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Patient-ventilator asynchrony during assisted mechanical ventilation

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Abstract *Objective:* The incidence, pathophysiology, and consequences of patient-ventilator asynchrony are poorly known. We assessed the incidence of patient-ventilator asynchrony during assisted mechanical ventilation and we identified associated factors. *Methods:* Sixty-two consecutive patients requiring mechanical ventilation for more than 24 h were included prospectively as soon as they triggered all ventilator breaths: assist-control ventilation (ACV) in 11 and pressure-support ventilation (PSV) in 51. *Measurements:* Gross asynchrony detected visually on 30-min recordings of flow and airway pressure was quantified using an asynchrony index. *Results:* Fifteen patients (24%) had an asynchrony index greater than 10% of respiratory efforts. Ineffective triggering and double-triggering were the two main asynchrony patterns. Asynchrony existed during both ACV and PSV, with a median number of episodes per patient of 72 (range 13–215) vs. 16 (4–47) in 30 min, respectively ($p = 0.04$). Double-triggering was more common during ACV than during PSV, but no difference was found for ineffective triggering. Ineffective triggering was associated with a less sensitive inspiratory trigger, higher level of pressure support (15 cmH_2O, IQR 12–16, vs. 17.5, IQR 16–20), higher tidal volume, and higher pH. A high incidence of asynchrony was also associated with a longer duration of mechanical ventilation (7.5 days, IQR 3–20, vs. 25.5, IQR 9.5–42.5). *Conclusions:* One-fourth of patients exhibit a high incidence of asynchrony during assisted ventilation. Such a high incidence is associated with a prolonged duration of mechanical ventilation. Patients with frequent ineffective triggering may receive excessive levels of ventilatory support.

Keywords Mechanical ventilation · Patient-ventilator interaction · Ineffective triggering · Pressure-support ventilation

Introduction

An important objective of assisted or patient-triggered mechanical ventilation is to avoid ventilator-induced diaphragmatic dysfunction by allowing the patient to generate spontaneous efforts [1, 2]. A second objective is to reduce the patient's work of breathing by delivering a sufficient level of ventilatory support [3]. Finally, intuition suggests that a good match between patient respiratory efforts and ventilator breaths optimizes patient comfort and reduces work of breathing, although this point remains unverified [4]. Patient-ventilator asynchrony can be defined as a mismatch between the patient and ventilator inspiratory and expiratory times [4, 5, 6]. Although inspiratory and expiratory delays are almost inevitable with most ventilatory modes [7], several patterns of major asynchrony exist and can be easily detected by clinicians. Among these, ineffective triggering occurs when the patient's inspiratory effort fails to trigger a ventilator breath. Factors associated with ineffective triggering include a weak inspiratory effort and presence of intrinsic positive end-expiratory pressure (PEEPi), which increases the effort required to trigger the ventilator [6, 8]. Another easily detected asynchrony is double-triggering, which occurs when the patient's ventilatory demand is high and the ventilator inspiratory time too short [9]. Lastly, autotriggering is a cycle delivered by the ventilator in the absence of patient effort and can be generated by cardiogenic oscillations or leaks in the ventilator circuit [10].

The incidence of major patient-ventilator asynchrony during mechanical ventilation is unknown. To our knowledge, only a single study has addressed the prevalence of ineffective triggering during weaning from mechanical ventilation [11]. Chao et al. [11] found that more than 10% of patients admitted to a weaning center exhibited patient-ventilator asynchrony while receiving assisted mechanical ventilation. Furthermore, weaning was less often successful in the patients with asynchrony. Thus, wasted diaphragmatic energy expenditure may have a deleterious effect on weaning from mechanical ventilation. Identifying factors that increase the incidence of asynchrony may help optimize ventilator settings and minimize patient-ventilator mismatches.

The aims of our study were to prospectively and noninvasively evaluate the incidence of major asynchrony during assisted mechanical ventilation and to identify patient characteristics and ventilation parameters associated with a high incidence of asynchrony. The preliminary results of this present study were presented at the 2004 meeting of the European Society of Intensive Care Medicine [12].

