

The effect of triggering type on post-triggering pressure variations during pressure support ventilation: a simplified surrogate for dyssynchrony

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Context Several studies comparing flow and pressure triggering using invasive and noninvasive techniques have mostly focused on the trigger phase and favored flow triggering. Recently, there have been advancements in the technology of pressure triggering to improve its performance.

Aims We sought to evaluate the effect of triggering type in old and new ventilators on patient's synchrony in the post-trigger phase using variations in airway pressures with the set inspiratory pressure as a surrogate for dyssynchrony.

Patients and methods Using three different ventilator types, 32 patients on pressure support ventilation were set on the two triggering types (at the same equivalent levels), each for 1 h, with all other ventilatory setting kept constant. At the end of the hour on each trigger mode, the measured peak pressure and its difference with the set inspiratory pressure [delta pressure (ΔP)], the mean airway pressure, and different ventilatory parameters and arterial blood gases were assessed.

Results Pressure triggering resulted in a significantly higher peak pressure, ΔP , and lower dynamic compliance at any equivalent sensitivity and pressure support regardless of the level (<0.05). Moreover, at higher sensitivity levels (3 cmH₂O and l/min), flow triggering produced higher mean airway

pressures and oxygenation (<0.05). However, there was no significant difference as regards tidal volume, minute volume, frequency, rapid shallow breathing index, or PCO₂.

Conclusion Despite advances in pressure-triggering technology, flow triggering results in less pressure variation and better patient's synchrony during pressure support ventilation; in this respect, ΔP and dynamic compliance are simple noninvasive measures for dyssynchrony.

Egypt J Bronchol 2018 12:41–48

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Egyptian Journal of Bronchology 2018 12:41–48

Keywords: dynamic compliance, flow triggering, patient–ventilator synchrony, post-trigger pressure variation, pressure support ventilation, pressure triggering

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Received 26 January 2017 Accepted 7 February 2017

Introduction

Patient–ventilator dyssynchrony may impose a significant burden on the respiratory system [1,2]. Trigger dyssynchrony per se may present as autotriggering, excessive triggering delay, ineffective efforts, and double triggering [3–5]. A recent advanced monitoring/alarming system could continuously disclose the degree of dyssynchrony, in which dyssynchrony has been found to be associated with increased morbidity and mortality in critically ill patients [6–9]. Many studies have previously compared pressure triggering and flow triggering [10–14]. Although most results are in favor of flow triggering, the improved technology in the new ventilators has overcome some of the differences between pressure and flow triggering [10].

We sought to compare the effects of pressure-triggered and flow-triggered pressure support ventilation (PSV) on work of breathing in old and new ventilator technology, and to study the effect of triggering type/level on the post-trigger phase dyssynchrony in patients on PSV.

Patients and methods

Study design and setting

This is a multicenter, interventional, post-test, within-subjects design performed at the 'Critical Care Department, Cairo University', 'Critical Care Unit, New Kasr El Aini Teaching hospital', and 'Intensive Care Unit, Al-Agoza hospital' during the time period between February 2009 and March 2013.

Patients

All consecutive adult (>18 years of age) patients ventilated due to various etiologies (multiple trauma, pneumonia, pancreatitis, etc.) and on PSV were included in the study. Informed consent was obtained from all patients or next-of-kin, and the protocol was approved by the local review board. Patients who were in postarrest status and those with cerebrovascular

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accidents involving the brain stem, as well as chronic obstructive pulmonary disease with air trapping, were excluded from the study.

Intervention

Preparation and baseline evaluation

To omit confounding variables, all examined patients were in a semirecumbent position without giving sedative agents during the study period. A standard set of corrugated ventilatory circuit tubing was used, together with a heat-moisture exchanger, when the cascade Humidifier was not in use. Water condensate was evacuated from the circuit before the study period. The system was carefully checked to avoid gas leaks. Patients were put on one old ventilator technology, Puritan Bennett 7200 series (Nellcor Puritan Bennett, Pleasanton, California, USA), and two new ventilator technologies, 840 series (Nellcor Puritan Bennett) and Galileo Gold (Hamilton Medical, Rhäzüns, Switzerland). Standard fluid administration and medication were continued throughout the study period.

