 (1.1 - 4.9)	1.8 (0.63 - 3.2)	0.0062*
Index (%)	8.6 (4.8 - 13.6)	5.7 (2.0 to 9.5)	0.0037*
Asynchrony Index > 10% (n.%)	14 (45.2%)	9 (29.0%)	0,059
Ineffective triggering (%)	0(0 to 0.4)	0 (0 - 0)	0.0676
Auto-triggering (%)	0.54 (0.4 to 1.3)	0.49 (0 to 1.4)	0.5615
Doubles triggering (%)	0(0 - 0.45)	0 (0 - 0.43)	0.7983
Inspiratory delay (%)	1.6 (0.5 to 13)	1.1 (0.4 to 6.2)	0.0015*
Late cycling (%)	0 (0 -0.2)	0 (0 - 0)	0.0029*

Table 3: *Comparison of asynchronies during the two phases.*

*Data are expressed as median (95% confidence interval) or counts (percentage) * p < 0.05.*

Taking into account only patients with COPD, we find a significant reduction in the number of asynchronies and the index of asynchrony. This reduction of overall asynchronies was due to a reduction of ineffective triggering and inspiratory delay (Table 4).

	Phase 1	Phase 2	*p*
Asynchronies			
Number (events/min)	2.6 (1.1 - 5.3)	1.9 (0.6 - 3.2)	0.0230*
Index (%)	10.5 (4.9 - 20.2)	5.9 (2.1 - 9.9)	0.0129*
Asynchrony Index > 10% (n.%)	12 (57.1%)	6 (28.6%)	0.034*
Ineffective triggering (%)	0 (0 - 0.5)	0 (0 - 0)	0.0186*
Auto-triggering (%)	0.5 (0.2 - 1.4)	0.4 (0 - 1.4)	0.3465
Doubles triggering (%)	0 (0 - 0.5)	0 (0 - 0.4)	0.6772
Inspiratory delay (%)	4.7 (0.3 to 16.6)	2 (0.3 to 7.1)	0.0159*
Late cycling (%)	0 (0 - 0)	0 (0 - 0)	0.0625

Table 4: *Comparison of asynchronies during the two phases in hospitalized patients for respiratory disease (n = 21).*

*Data are expressed as median (confidence interval 95%) or counts (percentage). * is significant with p lower than 0.05.*

Discussion

The present study showed a significant decrease in overall asynchronies each minute and in asynchrony index using Eadi signal in patients with prolonged weaning from ventilatory support. This decrease was mainly due to a decrease in micro-asynchronies, represented by inspiratory delay and late cycling.

Using an Eadi-based optimization strategy, we decreased pressure support level (11 *vs* 8 cmH_2O) and increased expiratory cycling (40% *vs* 50%). Slope, PEEP and inspiratory trigger in-flow were not changed between the two phases.

Expiratory cycling was earlier in the 2 phases than the initially proposed conventional setting at 25%, which generates a shorter insufflation time [19].

These settings had very little influence on ventilatory patient's parameters (respiratory rate, expiratory minute volume, and median Eadimax (14.8 *vs* 17 µvolts, ns). However, there was a significant difference in tidal volume (6.5 *vs* 6.2 mL/kg).

The decrease of both pressure support and tidal volume limit the risk of dynamic hyperinflation and decrease the work of breathing related to this dynamic hyperinflation. It also limits the emergence of late cycling and inspiratory delay on the next cycle. Extended breaths can induce intrinsic positive end-expiratory pressure (iPEEP), especially in COPD patients [8]. Optimizing expiratory cycling setting allows minimizing the risk of dynamic hyperinflation and iPEEP. In our study, the very early setting of expiratory cycling in phase 1 was due to the interest of practitioners in our service concerning difficult weaning of COPD patients, but we did not specifically measure iPEEP.

Tidal volume target between 6 and 8 mL/kg of PBW is recommended for protective ventilation in acute respiratory distress syndrome (ARDS) to reduce the risk of baro and volutrauma [20]. However, some studies suggest that protective ventilation may also reduce the risk of lung injury in ventilated patients without ARDS [21]. In our study, tidal volumes correspond to current recommendations [12,22]. During step 1, the risk of overassistance is very low because PSV is optimized to obtain this targeted tidal volume. The absence of overassistance is confirmed in phase 2. Indeed, mean Eadi max is not different, despite a decrease in pressure support, and lower tidal volume does not lead to an increase in respiratory rate or FiO_2.

PEEP level is comparable between the two phases. The use of an external PEEP in PSV is used to offset iPEEP and ineffective efforts, and thus decrease work of breathing [23] or optimize lung recruitment. A setting of external PEEP to 80% of intrinsic PEEP is recommended but is difficult to obtain in PSV mode (Marini). Intrinsic PEEP is variable from one cycle to another and depends on each cycle intensity and tidal volume. According to Tassaux., *et al.* optimal PEEP level to reduce ineffective efforts in COPD patients lies between 5 and 10 cm H_2O [24]. Our study found a median PEEP of 6 cmH_2O in both groups with few ineffective efforts.