Material and methods

Patients

Over a 21-week period, we treated 143 patients requiring invasive mechanical ventilation in our intensive care unit (ICU). Patients were included in the study as soon as they triggered all ventilator breaths during assist-control ventilation (ACV) or pressure-support ventilation (PSV). Exclusion criteria were shock or agitation at the time of possible study inclusion. There were 75 patients who either received ventilation for less than 24 h (*n* = 22) or were not able to trigger all ventilator breaths before death or transfer (*n* = 53). Recordings could not be obtained in 6 of the 68 remaining patients, leaving 62 patients for the study. The characteristics of these 62 patients are summarized in Table 1. The ventilatory mode was ACV in 11 patients (18%) and PSV in 51 patients (82%). In our ICU, patients are switched from ACV to PSV as soon as possible when they are awake and able to trigger the ventilator. Patients with severe illness, hypoxemia ($FIO_2 > 70\%$), or persistent coma are kept on ACV. The level of pressure support is set to obtain a tidal volume around 6–8 ml/kg and a respiratory rate less than 30 cycles/min. The standard cycling-off criterion is 25% of peak inspiratory flow. ACV is usually set at a tidal volume of 6–8 ml/kg delivered with a 60 l/min square inspiratory flow and with no inspiratory pause. As a result the inspiratory time is fairly short during ACV.

Table 1 Characteristics of the patients (*n* = 62) (*IQR* interquartile range, *SAPS* Simplified Acute Physiology Score, *SOFA* Sequential Organ Failure Assessment, *COPD* chronic obstructive pulmonary disease, *ACV* assist-control ventilation, *PSV* pressure-support ventilation)

Age (years; IQR)	70 (48–77)
Male sex	47 (76%)
SAPS II at admission, median (IQR)	59 (44–70)
SOFA at inclusion, median (IQR)	5 (3–9)
COPD	16 (26%)
Ramsay scale, median (IQR)	3 (2–5)
ACV	11 (18%)
PSV	51 (82%)
PaO_2/FIO_2, median (mmHg; IQR)	263 (194–320)
Duration of ventilatory support before study, median (days; IQR)	4.5 (3–7)
Duration of ventilatory support, median (days; IQR)	10 (5–27)
Tracheostomy	6 (10%)
Mortality	22 (35%)

Protocol

To minimize interference with patient-ventilator interactions, asynchrony was detected noninvasively using only flow and airway pressure signals. Airway pressure was measured at the distal end of the circuit using a differential pressure transducer (Validyne MP45, 100 cmH_2O, Northridge, Calif., USA). Flow was recorded using a Fleisch no. 2 pneumotachograph (Fleisch, Lausanne, Switzerland). Patients were ventilated with various ventilators: Evita 2 and Evita 4 (Dräger, Germany); Puritan Bennett 840 (Tyco, USA); Vela, T. Bird, and Bird 8400 (Viasys, USA). The signals were recorded continuously over a 30-min period after endotracheal suctioning. The ventilatory mode and all ventilatory settings were previously adjusted by the clinician before the recordings except the back-up rate set at the minimal value in patients ventilated in ACV (3–5 breaths/min). Investigators were not involved in setting the ventilator. Our institutional review board approved the study. The requirement for informed consent was waived because of the observational nature of the study.

Patterns of major patient-ventilator asynchrony

Asynchrony was detected by visual inspection of the recordings. Thus we investigated patterns of major asyn-

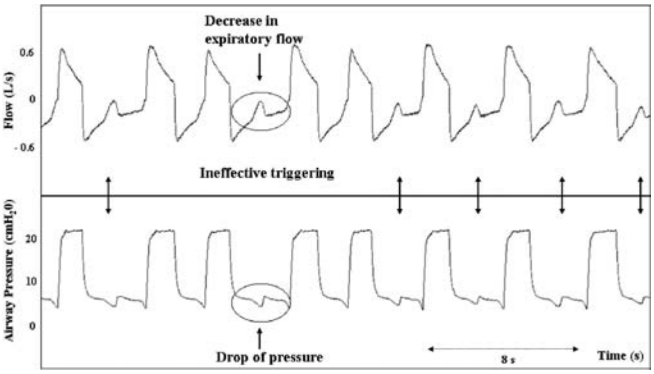

Fig. 1 Flow and airway pressure tracings showing ineffective triggering, i.e., a wasted effort, defined as an airway pressure drop simultaneous to a flow decrease during the expiratory period and not followed by a ventilator cycle, indicating that the patient's effort was not detected by the ventilator (*arrows*)

