Different modes of assisted ventilation in patients with acute respiratory failure

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Different modes of assisted ventilation in patients with acute respiratory failure. D. Chiumello, P. Pelosi, E. Calvi, L.M. Bigatello, L. Gattinoni. ©ERS Journals Ltd 2002.
ABSTRACT: The aim of the present study was to verify that the patient/ventilator interaction is similar, regardless of the mode of assisted mechanical ventilation (*i.e.* pressure- or volume-limited) used, if tidal volume (V_T) and peak inspiratory flow (PIF) are matched. Therefore, the authors compared the effects of three different modes of assisted ventilation on the work of breathing (WOB) and gas exchange in patients with acute respiratory failure.

For Protocol 1, in seven patients, the authors compared pressure support, assist pressure control and assist control (with square and decelerating wave inspiratory flow pattern) set to deliver the same V_T and PIF. For Protocol 2, in another 10 patients, the authors compared pressure support and assist control with high (0.8 $L·s^{-1}$) and low (0.6 $L·s^{-1}$) PIFs set to deliver the same V_T.

In Protocol 1, there was no difference in WOB and gas exchange between the three modes of assisted ventilation tested. In Protocol 2, the decrease of PIFs during assist control significantly increased WOB.

In conclusion, different modes of assisted ventilation similarly reduce work of breathing and provide adequate gas exchange at fixed tidal volume and peak inspiratory flow only. During assist control, tidal volume and peak inspiratory flow (set by the physician) are the main determinants of the patient/ventilator interaction.
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A primary goal of mechanical ventilation is to improve gas exchange and reduce the work of breathing (WOB) of patients with acute respiratory failure, without causing iatrogenic lung injury [1]. Assisted ventilation allows the patient to contribute to minute ventilation ($V'E$) and offers several advantages over controlled ventilation. It can reduce the need for sedation and paralysis, decrease the risk of barotrauma [2], improve intrapulmonary gas distribution [3], and prevent muscle atrophy [4, 5]. During assisted ventilation, the WOB is dependent on both the ventilator settings and the patient′s ventilatory demand and mechanics.

Pressure support ventilation (PSV) is a pressure-limited, flow-cycled mode of assisted ventilation, in which each breath is supported by a constant level of pressure at the airway (P_{aw}), so that the tidal volume (V_T) and inspiratory flow are more adaptable to the patient′s own ventilatory demand [6]. This manner of supporting the patient′s own ventilatory effort may be responsible for an improved comfort and synchrony with the ventilator, and has been shown to reduce the WOB and prevent diaphragmatic fatigue in patients with respiratory failure [7]. The main disadvantage of PSV is that it only works best in patients with stable

respiratory conditions (*i.e.* an adequate sufficient ventilatory drive and somewhat preserved respiratory mechanics [8, 9]).

Assist control ventilation (ACV) is a volume-limited mode of assisted ventilation, in which the V_T and the peak inspiratory flow (PIF) are set by the clinician and are not altered by the patient′s ventilatory demand. If the required V_T and inspiratory flow are higher than what the ventilator supplies, the WOB increases and the patient may develop fatigue [10]. Consequently, the main advantage of ACV over PSV is that ACV ensures a set V_T during conditions of variable ventilatory drive and mechanics [9]. However, several studies have found that during ACV the WOB can increase when the ventilator is unable to meet a patient′s high ventilatory demand [11–13]. Comparisons of PSV and ACV, matched to deliver the same V_T, are few and they have only evaluated one V_T in mixed-patient populations (*i.e.* acute and chronic respiratory failure) [14, 15].

Assist pressure control ventilation (APCV) is a pressure-limited mode of assisted ventilation similar to PSV in that the inspiratory flow is delivered at a variable rate and with a decelerating wave flow pattern. It differs from PSV in that the inspiratory

time is preset, thus providing a greater control of V_T [1]. A possible advantage of PSV and APCV compared to ACV is the decelerating wave flow pattern, which presents an inspiratory flow rate that is very high at the beginning and variable in a similar manner to a spontaneous breath. Several studies have shown that a decelerating wave flow pattern can reduce the WOB, improve lung mechanics and optimise gas exchange [16–19].

The aim of the present study was to verify that patient/ventilator interaction is similar, regardless of the mode of assisted mechanical ventilation tested, if V_T and PIF are matched. However, in contrast to previous studies, the authors investigated a homogeneous group of patients, accurately matching V_T and PIF.

In the first protocol (Protocol 1), the authors compared the effects of different levels of PSV and APCV with the effects of ACV set to deliver equal V_T and PIF. ACV was studied with two different wave flow patterns: square and decelerating. In the second protocol (Protocol 2), the effects of PSV and ACV set to deliver a high or low PIF at a constant V_T were compared.