All patients were subjected to standard laboratory evaluation, including arterial blood gases (ABGs), through an indwelling arterial line to ensure accuracy of samples. Hemodynamic parameters (arterial blood pressure, heart rate, and central venous pressure) were monitored throughout the time of the study period. Acute Physiology And Chronic Health Evaluation score was calculated on admission and before their inclusion into the study.

Protocol

The ventilator for each patient was set on both flow-triggered and pressure-triggered systems at the same ventilatory support levels. The trigger level was set for pressure triggering between 0.5 and 3 cmH₂O in different patients. As regards flow triggering, the baseline gas flow directed through the ventilatory circuit was 10 l/min automatically or manually adjusted, and flow sensitivity was set at the same pressure-triggering level for each patient.

Each triggering system (flow triggering followed by pressure triggering) was applied for 60 min, while keeping FiO₂ (0.5), continuous positive airway pressure (CPAP) (at 5 cmH₂O), inspiratory rise time, and expiratory trigger sensitivity (default value for each ventilator) levels constant throughout the study period.

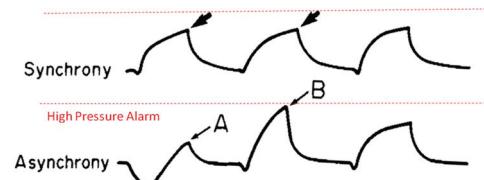
Study outcome

In the last 5 min of the 60-min period, the following variables were measured or calculated: (i) ventilatory parameters [respiratory frequency (f), tidal volume (V_t),

total minute ventilation (V_e), rapid shallow breathing index (RSBI), inspiratory and expiratory times (T_i and T_e , respectively), duration of respiratory muscle contraction expressed as the ratio of inspiratory time over total respiratory cycle time (T_i/T_{tot})]; (ii) airway pressures [peak pressure (P_{pk}) and mean airway pressure (P_{mean})]; and (iii) derived parameters, which included the following:

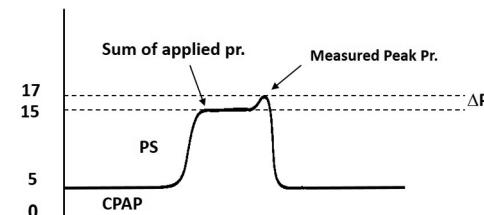
- (1) Dynamic compliance (C_{dyn}): $V_t/\text{driving pressure (PS)}$.
- (2) Delta pressure (ΔP): Pressure difference between measured P_{pk} and set total inspiratory pressure (CPAP+PS level) [$\Delta P = \text{measured } P_{pk} - (\text{CPAP+PS level})$]. We hypothesized that as both measurements should be similar, any additional elevation of the measured peak pressure is indicative of patient's active effort to the pressure applied (e.g. straining). We therefore used this measure as an indicator of patient-ventilator dyssynchrony (Figs 1 and 2).
- (3) Pressure-time index (PTI) ($=P_{mean} \times T_i/T_{tot}$).
- (4) Ventilatory efficiency ($=P_{mean} \times V_t/P_{pk}$): As the mean airway pressure is the main indicator for oxygenation and the inspired V_t for a given pressure, it is an indicator for ease of ventilation

Figure 1



Variable peak pressure during pressure support. Patient-triggered breaths on pressure support; in the upper panel, there are no variations in the pressure limit. In the lower panel, there is variation in the pressure limit in breath A as compared with breath B, which is an indicator of dyssynchrony

Figure 2



Difference between measured peak pressure and sum of the applied pressures (set pressure) in pressure support ventilation mode (ΔP). Illustration of a single pressure-supported breath, showing the difference between the set peak pressure (sum of CPAP and PS) and the measured peak pressure which is seen higher than the set pressure. This difference (ΔP) is taken as a surrogate of dyssynchrony (e.g. patients straining). CPAP, continuous positive airway pressure; Pr., pressure; PS, pressure support

per breath, and hence we used their product as an indicator for efficient use of the applied pressure.

All previous parameters were recorded from the ventilator at the completion of each trial in every patient by taking an average of three readings for each parameter.