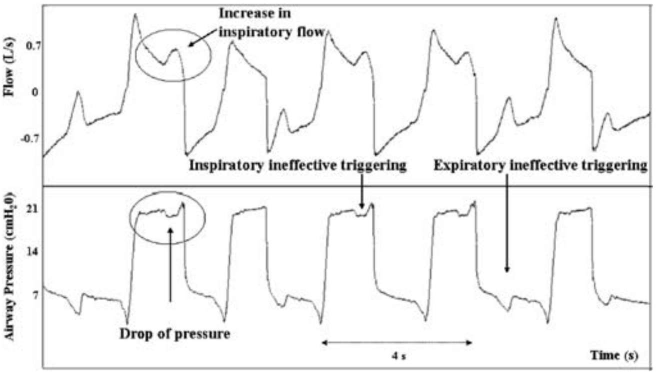

Fig. 2 Flow and airway pressure recordings showing ineffective efforts occurring both during the expiratory phase and during the inspiratory phase

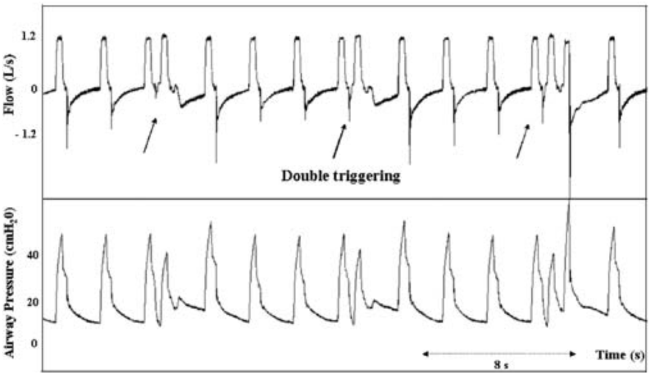

Fig. 3 Flow and airway pressure recordings showing double-triggering, defined as two consecutive ventilator cycles separated by an expiratory time less than one-half the mean inspiratory time. Double-triggering occurs when the ventilator inspiratory time is shorter than the patient's inspiratory time. The patient's effort is not completed at the end of the first ventilator cycle and triggers a second ventilator cycle

chrony that are easily detected by clinicians. Ineffective triggering was defined during both ACV and PSV as an abrupt airway pressure drop ($\geq 0.5\,\mathrm{cmH_2O}$) simultaneous to a flow decrease (in absolute value) and not followed by an assisted cycle during the expiratory period (Fig. 1). In PSV only, ineffective triggering could also happen during the inspiratory period but related to a flow increase (Fig. 2). Double-triggering was defined as two cycles separated by a very short expiratory time, defined as less than one-half of the mean inspiratory time, the first cycle being patient-triggered (Fig. 3). Autotriggering was defined as a cycle delivered by the ventilator without a prior airway pressure decrease, indicating that the ventilator delivered a breath that was not triggered by the patient. A short cycle was defined as an inspiratory time less than one-half the mean inspiratory time. A prolonged cycle was defined as an inspiratory time greater than twice the mean inspiratory time. The inspiratory time was defined as the time during which flow was positive, and mean inspiratory time was calculated over 30 cycles.

Asynchrony detection was based on flow and airway pressure signals only. We evaluated the validity of this approach using two methods. First, we assessed accuracy and reliability by examining recordings from another study including not only airway pressure and flow but also esophageal pressure signals [13]. Based on these recordings, we conducted a blind comparison of the number of asynchrony events detected using esophageal pressure signals and the number estimated from flow and airway pressure signals only. Second, we assessed reproducibility by having two investigators each perform two blinded analyses of all the study recordings.

Counting asynchrony events

We used an asynchrony index [11], defined as the number of asynchrony events divided by the total respiratory rate computed as the sum of the number of ventilator cycles (triggered or not) and of wasted efforts: asynchrony Index (expressed in percentage) = number of asynchrony events/total respiratory rate (ventilator cycles +wasted efforts) × 100. We defined a high incidence of asynchrony as an asynchrony index greater than 10%, based on a study by Vitacca et al. [14] evaluating the effect of several ventilator settings on the percentage of ineffective effort. The study showed that the mean value plus the standard deviation of the percentage of ineffective efforts did not exceed 10% when pressure support was removed.

Statistical analysis

Continuous data are expressed as medians and interquartile range (IQR) unless stated otherwise. Qualitative data were compared using the χ^2 test and Fisher's exact test and quantitative data using the Mann-Whitney *U* test. All *p* values at or below 0.05 were considered significant. To evaluate the validity of the method we used Spearman's correlation coefficient and the κ test. The statistical analysis was performed using the statistical software package SPSS 13.0 (SPSS, Chicago, Ill., USA).