Optimal setting of expiratory cycling using Eadi signal to superimpose the machine cycle to the patient's neural cycle could better define the PEEP level required.

Settings made by practitioners of our service with the changes of different ventilation parameters may explain the lack of asynchronies in our study compared to the literature [1,14]. Despite the low number of asynchronies in both phases, they were statistically lower in phase 2 (10,1% *vs* 5,9%, $p < 0.05$). This improvement concerned micro-asynchronies. Macro-asynchronies were particularly in small numbers in both groups.

According to Thille., *et al.* [7], an asynchrony index greater than 10% is associated with poor clinical course, increased duration of mechanical ventilation and increased rate of tracheotomy for ventilator weaning. Recent studies have shown that bedside interpretation of flow, pressure and volume curves was reliable for the detection of macro asynchronies permitting better settings of ventilation parameters [15,25]. This settings optimization decreased the number of double triggering in a publication concerning 30 patients [26]. Our work is consistent with previous studies, demonstrating a decrease in auto-triggering, double triggering and ineffective efforts even in phase 1, despite a population at risk for difficult weaning [7,27]. However, the micro-asynchronies are not directly visible on standard curves and would require monitoring of respiratory central order, which is currently not possible in clinical practice. It is possible to approach this nervous central command using EAdi signal and thereby locate these micro-asynchronies. This analysis could also detect all asynchronies at the bedside with a lower risk of undervaluation [16]. In our study, the significant decrease in the number of asynchronies was related to a decrease in the level of assistance and a better expiratory cycling setting. This data is confirmed by Tassaux., *et al.* who assessed impact of the reduction in inspiratory time on asynchrony and inspiratory effort of COPD patients [24]. This study showed that

increasing the expiratory cycling up to 80% of the peak flow decreased the asynchrony and the work of breathing. These results are also in agreement with Beloncle., *et al.* study which showed that Eadi monitoring allowed a better optimization of the regulation of the expiratory trigger and decreased micro-asynchronies. This adjustment is difficult to the patient's bed on the only help of the pressure and flow curves of PSV [14]. In our study, a clinically relevant (>10%) asynchrony index (AI) was only observed in 14/31 cases in phase 1 and 9/31 cases, without any statistical significance, and so without consequence on the duration of the ventilation [15].

In our study, when considering only COPD patients, decreases in asynchronies were due to a decrease in inspiratory delays and ineffective efforts. It is interesting to note that despite the special attention of the practitioners of our service on the adjustment of expiratory cycling in PSV concerning difficult weaning of COPD patients, the NAVA mode allowed to decrease the inspiratory delays and non-triggering breaths. These asynchronies were the consequence of an increase in intrinsic positive end-expiratory pressure and inspiratory flow in obstructive disease, which seem better managed in NAVA mode [24].

Our study has some limitations. First, the absence of randomization could have introduced bias in the study and could have influenced the results. Indeed, the two periods were recorded immediately one after the other, and baseline settings were not the same for both sequences. Second sequence baseline is already adjusted after the first sequence. Clinical relevance of these findings remains to be demonstrated and must be the object of future works. It was not the purpose of this one.

In addition, we are not sure that the improvement of the PaO_2/FiO_2 ratio in the second phase was only due to the second phase optimization but could be due to the cumulative effect of the 2 settings. On other side, the improvement of the trigger delay could not be objective in the 2 phases of the PSV mode, as underlined Beloncle., *et al.* or Spahija., *et al.* studies [14,28]. Another significant risk is to induce an earlier expiration with the setting of premature expiratory cycling and provoke other asynchronies. Expiration occurring too early despite patients wanting to continue inspiration could create a double triggering [29].

A multicenter study would permit more inclusions and avoid the center effect. This physiologic study included 31 patients that may contribute to a lack of power but this is more than several studies on this subject [11,14]. Moreover, if the primary outcome remains the decrease in asynchronies, a selection of a subgroup of patients who have frequent asynchronies might be more appropriate

Conclusion

The analysis of Eadi signal coupled with the analysis of respirator curves (pressure, flow, volume) permit an optimization of ventilator setting resulting in a decrease of patient-ventilator asynchronies, especially micro-asynchronies (inspiratory delay and late cycling) in PSV mode. In our study, the decrease of micro-asynchronies is due to the decreased pressure support level and increased expiratory cycling. This study shows that despite the expertise of physicians to adjust the ventilation, an Eadi-based optimization improves patient-ventilator interactions. The reduction of asynchronies with a decrease in breathing effort could shorten the duration of weaning from ventilatory support. Further studies are necessary in order to determine the clinical impact of this improved synchrony.

Declarations

Ethics Approval

Yes (The study was approved by the Ethics Research Committee of our institution (No. 25- 0413, April 17, 2013).

Consent for Publication

All authors consent for publication.

Availability of Data and Material

All the data are saved in our institution. Original data could be shared upon reasonable request.

Financial Support

None.

Conflicts of Interest

None.

Contributions of the Authors

Conception and design: BG, LC, JMC, VM; Analysis and interpretation: BG, LC, MG, AR; Drafting the manuscript for important intellectual content: BG, OF, SR, FVB.

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