Materials and methods

Patients

The two study protocols were carried out in patients with acute respiratory failure. Patients were included if they were mechanically ventilated with PSV, haemodynamically stable and without evidence of chronic obstructive pulmonary disease (COPD). Seven patients were enrolled in the first protocol and 10 patients were enrolled in the second. The patients' characteristics are reported in table 1, and one patient (patient no. 1) participated in both protocols. The study was approved by the Institutional Review Board and informed consent was obtained from each patient or next of kin.

General protocol

Three modes of assisted ventilation were compared: PSV, APCV, and ACV [20]. PSV is a pressure-limited, flow-cycled mode of assisted ventilation, in which the ventilator stops applying flow when the inspiratory flow rate decreases below a threshold value; in the authors' ventilator, 30% of PIF. APCV is a pressure-limited, time-cycled mode of assisted ventilation. ACV is a volume-limited, volume-cycled mode of assisted ventilation.

All patients were mechanically ventilated with a Bear® 1000 mechanical ventilator (Bear Medical Systems Inc., Riverside, CA, USA) [20]. The sensitivity of the pressure trigger was set at -2 cmH_2O. In the APCV and ACV modes, a back-up respiratory rate (RR) of zero was set, so that the RR depended only on the patient's own drive. The inspiratory oxygen fraction (F_{I,O_2}) and positive end-expiratory pressure (PEEP) level were previously set by the attending physician on the basis of a PEEP trial [21] and remained constant throughout the study. Patients were allowed to breathe in each modality for ≥20 min. Respiratory mechanics, arterial blood gas tensions and haemodynamic data were recorded at the end of each study period.

Table 1. – Baseline patients characteristics

Patient no.	Sex M/F	Age yrs	BMI kg·m^{-2}	PSV cmH$_2$O	PEEP cmH$_2$O	$P_{a,O_2}/F_{I,O_2}$ mmHg	$C_{st,rs}$ mL·cmH$_2$O^{-1}	$R_{tot,rs}$ cmH$_2$O·L^{-1}·s	Diagnosis
Protocol 1									
1	F	85	20.0	10	7	230	37.6	17.4	Peritonitis
2	F	76	29.3	8	4	140	31.1	17.7	Pneumonia
3	F	62	47.8	10	9	185	35.0	12.5	Pneumonia
4	M	65	27.7	20	8	120	27.0	10.7	Peritonitis
5	M	19	21.6	10	4	310	44.3	11.5	Politrauma
6	M	60	24.7	8	5	328	57.9	5.1	Pneumonia
7	F	40	21.5	8	4	377	24.4	10.4	Pneumonia
Mean±SD	4F/3M	58±22	27±9	10±4	6±2	241±99	36.9±11.3	12.2±4.4	
Protocol 2									
1	F	85	20.0	10	4	230	37.6	17.4	Peritonitis
2	F	63	47.8	18	8	108	32.6	8.8	Pneumonia
3	M	65	26.8	20	10	158	26.2	13.4	Peritonitis
4	M	69	21.6	6	2	230	60.1	15.1	Pneumonia
5	M	80	18.5	5	5	373	53.3	7.2	Pneumonia
6	M	35	26.3	8	7	317	78.0	7.5	Peritonitis
7	F	77	28.9	7	4	271	17.8	8.4	Pneumonia
8	M	61	28.7	14	4	210	58.0	12.2	Pneumonia
9	M	64	22.1	11	6	252	27.0	7.1	Pneumonia
10	F	56	21.9	15	6	192	27.0	2.1	Peritonitis
Mean±SD	4F/6M	65±14	26±8	11±5	6±2	234±76	44.8±19.1	9.9±4.6	

M: male; F: female; BMI: body mass index; PSV: pressure support ventilation (clinical level used at time of study); PEEP: positive end expiratory pressure; P_{a,O_2}: arterial oxygen pressure; F_{I,O_2}: inspiratory oxygen fraction; $C_{st,rs}$: static respiratory system compliance; $R_{tot,rs}$: total inspiratory respiratory resistance.

Protocol 1

First, one of three levels of PSV (5, 15 and 25 cmH$_2$O above PEEP) was applied in random order (sealed opaque envelopes). After full data collection, the ventilator was switched to either ACV with a square wave flow pattern, ACV with a decelerating wave flow pattern, or APCV. ACV was adjusted to provide the same VT and PIF developed at the tested level of PSV. When the wave flow pattern was changed from square to decelerating, the inspiratory time (tI) automatically increased to maintain a constant VT. APCV was set at three levels (5, 15, and 25 cmH$_2$O above PEEP), with a tI equal to the average tI obtained during PSV for the same level of support (fig. 1). These study periods

were then repeated in random order at each of the three levels of PSV, so that each patient had performed 12 trials of assisted ventilation by the end of the protocol.