Results

Validity of the method used to detect asynchrony

Based on 11 recordings (including four with an asynchrony index ≥ 10%), the number of asynchrony events detected using esophageal pressure signals was correlated closely with the number detected using flow and airway pressure signals only ($n = 11$, $\rho = 0.99$, $p < 0.01$). Based on the recordings of the present study using flow and airway pressure signals only, the numbers of asynchrony events detected by the two observers were closely correlated ($n = 62$, $\rho = 0.94$, $p < 0.01$), indicating good reproducibility. The κ test comparing the ability of the two observers to detect patients with asynchrony index values greater than 10% showed very high agreement, with a κ value of 0.96 ($p < 0.01$). These data support the reliability of noninvasive asynchrony detection based on flow and airway pressure signals, as suggested previously [15].

Table 2 Asynchrony according to ventilatory mode. Mean ± standard deviation number of asynchrony per patient and per minute for all patients. Comparison between patients ventilated in assist-control ventilation (*ACV*) versus pressure-support ventilation (*PSV*)

	ACV ($n = 11$)	PSV ($n = 51$)	*p*
Asynchronies	4.3 ± 4.8	1.9 ± 3.8	0.04
Ineffective triggering	3.0 ± 4.9	1.8 ± 3.7	0.38
Double-triggering	1.2 ± 2.3	0.1 ± 0.4	0.01

Study population

Median inspiratory time was shorter during ACV than during PSV [0.60 s (IQR 0.55–0.78) vs. 0.90 s (0.70–1.10); $p < 0.01$]. Compared to patients receiving PSV, those receiving ACV had a higher respiratory rate [(32 breaths/min (29–37) vs. 23 (17–30); $p < 0.01$], higher Ramsay score [6 (3.5–6) vs. 3 (2–4); $p < 0.01$], longer duration of mechanical ventilation [20 days (14–33) vs. 8 (4–24); $p = 0.03$) and higher mortality rate (91% vs. 24%; $p < 0.01$).

Table 3 Factors associated with a high prevalence of ineffective and double triggering (*IQR* interquartile range, *SAPS* Simplified Acute Physiology Score, *SOFA* Sequential Organ Failure Assessment, *COPD* chronic obstructive pulmonary disease, *ACV* assist-control ventilation)

	Ineffective triggering		Double triggering	
	Index < 10% ($n = 51$)	Index ≥ 10% ($n = 11$)	< 1/min ($n = 57$)	≥ 1/min ($n = 5$)
Age (years; IQR)	70 (48–75)	72 (67–79)	70 (48–77)	69 (48–71)
Male sex	36 (71%)	11 (100%)*	42 (74%)	5 (100%)
COPD	10 (20%)	6 (55%)*	16 (29%)	0
SAPS II at admission (IQR)	59 (42–68)	60 (51–75)	57 (43–67)	74 (53–88)
SOFA at inclusion (IQR)	5 (3–9)	5 (3–7)	5 (3–8)	9 (5–9)
Use of corticosteroids and/or paralytics agents	17 (33%)	2 (18%)	16 (28%)	3 (60%)
Ramsay scale (IQR)	3 (2–5)	3 (2–4)	3 (2–4)	6 (3–6)**
pH (IQR)	7.44 (7.39–7.49)	7.49 (7.45–7.51)*	7.45 (7.40–7.50)	7.45 (7.33–7.48)
Bicarbonates (mmol/l; IQR)	24 (21–30)	28 (23–35)*	24 (22–30)	28 (22–30)
$PaCO_2$ (mmHg; IQR)	36 (32–42)	41 (31–49)	36 (32–43)	40 (39–42)
PaO_2/FIO_2 (mmHg; IQR)	260 (192–316)	285 (194–395)	272 (201–320)	132 (131–187)**
Ventilatory mode: ACV	9 (18%)	2 (18%)	7 (12%)	4 (80%)**
VT (ml; IQR)	500 (400–550)	650 (500–700)*	500 (450–600)	450 (430–500)
Respiratory rate ventilator (breaths/min; IQR)	25 (20–30)	18 (11–25)*	22 (17–30)	30 (28–30)
Respiratory rate patient (breaths/min; IQR)	25 (20–30)	31 (13–35)	24 (20–33)	30 (28–30)
Inspiratory time (s; IQR)	0.85 (0.65–1.10)	1 (0.75–1.10)	0.90 (0.70–1.10)	0.60 (0.55–0.60)**
Trigger (l/min; IQR)	1.0 (1.0–1.0)	1.5 (1.0–3.0)*	1.0 (1.0–1.5)	2.0 (1.0–3.0)
Peak inspiratory pressure (cmH_2O; IQR)	20 (17–25)	25 (23–27)*	20 (18–24)	28 (26–37)**
PEEP (cmH_2O; IQR)	5 (4–6)	6 (5–7)	5 (4–6)	10 (8–10)**
Pressure support (cmH_2O; IQR)	15 (12–16)	18 (16–20)*	15 (13–16)	16 (16–16)