Statistical methods

The Statistical Package for Social Sciences (SPSS), version 15 (SPSS Inc., Chicago, IL, USA) was used for computerized data entry and analysis. Data were presented as mean \pm SD, or as frequency and percentage as appropriate. For comparison of the means of two groups, the *t*-test for independent variables was used. The χ^2 -test was used. A *P* value less than 0.05 was considered as statistically significant.

Results

Our study was carried out on 32 patients recovering from acute renal failure due to variable etiologies who were maintained on PSV. Patient's demographics are shown in Table 1.

Demographic data of the studied participants and ventilatory settings

Table 1 shows ventilatory settings before the study; however, upon the start of the study, as explained in the methods, both the FiO_2 and the CPAP levels were kept constant to eliminate their effects on the dyssynchrony (at 0.5 and 5 cmH₂O, respectively).

Comparison of patients on the two triggering types

Ventilatory parameters and lung mechanics

As evident from Table 2, with regard to the spontaneous ventilatory parameters, we could not find a statistically

Table 1 Clinical parameters and ventilatory settings of the study group

	Mean \pm SD (range)
Sex (male) [n (%)]	20 (62)
Age	59.5 \pm 15.08 (23–84)
APACHE-II (adm.)	27.75 \pm 10.38 (10–46)
APACHE-II (during study)	15.25 \pm 6.92 (3–29)
Days of MV [median (range)]	4.5 (1–31)
Settings	
FiO_2	0.455 \pm 0.1 (0.3–0.6)
CPAP	5.46 \pm 2.21 (3–13)
PS level	16.82 \pm 3.3 (12–25)
TIP	22.39 \pm 3.3 (17–27)
Sensitivity	2.14 \pm 0.88 (0.5–3)

APACHE, Acute Physiology And Chronic Health Evaluation; adm., admission; CPAP, continuous positive airway pressure; MV, mandatory ventilation; PS, pressure support; TIP, total inspiratory pressure.

significant difference between patients on flow triggering and those on pressure triggering at different levels.

As shown in Table 3, there was a statistically significantly higher P_{pk} and ΔP and statistically significantly lower P_{mean} with pressure triggering in relation to flow triggering. However, no statistical difference was found between the two types of triggering with regard to the cycle times (T_i , T_e , or T_i/T_{tot}). Moreover, with flow-triggering type, there was statistically significantly higher C_{dyn} , PTI, and ventilatory efficiency compared with pressure triggering type.

Arterial blood gases

As regards the ABG, with flow triggering there was a statistically significant increase in PaO_2 (120.65 \pm 46.57 vs. 107.64 \pm 34.51, *P*=0.002) compared with pressure triggering. However, there was no statistically significant difference in SaO_2 or PaCO_2 (97.87 \pm 2.51 vs. 97.45 \pm 2.48, *P*=0.057 and 36.01 \pm 9.34 vs. 35.87 \pm 9.11, *P*=0.78) for flow triggering versus pressure triggering, respectively.

We did not record any sign of respiratory distress/fatigue in the patients during the study period, including

Table 2 Comparison between flow triggering and pressure-triggering systems as regards V_e , V_t , f , and rapid shallow breathing index

Ventilatory parameters	Flow (mean \pm SD)	Pressure (mean \pm SD)	<i>P</i>
V_e	10.93 \pm 3.14	10.67 \pm 3.91	0.555
V_t	0.61 \pm 0.19	0.594 \pm 0.22	0.436
f	19 \pm 5.49	19.31 \pm 5.8	0.645
RSBI	38.97 \pm 23.72	39.81 \pm 20.57	0.824

f, frequency; RSBI, rapid shallow breathing index; V_e , minute volume; V_t , tidal volume.