Protocol 2

The present authors compared PSV to ACV set at two PIF rates, high and low, with both the square and decelerating wave flow patterns. The starting level of PSV was previously chosen by the attending physician on a clinical basis, to obtain an RR of <40 breaths per minute (bpm) and a VT of >4–5 mL·kg^{-1}. After a full baseline data collection, the ventilator was switched to
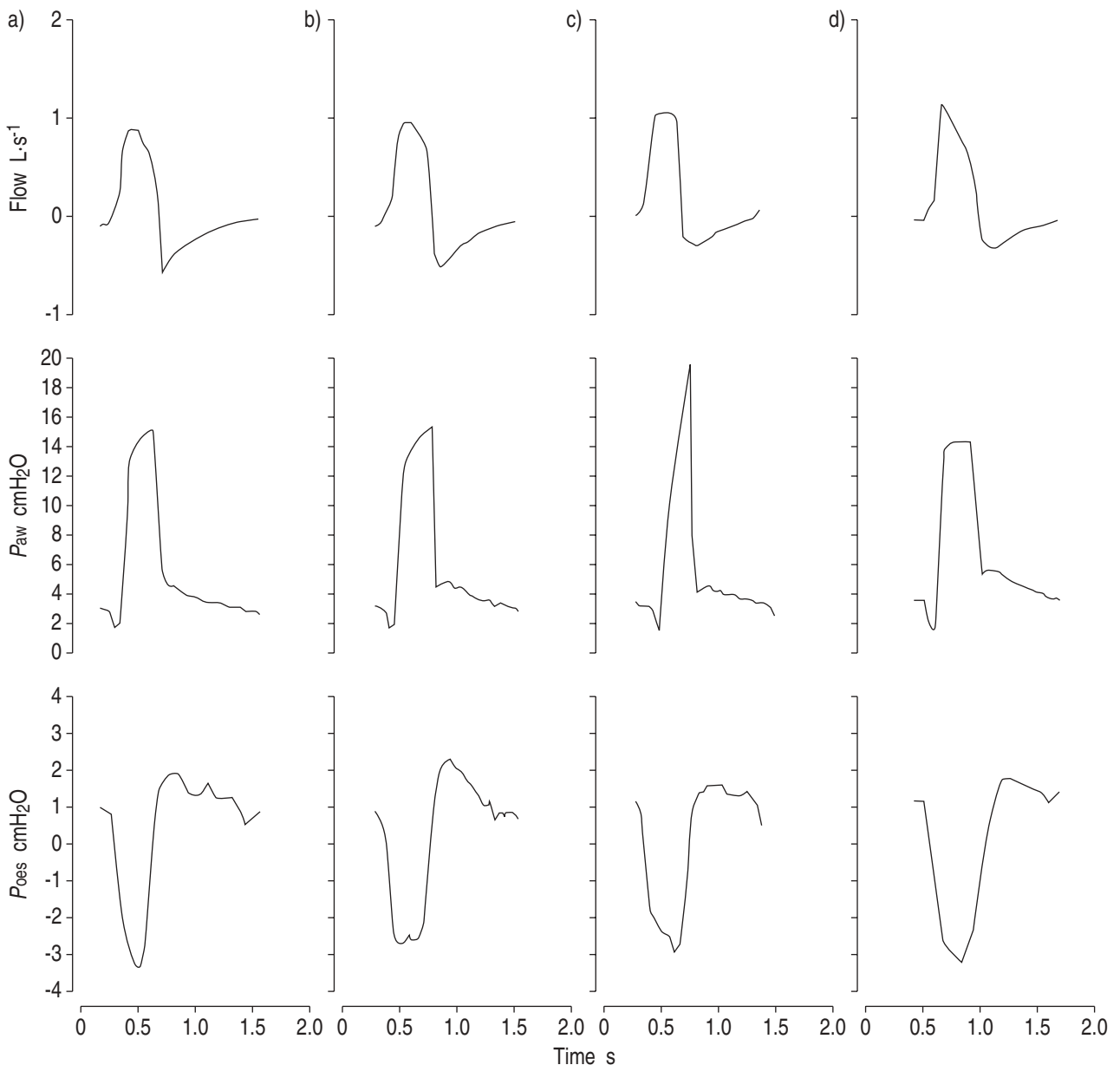

Fig. 1. – An example of airflow, airway (Paw) and oesophageal (Poes) pressure tracing during the different modes of assisted ventilation evaluated. a) Pressure support ventilation; b) assist pressure control ventilation; c) assist control ventilation (square wave); d) assist control ventilation (decelerating wave).