* $p < 0.05$ high vs. low prevalence of ineffective triggering (Mann Whitney test)
** $p < 0.05$ high vs. low prevalence of double triggering (Mann Whitney test)

Table 4 Comparison of the outcome between patients with and without a high prevalence of asynchronies (*IQR* interquartile range)

	Asynchrony index < 10% (*n* = 47)	Asynchrony index ≥ 10% (*n* = 15)	*p*
Duration of mechanical ventilation (days; IQR)	7 (3–20)	25 (9–42)	0.005
Duration of mechanical ventilation ≥ 7 days	23 (49%)	13 (87%)	0.01
Tracheostomy	2 (4%)	5 (33%)	0.007
Mortality	15 (32%)	7 (47%)	0.36

Prevalence of asynchrony

The median asynchrony index was 2.1% (IQR 0.7–8.6). Fifteen patients (24%) had an asynchrony index of 10% or greater, with a median of 26% (18–37). Ineffective triggering and double-triggering contributed more than 98% of the total number of asynchrony events (85% were ineffective triggering events, and 13% were double-triggering events). Among ineffective triggering events 78% occurred during the expiratory period and 7% during the inspiratory period. Autotriggering, short cycle, and prolonged cycle each contributed less than 1% of the asynchrony events. Asynchrony events were detected during both ACV and PSV. Double-triggering was more common during ACV than during PSV, but ineffective triggering was similar with the two modes (Table 2).

Factors associated with a high incidence of asynchrony

Among the 15 patients with a high incidence of asynchrony (asynchrony index ≥ 10%) 11 exhibited a high incidence of ineffective triggering (≥ 10% of respiratory efforts) and 4 a high incidence of double-triggering (> 1 per min). One patient with an asynchrony index less than 10% exhibited a high incidence of double-triggering (Table 3). Factors associated with a high incidence of double-triggering were a low PaO_2/FIO_2 ratio, ACV as the ventilatory mode, a shorter inspiratory time, a high maximal inspiratory pressure, and a high level of PEEP. Patient factors associated with a high incidence of ineffective triggering were male sex, presence of chronic obstructive pulmonary disease (COPD), elevated bicarbonates, and alkalosis. Severity of illness as assessed using the Simplified Acute Physiology Score II at admission [16] and Sequential Organ Failure Assessment score at inclusion [17] was similar in the two groups, with or without a high incidence of ineffective triggering. Ventilatory parameters associated with a high incidence of ineffective triggering were a poorly sensitive trigger, high tidal volume, high peak inspiratory pressure, and high level of pressure support (Fig. 4). True patient respiratory rates were similar in the groups with and without a high incidence of ineffective triggering, but the ventilator respiratory rate was lower in the ineffective triggering group.

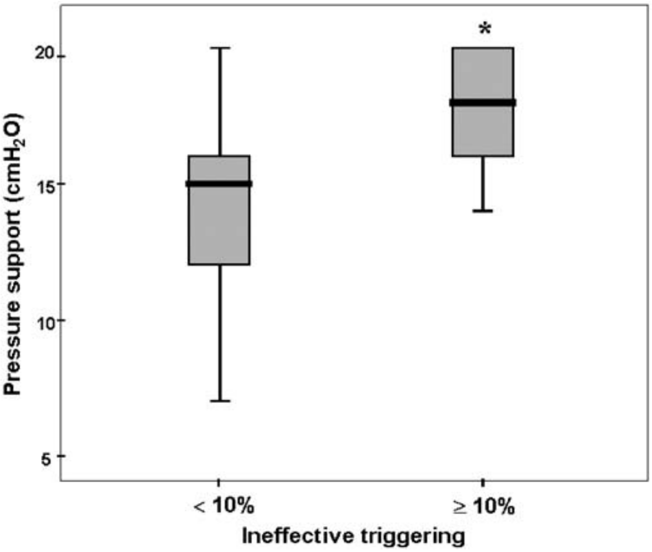

Fig. 4 Level of pressure support and frequency of ineffective triggering. *Box plots* show median, interquartile range (25–75th percentiles), and outliers (5–95th percentiles) of pressure support in patients with and without a high prevalence of ineffective triggering (> 10%). Pressure support was higher in patients with a high incidence of ineffective triggering. $*p < 0.05$ (Mann-Whitney test)

Outcome

Patients whose asynchrony index was greater than 10% had a longer duration of mechanical ventilation that did the other patients (Table 4). In addition, mechanical ventilation for more than 7 days and tracheostomy were more common in the group with a high incidence of asynchrony. Mortality was similar in the two groups. Among the 51 patients ventilated in PSV mode, those with a high incidence of asynchrony had a longer median duration of mechanical ventilation than the other patients [17 days (8–40) vs. 7 (3–20); $p = 0.04$].

