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PULSE AND SYSTOLIC PRESSURE VARIATION ASSESSMENT IN PARTIALLY ASSISTED VENTILATORY SUPPORT

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ABSTRACT. Objective. The use of pulse pressure variation (PPV) and systolic pressure variation (SPV) is possible during controlled ventilation (MV). Even in acute respiratory failure, controlled MV tends to be replaced by assisted ventilatory support. We tested if PPV and SPV during flow triggered synchronized intermittent mechanical ventilation (SIMV) could be as accurate as in controlled MV. **Methods.** Prospective case-controlled study. Thirty patients who met criteria of weaning from controlled MV. Twenty minutes pressure support ventilation with 3 min^{-1} flow triggered SIMV breathes (10 ml kg^{-1}) T1, then three consecutive breaths in controlled MV (respiratory rate 12 min^{-1},10 ml kg^{-1}) T2. PPV and SPV were measured in T1 and T2. Correlation and Bland–Altman analysis were used to compare respective values of PPV and SPV in the two modes of ventilation. **Results.** Significant correlations were found between dynamic indices in SIMV during pressure support ventilation and those in controlled MV mode. The mean differences between two measurements were: PPV $0.6 \pm 2.8\%$ (limit of agreement: -5.0 and 6.2), SPV 0.5 ± 2.3 mmHg (limit of agreement: -4.0 and 5.1). **Conclusions.** PPV and SPV measured during SIMV fitted with the findings in controlled MV. Dynamic indexes could be accurately monitored in patients breathing with assisted respiratory assistance adding an imposed large enough SIMV breath.

KEY WORDS. monitoring, cardiopulmonary ventilation, effects–ventilation, pressure support.

INTRODUCTION

Pulse pressure variation (PPV) and systolic pressure variation (SPV) seem to be better predictors of fluid responsiveness than static parameters during controlled mechanical ventilation (MV) with no breathing effort [1, 2] Spontaneous breathing may generate a tidal volume (TV) inadequate to change pulmonary venous flow and swing in pleural (transpulmonary) pressure, hence cardiac preload [2]. Moreover, active expiratory movements affect cyclic changes in intrathoracic and alveolar pressure, influencing ventricular preload and afterload [2–4].

Nowadays controlled MV tends to be limited even in the early phase of respiratory failure [5] to reduce barotrauma and need of deep sedation [6]. This prevents the use of dynamic hemodynamic monitoring during a substantial part of the ventilatory therapy [3, 7–10].

Our hypothesis was that during a pressure support assisted ventilatory approach (PSV) few imposed breaths

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[flow-triggered synchronized intermittent mechanical ventilation (SIMV)] for lung recruitment, could allow the monitoring of PPV and SPV.

This study compared PPV and SPV during PSV with SIMV and controlled MV in the same patient.

METHODS AND MATERIALS

All consecutive patients who met the weaning criteria from controlled MV were eligible. Patients meeting weaning criteria but having acute exacerbation of COPD, any significant cardiac arrhythmia, any sign of hypoperfusion (systolic blood pressure <90 mmHg), oliguria, or increased intra-abdominal pressure were excluded. The Hospital Ethics Committee approved the protocol and patients or next-of-kin gave informed consent.

Our weaning protocol from controlled MV states a 30 min trial in PSV plus SIMV for lung recruitment. Sedation is planned to obtain an oriented, cooperative and tranquil patient [11]. After the successful PSV trial we return to controlled MV and evaluate respiratory mechanics. Then, incremental periods of PSV plus SIMV are planned over the day.

Experimental protocol

PSV Trial test: Pressure support (PS) was set to obtain TV 6 ml kg^{-1}. Three SIMV acts per min (1 breath each 20 s) were added (10 ml kg^{-1}, duration 5 s, inspiration to expiration ratio 1:3, volume-controlled mode with flow trigger setting, SERVO C 300 Siemens-Elema AB, Solna, Sweden).

The patients who tolerated this test for 20 min were enrolled and dynamic indices were measured considering just SIMV breaths (T1). After data registration patients were shifted to volume-controlled MV (respiratory rate 12 min^{-1}, duration 5 s, inspiration to expiration ratio 1:3, TV 10 ml kg^{-1} [12], PEEP and FiO2 as in PSV) for three consecutive breaths, to register again dynamic indices (T2). Sedation (i.v. midazolam) was adjusted to avoid voluntary muscular contraction during measurements, checked from the respiratory curves.