ACV, to deliver an equal V_T and PIF (high) with each of the two wave flow patterns (square wave high PIF, decelerating wave high PIF, respectively). Using the square wave flow pattern, the PIF was then reduced (low) to reach a t_I equal to the average t_I previously obtained with the decelerating wave flow pattern (square wave low PIF). The decelerating wave flow pattern was selected, maintaining the constant V_T and PIF previously selected (decelerating wave low PIF). At the end of protocol, each patient had performed five trials of assisted ventilation.

Measurements

The following measurements were obtained for each study period. Gas flow rate was measured with a heated pneumotachograph (No. 2; Fleisch, Lausanne, Switzerland) inserted between the proximal tip of the endotracheal tube and the "Y"-piece of the breathing circuit. P_{aw} was measured proximalto the endotracheal tube by a pressure transducer (MPX 2010 DP, Motorola, Phoenix, AR, USA). Oesophageal pressure (P_{oes}) was measured using a radiopaque, thin-walled latex balloon (8-cm long, SmartCath Bicore; Bicore, Irvine, CA, USA), sealed over one end of a polyethylene catheter and connected to a pressure transducer (Bentley Trantec; Bentley Laboratories, Irvine, CA, USA). During measurements, the oesophageal balloon was inflated with 0.5–1.0 mL of air. Its position and the validity of the pressure signal were assessed by a chest radiograph and by the occlusion test [22]. Both flow and pressure signals were recorded on a polygraph (Brush 2400 S; Gould, Cleveland, OH, USA), processed through an analogue-to-digital converter (100 Hz per channel) and stored on a personal computer for subsequent analysis and computations (Colligo, Elekton, Milan, Italy). The static compliance ($C_{st,rs}$) and the total inspiratory resistance ($R_{tot,rs}$) of the respiratory system were calculated by the inspiratory occlusion method during constant flow inflation (ACV square wave). The $C_{st,rs}$ was computed as the ratio between V_T and the difference between the end-inspiratory and end-expiratory airway pressure. The $R_{tot,rs}$ (including endotracheal tube or tracheostomy resistance) was measured by dividing the difference between the peak P_{aw} and plateau pressure by the inspiratory flow [21].

All patients had an indwelling arterial cannula, connected to a disposable pressure transducer (Transpec IV L974; Abbott Ireland, Sligo, Ireland). Arterial blood gas tensions were measured by a gas analyser (IL-1312 Blood Gas Manager and IL-282 CO-Oximetry; Instrumentation Laboratory, Milan, Italy).

Breathing pattern and respiratory effort

At the beginning of each data collection period, the authors recorded, for 2 min, consecutive undisturbed breaths to measure PIF, V_T, t_I, RR, V'_E, peak P_{aw}, WOB of the patient per minute (WOB·min^{-1}) and per litre (WOB·L^{-1}), and dynamic intrinsic PEEP (PEEP$_I$). V_T was obtained by mathematical integration

of the flow signal, V'_E was calculated as V_T multiplied by RR, and t_I was the time from the start of inspiration to the point of zero flow between inspiration and expiration. The WOB was computed from a modified Campbell's diagram, where the elastic recoil pressure of the chest wall was taken at the point of sharp inspiratory deflection of the P_{oes} tracings, i.e. the onset of inspiratory effort [23]. The chest wall elastance was calculated at the end of the study by plotting P_{oes} against V_T while the respiratory muscles were kept in a state of relaxation by a brief period (3–5 min) of slight hyperventilation to achieve relaxation [24]. PEEP$_I$ was considered equal to the difference between the P_{oes} value at the onset of inspiratory deflection and its value corresponding to the first point at zero flow [25]. Expiratory muscle activity could increase the gastric and oesophageal pressure, and thus overestimate the PEEP$_I$. Therefore, the authors could reasonably exclude such expiratory muscle activity by clinical inspection of the abdomen and chest [23].

In the last 3 min of each data collection period, three end-expiratory occlusion manoeuvres, ~15 s apart, were performed using the appropriate knob on the ventilator to measure the mouth occlusion pressure ($P_{0.1}$), which estimates the patient's ventilatory drive as the value of P_{aw} at 0.1 s after the beginning of inspiration [26].

Statistical analysis

All results are expressed as mean±SD. Comparisons between different modes of assisted ventilation were made using one-way repeated measures analysis of variance (ANOVA) and the Bonferroni t-test for multiple comparisons. When the data did not have a normal distribution, a Friedman repeated measures ANOVA as well as a Tukey's test for multiple comparisons were used. A p-value of <0.05 was considered statistically significant.