Discussion

In our study approximately one-fourth of patients ventilated for more than 24 h and able to trigger the ventilator had a high incidence of asynchrony during assisted mechanical ventilation. Ineffective triggering and double-triggering were the two main patterns of asynchrony. A high pressure support level, high tidal volume, and alkalosis were associated with ineffective triggering. Patients with a high incidence of double-triggering had deeper hypoxemia with higher values of peak inspiratory pressure and PEEP, suggesting greater severity of lung injury. The duration of mechanical ventilation was longer and the risk of tracheostomy higher when the incidence of asynchrony was greater than 10%.

Ineffective triggering

Our results suggest that patients with frequent ineffective efforts may receive an excess of pressure support. Pressure support and tidal volume delivered by the ventilator were higher in this group, and arterial pH and bicarbonate levels were also higher. Ineffective triggering is associated with auto-PEEP resulting from a large tidal volume and the continuation of the mechanical inspiration into the neural expiration [5, 6, 7]. The frequency of ineffective triggering increases with the pressure support level, most notably in patients with COPD because high pressure levels generate dynamic hyperinflation ascribable to the larger tidal volume and shorter expiratory time [5, 8, 11, 14, 18, 19]. External PEEP has been shown to decrease ineffective triggering in patients with high auto-PEEP [18] by reducing the work of breathing needed to trigger the ventilator [20, 21, 22]. However, Chao et al. [11] found that the most effective method for eliminating asynchrony consisted in reducing the level of ventilator support and noted that application of external PEEP reduced but did not eliminate ineffective triggering.

Several studies have found that most patients with mismatching to the ventilator have COPD, although ineffective triggering also occurred in patients without this disease [11, 23]. Similarly, we found that presence of COPD was associated with ineffective triggering. A high frequency of ineffective triggering was also associated with alkaline pH and bicarbonate elevation. Mechanical ventilation for acute COPD exacerbation may produce alkalemia via excessive PCO_2 reduction in patients with chronic bicarbonate elevation. Alkalemia in this situation is related both to an excess in ventilatory support and to a high baseline bicarbonate level. This high bicarbonate level may reflect a higher chronic PCO_2 level or an associated cause of metabolic alkalosis such as diuretic therapy. Alkalemia can depress the respiratory drive and increase the incidence of ineffective triggering [24].

A less sensitive trigger threshold was associated with a higher incidence of ineffective triggering (flow triggering only was used). Flow triggering is known to be associated with a low triggering effort [25], but this effort increases with the inspiratory trigger threshold.

Double-triggering

Five patients had a high incidence of double-triggering, defined as more than one double-triggering event per minute. Double-triggering occurs when the patient's ventilatory demand is high, and the inspiratory time set on the ventilator is too short [9]. Double-triggering was associated with a short inspiratory time and with ACV. The PaO_2/FIO_2 ratio was lower and peak inspiratory pressure was higher than in patients without this asynchrony, suggesting that double-triggering is associated with greater severity of lung injury and probably with a greater drive to breathe as indicated by a higher respiratory rate. The lower number of double-triggering events during PSV than during ACV may be due to the partial dependency of inspiratory time on the patient's ventilatory demand whereas it is preset on the ventilator during ACV. In addition, the inspiratory time during PSV tends to be longer than the patient's neural inspiratory time [7]. The shorter inspiratory time during ACV may explain the higher frequency of double-triggering than with PSV.

Outcome and duration of mechanical ventilation

Although the duration of mechanical ventilation at the time of recording was similar, patients with a high incidence of asynchrony required a longer duration of mechanical ventilation than those with a low incidence. This may indicate greater disease severity, but no difference existed in terms of illness severity score, or inappropriate ventilator settings in the patients with frequent asynchrony. Nava et al. [18] found that a lower level of pressure support was not associated with an increase in diaphragmatic energy expenditure because wasted efforts were less common. Optimization of pressure support and PEEP level reduces the frequency of wasted efforts and may improve the quality of sleep in chronically ventilated patients [26]. Wasting of energy expenditure by the diaphragm may have a deleterious effect on weaning from mechanical ventilation by promoting an injurious diaphragmatic pattern, decreasing patient comfort, or leading to errors in the assessment of readiness for weaning.