Table 3 Comparison between flow triggering and pressure-triggering types as regards the airway pressures, cycle times, and derived parameters

Parameters	Flow (mean \pm SD)	Pressure (mean \pm SD)	<i>P</i>
P_{pk}	23.28 \pm 3.59	23.77 \pm 3.53	0.001
P_{mean}	10.76 \pm 2.14	10.04 \pm 2.42	0.008
ΔP	1.5 \pm 2.32	2.03 \pm 2.26	0.001
C_{dyn}	39.91 \pm 15.1	35.17 \pm 13.73	0.0006
T_i	0.98 \pm 0.345	0.92 \pm 0.34	0.242
T_e	2.46 \pm 0.82	2.47 \pm 0.84	0.883
T_i/T_{tot}	0.29 \pm 0.068	0.28 \pm 0.07	0.093
PTI	3.14 \pm 0.98	2.80 \pm 1.08	0.0288
Ventilatory efficiency	0.28 \pm 0.089	0.25 \pm 0.094	0.0016

ΔP , difference between measured and calculated peak pressure; C_{dyn} , dynamic compliance; P_{mean} , mean airway pressure; P_{pk} , peak airway pressure; PTI, pressure time product; T_i , inspiratory time; T_e , expiratory time; T_i/T_{tot} , (fraction on inspiratory time to total cycle time).

sweating, use of accessory muscles of respiration, or any significant change in hemodynamics, blood pressure, heart rate, or the presence of any arrhythmias during the study.

Comparison of the two triggering systems at different sensitivity levels

Our patients were further segregated according to the trigger sensitivity level into those having a sensitivity of 3 cmH₂O l/min) and those with a lower sensitivity. We compared the two triggering systems at the variable sensitivity levels.

At sensitivity level less than 3 cmH₂O l/min

Setting a trigger sensitivity level less than 3 cmH₂O l/min, there was a statistically significant higher P_{pk} and ΔP , and a significantly lower C_{dyn} and V_e with pressure triggering than with flow triggering. Tidal volume did not differ between patients on either trigger types (Table 4).

At sensitivity level of 3 cmH₂O l/min)

In patients with a set sensitivity level of 3 cmH₂O l/min, there was still a significantly higher P_{pk} and ΔP and a significantly lower P_{mean} and C_{dyn} during the pressure triggering than during the flow triggering system. The inspiratory time was higher in flow-triggered breaths than that in pressure-triggered ones; however, this did

not reach statistical significance. Moreover, the PaO₂ was significantly higher in flow-triggering type than in pressure-triggering type (Table 5).

Furthermore, we sought to evaluate the effect of pressure support while negating the effect of trigger sensitivity levels on the post-triggering pressure variation in the two triggering types.

Hence, we segregated our patients according to the pressure support level into those with PS of 15 cmH₂O or less and those with PS above 15 cmH₂O, using the same trigger sensitivity levels (3 or below).

Comparison of the two triggering types at lower PS levels (≤ 15 cmH₂O) (n=15 patients)

At trigger sensitivity less than 3 cmH₂O l/min

There were no significant differences between flow triggering and pressure-triggering system as regards any of the ventilatory parameters: P_{pk} (21.13±3.57 vs. 21.37±3.35, $P=0.190$), ΔP (0.78±0.71 vs. 1.03±0.54 $P=0.190$), P_{mean} (10.24±2.88 vs. 10.13±3.44, $P=0.864$), C_{dyn} (40.31±12.04 vs. 32.19±12.47, $P=0.085$), or PaO₂ (145.6±53.2 vs. 124.0±37.95, $P=0.123$) set at lower levels of PS with lower trigger sensitivity (<3).

At trigger sensitivity of 3 cmH₂O l/min

There was a significantly lower P_{pk} (20.40±2.99 vs. 21.44±3.46, $P=0.039$) and ΔP (0.39±1.32 vs. 1.43

Table 4 Comparison between flow triggering and pressure-triggering systems at sensitivity below 3 cmH₂O l/min (n=17)

Variables	Flow (mean±SD)	Pressure (mean±SD)	P
V_e	10.41±3.43	9.32±3.25	0.04
V_t	0.54±0.16	0.51±0.18	0.22
f	20.06±5.24	19.83±6.167	0.84
RSBI	40.85±15.99	45.05±21.93	0.36
P_{pk}	23.52±3.24	23.81±3.11	0.01
ΔP	0.93±1.06	1.28±0.78	0.02
P_{mean}	10.61±1.92	10.04±2.33	0.13
C_{dyn}	34.30±10.73	29.44±10.18	0.02
T_i	0.89±0.34	0.89±0.40	0.94
T_e	2.32±0.69	2.44±0.81	0.43
T_i/T_{tot}	0.28±0.07	0.26±0.08	0.27
PTI	2.96±0.97	2.64±0.96	0.06
Ventilatory efficiency	0.24±0.08	0.21±0.08	0.07
PaO ₂	111.88±45.98	102.29±33.31	0.09
PaCO ₂	39.19±9.63	38.97±8.89	0.66
O ₂ saturation	97.15±2.77	96.61±2.70	0.14
MAP	89.43±12.87	89.61±11.62	0.78
HR	95.38±13.02	94.19±12.99	0.478