Results

Protocol 1

Increasing the level of support during PSV and APCV from 5–25 cmH$_2$O caused an increase in V_T and PIF (p<0.05) and a decrease in RR (p<0.05), with no significant change in V'_E (table 2). Similarly, the increase of V_T during ACV caused a decrease in RR (p<0.05) without significant changes in V'_E. By increasing the level of P_{aw} or V_T from the lowest to the highest studied, the average WOB·min^{-1} decreased from 12.2±3.7 J·min^{-1} to 1.4±0.3 J·min^{-1} (p<0.05), and the average WOB·L^{-1} decreased from 0.97±0.3 J·L^{-1} to 0.04±0.07 J·L^{-1} (p<0.05).

With the exception of the peak P_{aw}, which was higher with ACV with the square wave flow pattern, no differences were found in breathing pattern, WOB, $P_{0.1}$ and gas exchange when comparing PSV, APCV and ACV matched for the same V_T and PIF (table 2).

During ACV, the t_I was lower with the square wave

Table 2. – Comparison of pressure support ventilation (PSV), assist pressure control ventilation (APCV), and assist control ventilation (ACV)

	5 cmH$_2$O				15 cmH$_2$O				25 cmH$_2$O			
	PSV	APCV	ACV square wave	ACV decel. wave	PSV	APCV	ACV square wave	ACV decel. wave	PSV	APCV	ACV square wave	ACV decel. wave
PIF L·s^{-1}	0.67±0.11*,#	0.77±0.25*	0.70±0.13*	0.75±0.14*	0.92±0.19*	0.95±0.23*	0.91±0.24*	0.88±0.26*	1.18±0.24	1.16±0.30	1.17±0.26	1.14±0.28
V_T L	0.33±0.85*	0.36±0.33*	0.35±0.42*	0.34±0.47*	0.44±0.10*	0.44±0.10*	0.43±0.86*	0.42±0.84*	0.67±0.25	0.66±0.26	0.65±0.21	0.65±0.22
t_I s	0.72±0.10	0.77±0.13	0.64±0.10	0.73±0.13*	0.77±0.16	0.80±0.21	0.70±0.16	0.79±0.18	0.92±0.23	0.97±0.25	0.77±0.20	0.94±0.25
RR bpm	36±6*,#	33±4*	36±9*	35±5*	26±5	28±4	30±5	28±5*	21±5	20±5	22±5	19±7
V'_E L·min^{-1}	12±2.5	12.1±2.2	12.8±3.5	12.2±2.6	11.8±2.8	12.1±0.7	13.1±2.9	12.2±3	13.9±3.6	12.4±0.5	14.7±6	12.3±3.5
Peak P_{aw} cmH$_2$O	4.4±1.4*,#	4.9±1.2*,#	7.8±4.2*,#	4.3±3.1*,#	14.1±1.2*,¶	13.8±2.3*,¶	20.0±5.5*	15.2±5.7*,¶	24.0±0.8*,¶	23.4±1.6*,¶	32.8±5.8	25.2±2.9*,¶
PEEP$_I$ cmH$_2$O	1.9±0.7	2.5±1.9*	1.8±1.1	1.3±0.8	1.2±1.1	1.8±1.6	1.3±1.2	1.5±2	0.3±0.3	0.5±0.6	0.2±0.3	0.3±0.4
WOB$_{min}$ J·min^{-1}	10.6±6.2*	17.5±9.7*,#	8.9±6.9*	12.1±10*	4.7±6.1	6.0±6.0	3.7±4.5	3.8±4.3	1.3±3.0	1.9±3.0	1.2±2.9	1.2±3.0
WOB$_L$ J·L^{-1}	0.88±0.5*	1.40±0.6*,#	0.67±0.4*	0.95±0.7*	0.40±0.4	0.50±0.49	0.30±0.33	0.33±0.3	0.01±0.2	0.03±0.2	0.01±0.2	0.01±0.2
$P_{0.1}$ cmH$_2$O	2.2±2.0	2.4±2.2	2.4±2.1	2.5±2.1	0.8±0.4	1.2±0.7	1.0±0.4	1.0±0.7	0.2±0.6	0.4±0.3	0.5±0.6	0.3±0.4
P_{a,O_2} mmHg	96±18	103±24	106±19	100±21	104±22	109±23	105±25	106±27	102±18	109±18	109±19	108±15
P_{a,CO_2} mmHg	37±7	37±9	35±6	35±8	35±8	35±7	36±5	36±5	33±5	33±7	32±7	32±6