Limitations of the study

We used a noninvasive method based on flow and airway pressure readings, which could not accurately define the start and end of patient inspiration. Although esophageal pressure or diaphragm electrical activity would have ensured greater accuracy in detecting asynchrony, these methods are invasive, and insertion of an esophageal catheter may alter patient-ventilator interactions. Moreover, indirect estimates of the onset and duration of neural inspiratory time based on esophageal pressure and flow also lead to errors, compared to neural inspiratory time measurement using diaphragmatic electromyography [27]. We focused only on gross asynchronies easily identified without an invasive method. Consequently, one limit of our study is to miss other asynchronies such as delayed triggering or delayed cycling [6, 7]. Although delayed cycling can sometimes be suggested by an abrupt increase in airway pressure signal pressure during the final part of ventilator pressurization generated by an active expiratory effort, the same phenomenon can also be caused by the relaxation of inspiratory muscles [28]. Our method did not allow this expiratory asynchrony to be detected, which, however, can be an important problem especially in obstructive patients.

Simultaneous recordings of esophageal pressure obtained in another study were used to assess the validity of our method for detecting asynchrony based on flow and airway pressure. We found a good correlation between ineffective triggering detected from flow/airway pressure and from esophageal pressure signals. Giannouli et al. [15] compared estimates of ineffective efforts using the two techniques and also reported excellent agreement. Furthermore, reproducibility was evaluated by having a second observer examine the recordings. The results showed a close correlation between the numbers of asynchrony events detected by the two observers.

The incidence of asynchrony is a reflection of any center's practice and expertise of mechanical ventilation; therefore the figures reported here might be applicable to that center only. Another limitation is that various types of ventilators were used in the study patients. Ventilator performance may affect the occurrence of asynchrony, as the trigger function and pressurization process differ across ventilators [29]. However, no difference was found concerning the incidence of asynchrony between turbine and classical servo valve ventilators.

Conclusion

Patient-ventilator asynchrony is common during assisted mechanical ventilation. Ineffective triggering and double-triggering are the two main patterns of asynchrony. Asynchrony may have deleterious effects related to increased energy expenditure, abnormal diaphragmatic pattern or problems in identifying readiness to wean. In our study patients with high rates of asynchrony also had longer times on mechanical ventilation. Whether optimization of ventilatory settings would shorten the duration of mechanical ventilation by reducing the occurrence of asynchrony cannot be determined from the present study and should be the subject of future investigations.

References

1. Vassilakopoulos T, Petrof BJ (2004) Ventilator-induced diaphragmatic dysfunction. Am J Respir Crit Care Med 169:336–341
2. Sassoon CS, Zhu E, Caiozzo VJ (2004) Assist-control mechanical ventilation attenuates ventilator-induced diaphragmatic dysfunction. Am J Respir Crit Care Med 170:626–632
3. Brochard L, Harf A, Lorino H, Lemaire F (1989) Inspiratory pressure support prevents diaphragmatic fatigue during weaning from mechanical ventilation. Am Rev Respir Dis 139:513–521
4. Sassoon CS, Foster GT (2001) Patient-ventilator asynchrony. Curr Opin Crit Care 7:28–33
5. Tobin MJ, Jubran A, Laghi F (2001) Patient-ventilator interaction. Am J Respir Crit Care Med 163:1059–1063
6. Parthasarathy S, Jubran A, Tobin MJ (1998) Cycling of inspiratory and expiratory muscle groups with the ventilator in airflow limitation. Am J Respir Crit Care Med 158:1471–1478
7. Beck J, Gottfried SB, Navalesi P, Skrobik Y, Comtois N, Rossini M, Sinderby C (2001) Electrical activity of the diaphragm during pressure support ventilation in acute respiratory failure. Am J Respir Crit Care Med 164:419–424
8. Leung P, Jubran A, Tobin MJ (1997) Comparison of assisted ventilator modes on triggering, patient effort, and dyspnea. Am J Respir Crit Care Med 155:1940–1948
9. Tokioka H, Tanaka T, Ishizu T, Fukushima T, Iwaki T, Nakamura Y, Kosogabe Y (2001) The effect of breath termination criterion on breathing patterns and the work of breathing during pressure support ventilation. Anesth Analg 92:161–165
10. Imanaka H, Nishimura M, Takeuchi M, Kimball WR, Yahagi N, Kumon K (2000) Autotriggering caused by cardiogenic oscillation during flow-triggered mechanical ventilation. Crit Care Med 28:402–407
11. Chao DC, Scheinhorn DJ, Stearn-Hassenpflug M (1997) Patient-ventilator trigger asynchrony in prolonged mechanical ventilation. Chest 112:1592–1599
12. Thille A, Lellouche F, Brochard L (2004) Patient-ventilator asynchrony during invasive mechanical ventilation: prevalence and risk factors. Intensive Care Med 30:A263
13. Deye N, Lellouche F, Maggiore S, Taille S, Lefrançàis F, Pignataro C, Fartoukh M, Harf A, Brochard L (2002) Influence of body posture on the effort to breathe in mechanically ventilated patients. Am J Respir Crit Care Med 165:A788
14. Vitacca M, Bianchi L, Zanotti E, Vianello A, Barbano L, Porta R, Clini E (2004) Assessment of physiologic variables and subjective comfort under different levels of pressure support ventilation. Chest 126:851–859