C_{dyn} , dynamic compliance); HR, heart rate; MAP, mean arterial blood pressure; ΔP , difference between measured and calculated peak pressure); P_{mean} , mean airway pressure; P_{pk} , peak airway pressure; PTI, pressure time product; T_e , expiratory time; T_i , inspiratory time; T_i/T_{tot} , fraction on inspiratory time to total cycle time.

Table 5 Comparison between flow triggering and pressure-triggering systems at sensitivity of 3 cmH₂O l/min (n=15)

Variables	Flow (mean±SD)	Pressure (mean±SD)	P
V_e	11.60±2.68	12.42±4.11	0.25
V_t	0.70±0.19	0.71±0.22	0.42
f	18.07±5.37	18.64±5.44	0.163
RSBI	36.55±31.56	33.07±17.11	0.59
P_{pk}	22.97±4.10	23.78±4.03	0.009
ΔP	0.75±1.37	1.56±1.76	0.01
P_{mean}	10.96±2.47	10.04±2.62	0.02
C_{dyn}	47.13±17.13	42.53±14.50	0.019
T_i	1.1±0.35	1.00±0.30	0.09
T_e	2.51±0.81	2.48±0.83	0.73
T_i/T_{tot}	0.31±0.07	0.29±0.06	0.22
PTI	3.43±1.16	2.95±1.01	0.0616
Ventilatory efficiency	0.33±0.08	0.29±0.09	0.001
PaO ₂	131.93±46.51	114.5±36.02	0.009
PaCO ₂	31.91±7.40	31.9±7.99	0.96
O ₂ saturation	98.80±1.84	98.52±1.71	0.22
MAP	94.25±13.37	93.75±10.51	0.70
HR	90.36±26.20	90.64±25.98	0.80

C_{dyn} , dynamic compliance; HR, heart rate; MAP, mean arterial blood pressure; ΔP , difference between measured and calculated peak pressure); P_{mean} , mean airway pressure; P_{pk} , peak airway pressure; PTI, pressure time product; T_i , inspiratory time; T_e , expiratory time; T_i/T_{tot} , fraction on inspiratory time to total cycle time.

± 1.92 , $P=0.0039$) and a significantly higher C_{dyn} (54.56 ± 15.02 vs. 47.28 ± 13.99 , $P=0.021$) during the flow triggering than during the pressure-triggering system. However, there was no significant difference as regards P_{mean} (11.33 ± 3.11 vs. 10.91 ± 3.09 , $P=0.302$), PTI (3.47 ± 1.37 vs. 3.18 ± 1.25 , $P=0.269$), and minute volume (11.2 ± 2.71 vs. 11.74 ± 3.35 , $P=0.334$) for flow triggering versus pressure triggering.

Comparison of the two triggering types at higher PS levels ($>15 \text{ cmH}_2\text{O}$):($n=17$ patients)

At trigger sensitivity less than $3 \text{ cmH}_2\text{O l/min}$

In the flow triggering type, there was a significantly lower P_{pk} and ΔP than in the pressure-triggering type (25.03 ± 1.91 vs. 25.36 ± 1.70 and 1.01 ± 1.25 , $P=0.03$ vs. 1.44 ± 0.88 , $P=0.049$, respectively). Although P_{mean} was higher in the flow-triggering type, this did not reach statistical significance. (10.85 ± 1.07 vs. 9.99 ± 1.47 , $P=0.07$). There was no significant difference as regards C_{dyn} , PTI, and V_e (30.47 ± 8.02 vs. 27.70 ± 8.61 , $P=0.089$; 2.87 ± 0.83 vs. 2.51 ± 0.79 , $P=0.11$; and 10.09 ± 4.13 vs. 9.27 ± 3.76 , $P=0.18$, respectively) for flow triggering versus pressure triggering.