Data are presented as mean±SD. PIF: peak inspiratory flow; V_T: tidal volume; t_I: inspiratory time; RR: respiratory rate; V'_E: minute ventilation; P_{aw}: airway pressure; PEEP$_I$: intrinsic positive end-expiratory pressure; WOB$_{min}$: work of breathing per minute; WOB$_L$: work of breathing per litre; $P_{0.1}$: occlusion pressure in the first 100 ms; P_{a,O_2}: arterial oxygen pressure; P_{a,CO_2}: arterial carbon dioxide pressure; decel.: decelerating; bpm: breaths per minute. Considering comparison across the three cells of the table within one mode of ventilation. Considering comparison within the cell of the table between modes of ventilation. *: p<0.05 versus second cell. #: p<0.05 versus third cell. ¶: p<0.05 versus square wave.

flow pattern compared to the decelerating wave flow pattern, although not significantly. There were no differences in RR, V'E, WOB, P0.1 and gas exchange with the square and the decelerating wave flow patterns matched for the same VT.

Protocol 2

The mean level of PSV was set clinically at 11 ± 5 cmH$_2$O. PSV compared to ACV with the square and decelerating wave flow pattern matched for the same VT and PIF, showed no differences in RR, V'E, WOB, P0.1 and gas exchange (table 3). During ACV with the square wave, the peak Paw was higher than during PSV and ACV with the decelerating square wave flow pattern ($p<0.05$). The tI was significantly lower with the square wave flow pattern than with the decelerating wave flow pattern.

The decrease of PIF maintaining constant VT caused an increase in WOB·L^{-1} from 0.29 ± 0.47 J·L^{-1} to 0.40 ± 0.51 J·L^{-1} with the square wave flow pattern ($p<0.05$) and from 0.32 ± 0.43 J·L^{-1} to 0.40 ± 0.50 J·L^{-1} with the decelerating wave flow pattern ($p<0.05$, table 3).

Discussion

The main finding of the present study was that in a uniform patient population recovering from acute respiratory failure, three different modes of assisted ventilation (PSV, APCV and ACV) maintaining a similar VT and PIF, had comparable effects on breathing pattern, WOB and gas exchange. With ACV, a lower PIF was associated with an increase in the WOB.

Pressure support ventilation and assist pressure control ventilation

During PSV, the mechanical inflation stops when the inspiratory flow falls below a threshold value. Thus, PSV may adapt to breath-to-breath variability [27] more precisely than APCV, where the mechanical inflation stops after a preset time. However, the present authors found no differences in the patient/ventilator interaction [28] (*e.g.* breathing pattern, WOB, gas exchange) with PSV and APCV. Possible explanations are that: 1) breath-to-breath variability of the study's population in terms of RR, VT and PIF ($<10\%$ during 2 min of recording) was low at each level of selected inspiratory pressure; and 2) by design, the ventilatory settings on PSV and ACV had to be similar, in order to maintain a similar VT and PIF.

At high levels of pressure, the cycling criteria of PSV could be a problem in some COPD patients because the inspiratory flow changes more slowly without reaching the threshold value, so these patients start to exhale while the ventilator is still given gas [29]. However, this mainly occurs when the pressure set is excessive, dictating both the breathing pattern and VT according to the patient impairment of respiratory mechanics. In these situations, if the physician wants to assist the patient breathing pattern, it would be better to reduce the inspiratory pressure. Conversely, if the physician wants to control the delivered VT, the APCV seems more appropriate.

Assist control ventilation and pressure support ventilation

During ACV, all breaths are delivered with a set VT, PIF and wave flow pattern (square or decelerating). The fast and variable inspiratory flow characteristics of PSV may be more suitable than a fixed flow to match the patient's own ventilatory effort [6]. However,

Table 3. – Comparison of pressure support ventilation (PSV) and assist control ventilation (ACV) with high and low inspiratory peak inspiratory flow (PIF)

	PSV	High PIF		Low PIF	
		ACV square wave	ACV decel. wave	ACV square wave	ACV decel. wave
PIF L·s^{-1}	0.78 ± 0.19	0.80 ± 0.20	0.78 ± 0.20¶	0.65 ± 0.19*,#	0.64 ± 0.18+,#,*
VT L	0.37 ± 0.11	0.38 ± 0.12	0.37 ± 0.11	0.38 ± 0.11	0.37 ± 0.12
tI s	0.89 ± 0.39	0.76 ± 0.33#	0.88 ± 0.35*	0.86 ± 0.35	1.10 ± 0.47*,+,¶,#
RR bpm	31 ± 11	31 ± 10	30 ± 10	30 ± 9	29 ± 9
V'E L·min^{-1}	10.8 ± 3.2	11.5 ± 3.3	10.8 ± 3.0	11.3 ± 3.0	9.9 ± 2.7
Peak Paw cmH$_2$O	9.9 ± 5.7*,¶	17.1 ± 6.3	10.9 ± 4.8*,¶	14.7 ± 6.8	9.3 ± 3.0*,¶
PEEPI cmH$_2$O	1.5 ± 1.3	1.7 ± 2.2	1.8 ± 2.2	1.7 ± 2.2	2.6 ± 3.0
WOBmin J·min^{-1}	2.9 ± 3.0	3.3 ± 5.8	3.5 ± 5.2	4.1 ± 6.1*	3.8 ± 5.3
WOBL J·L^{-1}	0.30 ± 0.28	0.29 ± 0.47	0.32 ± 0.43	0.40 ± 0.51*	0.40 ± 0.50+
P0.1 cmH$_2$O	1.5 ± 1.0	1.5 ± 1.0	1.8 ± 1.9	1.9 ± 1.8	1.9 ± 1.7
Pa,O$_2$ mmHg	97 ± 16	96 ± 17	93 ± 12	99 ± 21	96 ± 16
Pa,CO$_2$ mmHg	39 ± 6	39 ± 8	39 ± 7	40 ± 9	40 ± 9