Patients were monitored through a radial arterial catheter and a central venous line connected to a multifunctional monitor (H–P Monitor M1046A; Hewlett-Packard, Andover, MA). Transducers were positioned at the mid-axillary level and zeroed with atmospheric pressure. Throughout 20 min of data collection saline infusions were kept constant (3 ml h^{-1}) without performing any fluid loading. All measurements were taken simultaneously. The monitor and ventilator were connected to an analog-to-digital converter (Colligo, Elekton, Milan, Italy). Sampling frequency was 200 Hz.

Data collected on-line were stored on a computer for off-line analysis, carried out by one observer blinded to patient's identity and ventilation mode. PPV and SPV were calculated as average over a 2 min period; each minute of this period included three mechanical breaths (SIMV) alternated with breathings in pressure support (T1) or three mechanical breaths in volume control (T2). Arterial pressure, heart rate, and central venous pressure were calculated as the mean during a 3 s expiratory pause, airway pressures were recorded as the mean, inspiratory peak, and inspiratory plateau (Pplat). Quasi-Static Compliance of the respiratory system (TLSC) was calculated as TV $(\text{Pplat}-\text{PEEP})^{-1}$. The clinical severity at admission was determined with the severity score (SAPS II [13]).

Statistical analysis

The analysis was done using Stata Statistical Software, release 9.0 (Stata Corporation, College Station, TX). Results are reported as mean $\pm$ standard deviation for normally distributed values, median and quartiles [Q1–Q3], as appropriate. Student's paired two-tail *t*-test or Wilcoxon's signed rank test were used as needed. The correlation between PPV-SPV at T1 and T2 was tested using the Spearman rank method. To compare each variable at T1 and T2 Bland–Altman analysis was used. Significance was set at $P < 0.05$.

RESULTS

Twenty-two men and eight women were enrolled, the population features are express in Table 1. All patients had respiratory failure due to major abdominal surgery (15), pneumonia (6), head–neck surgery (3), peritonitis (3), pancreatitis (2), and hip replacement (1). The study was conducted after 2.9 ± 2.6 days in the ICU. The main

Table 1. Population features

Age (years)	65.4 ± 13.5
Body weight (kg)	70.3 ± 11.3
SAPS II	37 ± 16.6
Quasistatic lung compliance (ml cmH_2O^{-1})	25 [16–30]
Midazolam dose (mg)	4.3 ± 0.1
Height (cm)	168.1 ± 7.8
BMI	24.8 ± 3.5

Data presented as mean $\pm$ standard deviation; SAPS: Simplified Acute Physiology Score; BMI: Body Mass Index.

Table 2 . Hemodynamic and respiratory variables during the experiment

	T1 (PSV + SIMV)	T2 (VC)	*P*
RR (min^{-1})	10 [8.4–10]	12	NS
PS level (cmH_2O)	9 [5.5–11.2]	/	
PEEP, cmH_2O	5 [5–9]	5 [5–9]	NS
P peak (cmH_2O)	25 [16–30]	25 [22–29.5]	NS
FiO2 (%)	0.5 ± 0.1	0.5 ± 0.1	NS
TV (ml)	607.2 ± 79.3	622.5 ± 69.5	NS
TV PS (ml)	617.8 ± 95.3	/	
AP (mmHg)	83.0 ± 13.5	79.8 ± 16.2	NS
HR (beats min^{-1})	85.9 ± 18.8	85.4 ± 18.0	NS
CVP (mmHg)	9.2 ± 4.0	8.9 ± 4.2	NS
PPV (%)	8.2 [5.7–13.4]	7.2 [4.5–12.2]	NS
SPV (mmHg)	7.4 [5.3–10.6]	7.1 [4.5–9.6]	NS

Data presented as mean ± standard deviation or median [quartiles Q1–Q3]. RR: respiratory rate; PS: pressure support; PEEP: positive end expiratory pressure; TV: tidal volume; SIMV: synchronized intermittent mechanical ventilation; AP: mean arterial pressure; CVP: mean central venous pressure; PPV: pulse pressure variation; SPV: systolic pressure variation; /: no data; NS: not significant.

hemodynamic and respiratory variables at T1 and T2 are summarized in Table 2.

Significant correlations were found between dynamic indices in controlled MV mode and SIMV during PSV: PPV ($R = 0.89$, $P < 0.001$), SPV ($R = 0.86$, $P < 0.001$). Figure 1 shows Bland–Altman plots to assess agreement between variables.

The mean differences between times T2 and T1 were: PPV 0.6 ± 2.8% (limit of agreement: −5.0 and 6.2), SPV 0.5 ± 2.3 mmHg (limit of agreement: −4.0 and 5.1).