At a trigger sensitivity of $3 \text{ cmH}_2\text{O l/min}$

In the flow triggering system, there was a significantly higher P_{mean} than in the pressure-triggering system (10.47 ± 1.33 vs. 8.87 ± 1.27 , $P=0.049$). There was no significant difference as regards P_{pk} (26.4 ± 2.53 vs. 26.73 ± 2.89 , $P=0.36$), ΔP (4.73 ± 3.39 vs. 5.07 ± 3.28 , $P=0.36$), C_{dyn} (37.23 ± 15.54 vs. 36.22 ± 13.73 , $P=0.53$), or V_e (12.13 ± 3.35 vs. 13.32 ± 5.14 , $P=0.47$) for flow triggering versus pressure triggering.

Discussion

Main findings

Pressure triggering resulted in a significantly higher P_{pk} and ΔP and lower C_{dyn} at any equivalent sensitivity and PS regardless of the level. Moreover, at higher sensitivity levels, flow triggering resulted in a significantly higher P_{mean} , C_{dyn} , PTI, and PaO_2 . There was no significant difference between flow triggering and pressure triggering as regards the V_t , V_e , f , or RSBI variables of the cycle length, nor the ABG parameters ($P>0.05$).

The differences between the two triggering systems were most diverse with lower PS of less than $15 \text{ cmH}_2\text{O}$ and a high trigger sensitivity level of 3 resulting in a significantly higher P_{pk} and ΔP and lower C_{dyn} , ventilatory efficiency, and oxygenation with pressure triggering. At a higher PS level, it resulted in negation of such difference between the

two triggering systems as regards the work of breathing parameter and oxygenation, although a significantly higher P_{pk} remained with pressure triggering at different sensitivity levels, and only a lower ventilatory efficiency at higher trigger sensitivity.

Triggering type/level and airway pressures.

This higher P_{pk} and ΔP produced during pressure triggering can be attributed to the gush of air after triggering after an initial decline of pressure during the trigger phase. This initial decline may be inequivalent with the initial flow of the pressure-supported breath, leading to fluctuations of the airway pressures. Although this imbalance could have been annulled by adjusting the initial rise time/ramp, this was deliberately kept constant to evaluate the effect of the triggering phase only. Moreover, the ventilator response time may have contributed to this dyssynchrony; however, this variable is inherent to each ventilator and was constant with both types of triggering throughout the study.

Alternatively, this higher peak pressure could have been attributed to the patient's effort or straining to trigger expiration at the end of the inspiratory phase; in such case, the elevation in peak pressure is an expiratory cycling phenomenon rather than a triggering one. However, if the latter was the case, it would not have differed between the two triggering types, which proves that even if this is the primary cause it is an indicator of dyssynchrony that is affected by the trigger type and level.

Although recent types for triggering, which utilize the diaphragmatic pressure-driven servoventilation and the electrical activity of the diaphragm (neurally adjusted ventilatory assist) as triggering signals, have undoubtedly improved patient's triggering, they are not readily available on ventilators, and still the chief types of triggering used are the flow triggering and pressure triggering ones [15–19].

The effect of the triggering type on the work of breathing in the trigger phase has been previously evaluated using several techniques, one of which is using the airway graphics for evaluating the decline of pressure during the trigger phase. However, this may not be accurate or may be misleading as flow triggering may not have an effect on the pressure curve if the bias flow is high enough. Alternatively, it can be performed by measuring the transdiaphragmatic pressure, either internally using an esophageal sensor or externally using an electromyography. However, the techniques are invasive or require additional equipment that may

not be readily available for each patient in every ICU [20]. Hence, we sought to evaluate the effect of triggering type on patient's synchrony in the post-trigger phase using the measured P_{pk} and ΔP as the pressure surrogates for the degree of synchrony, when all other parameters are constant.

Several studies have found that pressure triggering induces more patient–ventilator dyssynchrony and higher work of breathing, either in the assisted breath in synchronized intermittent mandatory ventilation or in the spontaneous pressure-supported breaths [20–22]. Moreover, others have found that flow-triggered breaths induce less fluctuations of the airway pressures compared with pressure triggering [23,24].