Data are presented as mean±SD. PIF: peak inspiratory flow; VT: tidal volume; tI: inspiratory time; RR: respiratory rate; V'E: minute ventilation; Paw: airway pressure; PEEPI: intrinsic positive end-expiratory pressure; WOBmin: work of breathing per minute; WOBL: work of breathing per litre; P0.1: occlusion pressure in the first 100 ms; Pa,O$_2$: arterial oxygen pressure; Pa,CO$_2$: arterial carbon dioxide pressure; decel.: decelerating; bpm: breaths per minute. *: $p<0.05$ *versus* ACV square wave high PIF; #: $p<0.05$ *versus* PSV; ¶: $p<0.05$ *versus* ACV square wave low PIF; +: $p<0.05$ *versus* ACV decelerating wave high PIF.

PSV has potential shortcomings. Hypoventilation may occur in the presence of abnormal respiratory mechanics, (*i.e.* decreased compliance and/or increased resistance), due to the imbalance between the inspiratory pressure delivered and the high inspiratory workload, even with an adequate respiratory drive.

TEJEDA *et al.* [14] and KREIT *et al.* [15] evaluated the effects of ACV and PSV on gas exchange and respiratory mechanics, respectively. As in the present study, these authors did not find significant differences between the two modes of ventilation. These studies, however, only investigated one level of VT.

The WOB performed by the authors' patients was identical during ACV and PSV (table 2) and was similar to values measured in patients with acute respiratory failure ventilated with ACV [15]. A seminal paper by MARINI *et al.* [11] showed that substantial work could be generated by patients during ACV when their own inspiratory flow did not match the pattern delivered by the ventilator. In some cases, the patients' WOB during machine-assisted cycles exceeded that of unsupported breaths [11, 12].

The present results differ from the observations of MARINI *et al.* [11] for the following reasons. 1) In the present study, ACV was better matched (using fixed VT and PIF) to the patients' ventilatory demands and breathing patterns. 2) MARINI *et al.* [11] used an older type of mechanical ventilator (*i.e.* one with a slower trigger phase and inspiratory flow delivery). In the present protocols, the trigger sensitivity was the same with ACV and PSV and the time delay was minimal, <100 ms. 3) The patient populations studied are different. The present authors' patients were all in a stable phase of acute respiratory failure, with no significant pre-existent respiratory disease. Those studied by MARINI *et al.* [11] were more heterogeneous, including patients with both acute and chronic respiratory failure, with and without the use of PEEP as part of the ventilatory strategy. Excluding COPD patients from the present study may have prevented increased WOB was caused by abnormality of the patients' respiratory mechanics (*e.g.* increased airway resistance, PEEPi), rather than or in addition to a characteristic of the ventilator.

The present authors found that ACV with the square wave form was as effective as PSV when the inspiratory time was reduced to obtain the same PIF. However, this is not commonly performed in clinical practice because of the risk of high peak Paw (tables 2 and 3) and possible patient/ventilator disynchrony. The authors found that the patient/ventilator interaction was similar, independent of the mode, if VT and PIF were matched. However, the PSV may be the easiest to set at the bedside, even when possible risks are into account.