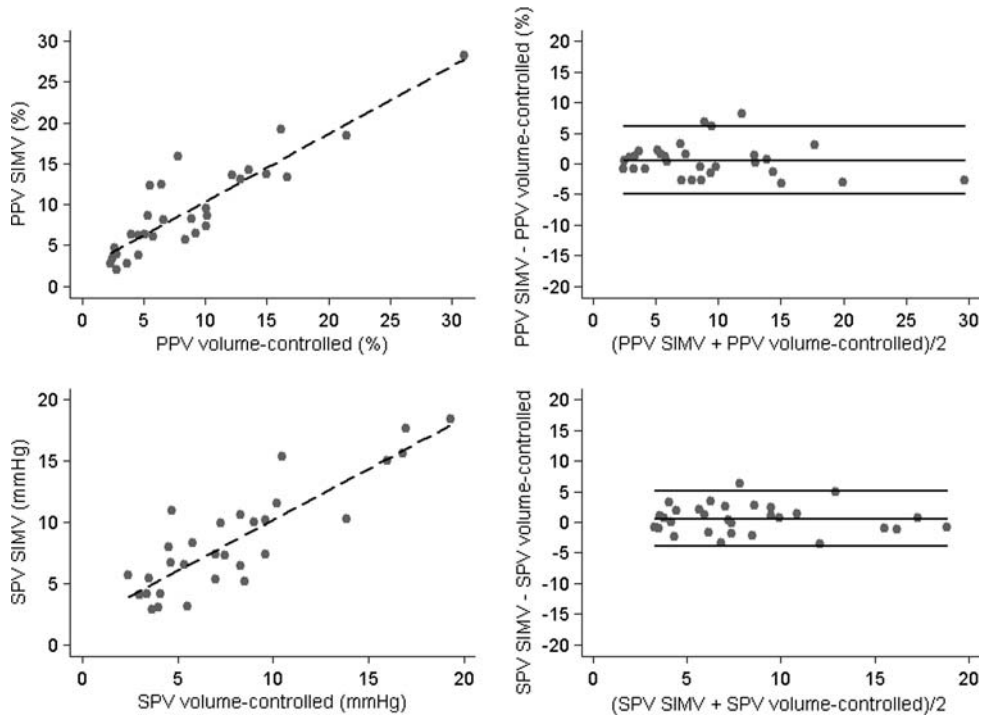

Fig. 1 . Scatter plots with linear regression lines (left part) and Bland–Altman plots (right part) of pulse pressure variation (PPV: upper half) and systolic pressure variation (SPV: lower part) to assess the agreement between SIMV and volume controlled MV.

DISCUSSION

The use of dynamic indices in patients with some breathing effort is considered not reliable [3, 7–10, 14, 15]. However, this assumption is based on patients breathing spontaneously without ventilation assistance [7, 9], with non invasive ventilation support [3] or in a mixed ventilation mode [8].

We found that the measurement of PPV and SPV during a flow-triggered SIMV act at TV 10 ml kg^{-1} [12] added to PSV, results in comparable values with those acquired later in the same patients treated with the same ventilatory parameters in controlled mode. Even if not entirely unexpected, this novel finding is important because it emphasizes that whichever the modality of assisted ventilation provided, simply adding a flow-triggered SIMV act of 10 ml kg^{-1}, an ICU patient can be monitored with SPV and PPV during the whole ventilatory period.

The study was not designed to predict fluid responsiveness but only to compare dynamic indices obtained with two modes of ventilation.

Several reasons might explain this: SIMV keeps pleural pressure positive throughout the respiratory cycle, delivers an imposed TV large enough to significantly affect venous return, as in controlled MV [12]; the flow trigger avoids any negative inspiratory effort which can alter pleural pressure [16] and expiratory effort is negligible because of the synchronous thoraco-abdominal expansion during inspiration when high enough inspiratory pressure is reached [17]. Finally, variability in the respiratory rate does not affect the number of heart beats per respiratory cycle as the duration of SIMV acts is fixed.

Our study has some limitations. The sequence of interventions was not randomized, because the experiment started when each patient successfully tolerated the first 20 min of the PSV trial test, and because the T2 period in controlled MV, had to be performed with the minor delay to guarantee the same hemodynamic pattern. Lung compliance was near-normal allowing a large enough TV to calculate dynamic indices [12] without exceeding safe respiratory pressure [18]. In patients with very severe acute lung injury this ventilatory set must be applied with care. Another limitation is the range of the limits of agreement in addition to the wide range of variability of threshold values (at least for PPV [19]). However, the best way to assess the fluid responsiveness is to perform two measurements, before and after a fluid challenge. In this scenario, considering the reliability of SPV and PPV monitoring in partial assisted ventilation (now available on-line at bedside [20]), it will be possible to widen the clinical application of heart–lung interaction during the whole ventilatory treatment in ICU.

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