In contrast, others found that a pressure trigger of $-0.5\text{ cmH}_2\text{O}$ was found to be more sensitive compared with flow triggering. However, in this study, a sophisticated uncommonly used respiratory monitoring system, whose accuracy was not well defined, was used for data analysis. In addition, very minute values were recorded in their study (e.g. 0.002), and hence subtle changes in any parameter could have affected the results. Moreover, they did not use a flow trigger sensitivity of less than 2 l/min ; perhaps using lower values that did not lead to autotriggering could have changed the results. Moreover, unlike our protocol, they used a very short time period before data collection, only 5 min for each triggering system [5].

However, using pressure triggering, the P_{pk} was significantly higher, but the P_{mean} was significantly lower than that using flow triggering. This can be attributed to the fact that the inspiratory time was longer during flow triggering than during pressure triggering. However, this difference in inspiratory time did not reach significance. This is in agreement with a clinical study performed on PSV using Puritan Bennet 7200AE ventilators, which showed a similar finding [10].

Our findings as regards the effect of the trigger type were consistent at all trigger sensitivity levels, indicating that, when patients were put on lower sensitivities, due to weaker respiratory muscles, more advanced diseases, or when ineffective triggering occurred at higher trigger sensitivity levels, patients became more synchronous when they were kept on flow triggering at the same sensitivity level. However, when patients were put on higher sensitivity levels when their muscle strength and disease process allowed or autotriggering was present, they showed

better synchrony when put on flow triggering, to the extent that this has affected their oxygenation.

The problems of inappropriate triggering (ineffective, auto, or double triggering) may be due to patient factors such as cardiac contractions, retained secretions, involuntary movements, or ventilator/circuit factors such as swinging tubing/water, system leakage, or noncompliant inspiratory valve. In our study, ventilator/circuit factors were regularly checked and avoided, whenever possible. We did not encounter any significant trigger abnormalities throughout the study period, although these trigger abnormalities are known to be fleeting and could have been present outside the study window. Other factors could have been present but were common factors for both triggering systems.

Triggering type and other ventilatory parameters and ABG
The trigger type showed no significant difference in V_t , f , and their derivatives (V_e and RSBI). This indicates that, although there was evidence of dyssynchrony between the two triggering types, this did not reach significance to affect the ventilation, as these parameters are mainly dependent on post-triggering pressure support level (i.e. the driving pressure) in spontaneously breathing patients rather than the triggering phase.

This is in agreement with different studies comparing the two trigger levels and found no difference in the breathing patterns or the minute ventilation [11,20]. However, other studies found that flow triggering when added to PSV resulted in a significant improvement in the respiratory rate, rapid shallow breathing index, and tidal volume (V_t) as well as V_e [20,25].

Similarly, the trigger types in our study showed no significant difference as regards the PCO_2 . This can be attributed to the fact that there was no difference between the triggering types as regards the volumes (V_t , and V_e). In addition, this finding may also point out that the difference between the two trigger types and the resultant difference in work of breathing by the respiratory muscles was not large enough to increase the CO_2 production by the respiratory muscles having the same dead space factor.

However, oxygenation was significantly higher with flow triggering. This is in line with the finding that the P_{mean} , the main determinant of oxygenation, was significantly higher with flow triggering. It may also suggest that better synchrony resulted in better ventilation perfusion matching.

Tutuncu and colleagues did not find any significant difference between the two types of triggering as regards the PCO_2 or PO_2 . They attributed the absence of difference to the fact that they used a Servo 300 that has an advanced pressure triggering technology that approximates flow triggering [14].

Importantly, our results were consistently present in both the old and new ventilator technologies that we used. This indicates that the pressure-triggering type needs further development. Alternatively, such difference is due to the inherent nature of triggering in which pressure triggering needs a closed system to operate properly.

Our results showed that C_{dyn} was significantly higher during flow triggering than during pressure triggering. Although there was no significant difference as regards the V_t , the P_{pk} (and driving pressure) with flow triggering was significantly lower to explain the higher dynamic compliance. We assumed that the elevated P_{pk} is dependent on the PS level (and the generated V_t), baseline CPAP level, and patient-ventilator interaction. Further, as the PS and CPAP levels were similar in the two trigger types and the generated V_t was found to be similar, the P_{pk} from which the dynamic compliance is derived was mainly dependent on the patient-ventilator interaction (i.e. no added pressure to the pressure limit for the similar V_t generated) and therefore was assumed to be another indicator for synchrony and ease of breathing in our study. To our knowledge, this is the first study to find the dynamic compliance as a feasible surrogate for synchrony in evaluating the triggering phase.