Assist control ventilation and assist pressure control ventilation

APCV is characterised by a rapid increase of pressure at the airway, with a decelerating wave flow pattern [19, 30]. In a similar manner to PSV, the resulting VT is determined by the capability of the ventilator and the patient's respiratory mechanics. As in any pressure-limited mode, the VT can be substantially reduced by acute changes in respiratory mechanics, with the consequent risk of hypoventilation, although its clinical significance is not clearly documented. KALLET *et al.* [31] found a lower WOB with APCV than with volume control ventilation, due to the advantage of the high inspiratory flow pattern of APCV. CINNELLA *et al.* [32] observed a decrease in WOB using APCV compared to ACV only at a moderate VT (*i.e.* VT of 470 mL) and low inspiratory flow rate. In contrast, no differences in WOB with ACV (either with square or decelerating wave flow pattern) and APCV were found in this study. This discrepancy with the results of CINNELLA *et al.* [32] may be due to the different patient population studied, *i.e.* patients with COPD and air flow limitation, in whom the particular flow pattern of APCV, variable and maximum at the beginning of inspiration, is likely to be beneficial and generate higher VT. Furthermore, CINNELLA *et al.* [32] did not match the two modes for the same PIF.

Assist control ventilation: effect of square and decelerating wave flow pattern

Traditionally, volume-limited breaths during ACV are delivered with a square flow pattern (*i.e.* constant flow). In the latest generation of mechanical ventilators, however, the decelerating wave flow pattern is often available even in the volume-limited modes [33]. With the square wave flow pattern, the flow rate remains relatively constant after reaching the PIF, while with the decelerating wave flow pattern, the flow rapidly reaches the PIF value and then decreases linearly afterwards to 50% by the end of inspiration [20]. In patients with normal respiratory mechanics, different wave flow patterns have no major effect on gas exchange [34]. Also, no differences were found in gas exchange and WOB between the two wave forms. The present results differ with those of AL SAADY and BENNETT [33], in which the decelerating wave flow pattern produced a significant increase in arterial oxygenation. The decelerating wave flow pattern may be more effective in patients with heterogeneous respiratory mechanics and prolonged alveolar time constants (*i.e.* high compliance and high resistance) characteristic of COPD, or in patients with more severe lung injury [32]. In these circumstances, a high PIF followed by a slow decline may allow more time for gas distribution in the alveoli.

The square wave, which maintained a high flow throughout the inspiratory time, raised peak Paw more than the decelerating wave flow pattern during volume and pressure target ventilation (tables 2 and 3). An excessive PIF and a high peak Paw might cause patient discomfort and patient/ventilator disynchrony [6].

Assist control ventilation: effect of different peak inspiratory flow

Varying the PIF setting between 0.6–0.8 L·s^{-1} had a mild impact on the patients' WOB; with the square

waveform, the lower PIF was associated with a slightly but significantly higher WOB (table 3). WARD *et al.* [35] measured the WOB in patients with acute respiratory failure, changing the PIF between 0.4–1.1 $L \cdot s^{-1}$, while maintaining a constant V_T. It was demonstrated that at low PIF, the patient′s WOB approached 65% of the total inspiratory work. When the PIF was progressively raised to 0.9 $L \cdot s^{-1}$, the patient′s WOB was reduced to 10% of the total inspiratory work. Further increases of PIF $\leqslant 1.1$ did not affect the WOB [5]. In a previous study, similar results were obtained by the present authors, by increasing the PIF during PSV, where the highest PIF did not further reduce the WOB and was associated with patient discomfort [36]. Both these studies suggest that there is a threshold of flow demand below which a lower flow rate increases the WOB and above which a further increase of flow rate is not beneficial and may be uncomfortable [33, 35]. In the present study, only minor variations of PIFs were applied, according to clinical indications, and the authors did not find such a flow threshold, which would probably occur at a higher flow rate.

Limitations

The present study presents several limitations. First, only one type of mechanical ventilator was used to provide all the modes of assisted ventilation compared. The results obtained with this ventilator may not be applicable to other ventilators available. However, using a single ventilator capable of providing all of the modes of assisted ventilation that the authors wanted to compare, offered the advantage of a high homogeneity of experimental conditions.

Secondly, the patient population investigated had a low breath-to-breath variability in terms of RR, V_T, and PIF at each level of selected inspiratory pressure and with relatively preserved respiratory mechanics. All patients were without COPD and were recovering from acute respiratory failure. Thus, the authors′ values of WOB were relatively lower than in some other studies that investigated patients with higher ventilatory demands [31, 32].

Thirdly, setting APCV and ACV accordingly to a breathing pattern obtained during PSV facilitated the choice of comfortable setting of APCV and ACV. This is not what occurs most frequently in clinical practice, where patients are generally switched to PSV from APCV and ACV. However, the aim of the present study was to compare the three modes in the most objective way, which the authors believed they were able to accomplish with this design.

In conclusion, in a patient population with relatively preserved respiratory mechanics, it is not so important which ventilatory mode is chosen, but how each mode is regulated. The present authors identified tidal volume and peak inspiratory flow as the main parameters useful to optimising ventilatory settings. A further problem is how to find parameters, which are easily obtained at the bedside, to correctly regulate tidal volume and peak inspiratory flow for each ventilatory mode.

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