15. Giannouli E, Webster K, Roberts D, Younes M (1999) Response of ventilator-dependent patients to different levels of pressure support and proportional assist. Am J Respir Crit Care Med 159:1716–1725
16. Le Gall JR, Lemeshow S, Saulnier F (1993) A new Simplified Acute Physiology Score (SAPS II) based on a European/North American multicenter study. JAMA 270:2957–2963
17. Vincent JL, Moreno R, Takala J, Willatts S, De Mendonca A, Bruining H, Reinhart CK, Suter PM, Thijs LG (1996) The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. On behalf of the Working Group on Sepsis-Related Problems of the European Society of Intensive Care Medicine. Intensive Care Med 22:707–710
18. Nava S, Bruschi C, Rubini F, Palo A, Iotti G, Braschi A (1995) Respiratory response and inspiratory effort during pressure support ventilation in COPD patients. Intensive Care Med 21:871–879
19. Fabry B, Guttmann J, Eberhard L, Bauer T, Haberthur C, Wolff G (1995) An analysis of desynchronization between the spontaneously breathing patient and ventilator during inspiratory pressure support. Chest 107:1387–1394
20. Smith TC, Marini JJ (1988) Impact of PEEP on lung mechanics and work of breathing in severe airflow obstruction. J Appl Physiol 65:1488–1499
21. Mancebo J, Albaladejo P, Touchard D, Bak E, Subirana M, Lemaire F, Harf A, Brochard L (2000) Airway occlusion pressure to titrate positive end-expiratory pressure in patients with dynamic hyperinflation. Anesthesiology 93:81–90
22. MacIntyre NR, Cheng KC, McConnell R (1997) Applied PEEP during pressure support reduces the inspiratory threshold load of intrinsic PEEP. Chest 111:188–193
23. Nava S, Bruschi C, Fracchia C, Braschi A, Rubini F (1997) Patient-ventilator interaction and inspiratory effort during pressure support ventilation in patients with different pathologies. Eur Respir J 10:177–183
24. Javaheri S, Kazemi H (1987) Metabolic alkalosis and hypoventilation in humans. Am Rev Respir Dis 136:1011–1016
25. Aslanian P, El Atrous S, Isabey D, Valente E, Corsi D, Harf A, Lemaire F, Brochard L (1998) Effects of flow triggering on breathing effort during partial ventilatory support. Am J Respir Crit Care Med 157:135–143
26. Fanfulla F, Delmastro M, Berardinelli A, Lupo ND, Nava S (2005) Effects of different ventilator settings on sleep and inspiratory effort in patients with neuromuscular disease. Am J Respir Crit Care Med 172:619–624
27. Parthasarathy S, Jubran A, Tobin MJ (2000) Assessment of neural inspiratory time in ventilator-supported patients. Am J Respir Crit Care Med 162:546–552
28. Georgopoulos D, Prinianakis G, Kondili E (2006) Bedside waveforms interpretation as a tool to identify patient-ventilator asynchronies. Intensive Care Med 32:34–47
29. Richard JC, Carlucci A, Breton L, Langlais N, Jaber S, Maggiore S, Fougere S, Harf A, Brochard L (2002) Bench testing of pressure support ventilation with three different generations of ventilators. Intensive Care Med 28:1049–1057