This is in disagreement with the findings of Barrera *et al.* [20], who found no statistically significant difference between the two triggering systems as regards dynamic compliance, which can be attributed to the method of measurements, which used esophageal balloon for measuring dynamic compliance.

Pressure support and patient's synchrony

The main effect of PS, the driving pressure, is on the inspiratory flow/pressure synchrony, in the post-trigger phase. To optimize patient-ventilator synchrony with PS, it is imperative to modulate the initial flow and breath termination individually in each patient. Our results have shown that the difference between the two trigger types was most evident at lower PS of less than 15 cmH_2O and higher triggering sensitivity levels. However, with higher PS, the increase in inspiratory pressure negated the difference in work of breathing parameters only. However, the higher level of pressure

support did not nullify the difference in P_{pk} , which remained to be significant between the two trigger types - that is, it was not capable of abolishing the degree of dyssynchrony induced with pressure triggering.

Stating our results differently, at higher sensitivity, there was a significant difference in P_{pk} pressure between the two triggering systems at all corresponding levels of pressure support. This indicates that the main effect of dyssynchrony was mainly related to the type of trigger rather than the pressure support level.

In accordance with our results, Tutuncu and colleagues showed that the application of flow-triggered pressure support ventilation led to a significant reduction in both peak and mean airway pressures at identical levels of ventilatory support. Peak airway pressures were slightly higher during pressure-triggered pressure support ventilation conditions for full and for partial ventilator/support [14]. Recently, the effects of the driving pressure as an indicator of dyssynchrony has been used, however, in the post-trigger phase [2,4,26].

Our study has several limitations: first, a counterbalanced design was not used in the study design, as all patients were basically kept on pressure triggering at the enrolment phase. Hence, this was not considered a crossover, but rather a post-test within-subject design. Moreover, we did not use the initial measurement of pressure triggering as the pretest (control), but we opted to put the patient again on pressure triggering (after flow) to measure the outcome after the same testing period of 60 min, to omit the fatigue/improvement variable.

Although we have excluded known chronic obstructive pulmonary disease patients with evident air trapping from our study, auto-positive end expiratory pressure (auto-PEEP) in spontaneous breathing may still have been present in some patients and could have affected the trigger. However, a constant baseline CPAP of 5 cmH_2O was used in all our patients to counteract the possible auto-PEEP level. Moreover, auto-PEEP, if present, may have had a similar effect during the two types of triggering. Similarly, C_{dyn} could have been measured using esophageal balloon to evaluate the true pressure generated (decline) by the patient to produce the inspired tidal volume, and may have shown a higher difference. However, esophageal balloon to measure both, the auto-PEEP and C_{dyn} , is not a readily available tool in every ICU.

Finally, the equivalent sensitivity levels of flow and pressure triggering are not exactly known, and to our

knowledge this is the first study to show that equivalent values of sensitivities are better with flow triggering than with pressure triggering. However, in our study we arbitrarily chose the same numerical values for simplicity.

Conclusion

At different trigger and pressure support levels, flow triggering, as opposed to pressure triggering, significantly improved patient–ventilator synchrony in a short-term study on patients on pressure support ventilation on an old and new ventilator technology.

During pressure support ventilation, peak pressure variation and ΔP are simple noninvasive indicators of trigger dyssynchrony, whereas dynamic compliance can be used as a possible surrogate for the total breath synchrony. Further improvements in readily available trigger types are imperative to ensure better patient synchrony.

Clinical implications and future directions

This study highlights the current limitations in triggering on patient's synchrony. Further developments are still required in such field. Moreover, detection algorithms for dyssynchrony are currently available; however, future directions for automated adjustments in trigger sensitivity could help in immediate improvements in patient–ventilator synchrony. This current study presents additional indicators that can be used for automatic detection and adjustments of the trigger sensitivity.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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