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Noninvasive Assisted Pressure-Controlled Ventilation: As Effective as Pressure Support Ventilation in Chronic Obstructive Pulmonary Disease?

Cenk Kirakli[a] Tutku Cerci[b] Zeynep Zeren Ucar[a] Onur Fevzi Erer[b]
Hakan Alp Bodur[c] Semra Bilaceroglu[a] Serir Aktogu Ozkan[b]

[a]Intensive Care Unit and [b]Pulmonary Division, Izmir Training and Research Hospital for Thoracic Medicine and Surgery, and [c]Medical Intensive Care Unit, School of Medicine, Dokuz Eylul University, Izmir, Turkey

Key Words
Chronic obstructive pulmonary disease · Hemodynamics · Noninvasive ventilation · Pressure-controlled ventilation · Pressure-support ventilation

Abstract
Background: Noninvasive ventilation (NIV) is being increasingly used in hypercapnic chronic obstructive pulmonary disease (COPD) patients but the most appropriate ventilation mode is still not known. ***Objectives:*** The aim of this study was to investigate if assisted pressure-controlled ventilation (APCV) can be a better alternative to pressure-support ventilation (PSV) for NIV in COPD patients with acute hypercapnic respiratory failure (AHRF). ***Methods:*** In this prospective randomized study, we evaluated the early effects of noninvasive APCV and PSV in 34 consecutive COPD patients with AHRF. Patients were randomized into 1 of the 2 modes, and respiratory and hemodynamic values were compared before and after 1 h of NIV. ***Results:*** Baseline values did not differ between the 2 groups. There were significant improvements in partial arterial carbon dioxide pressure and pH levels in the APCV group when compared with baseline ($p < 0.05$). Cardiac output and cardiac index decreased in both groups ($p < 0.05$) but more significantly in the PSV group ($p < 0.0001$). The decreases in stroke volume index and increases in arterial oxygen content after NIV were also considerable in both groups ($p < 0.05$). Central venous pressure and systemic vascular resistance index values increased notably only after PSV ($p < 0.05$). ***Conclusions:*** From these data, we deduce that APCV can be a better alternative to PSV for NIV in COPD patients with AHRF owing to its more beneficial physiological effects.

Introduction

Although invasive mechanical ventilation (IMV) is a life-supporting procedure in chronic obstructive pulmonary disease (COPD) patients with acute hypercapnic respiratory failure (AHRF), it is also associated with an increase in the risk of complications such as barotrauma and infections leading to prolonged hospital and intensive care unit (ICU) stay [1, 2]. Several prospective randomized studies have shown that noninvasive ventilation (NIV) can improve respiratory parameters, and also decrease the length of hospital and ICU stay, intubation rates and risk of complications (and thus mortality), especially in hypercapnic respiratory failure due to COPD

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Cenk Kirakli, MD
1671 sok. No: 159 D: 5
TR–35530 Karsiyaka Izmir (Turkey)
Tel. +90 5052 35 2024, Fax +90 2324 58 7262
E-Mail ckirakli@hotmail.com

or severe acute cardiogenic pulmonary edema [3, 4]. Therefore, it is being increasingly used as first-choice ventilatory support in patients with AHRF [2–11].

Theoretically, NIV can be delivered using similar modalities and ventilators as those used with IMV. However, in the clinical setting this may not be feasible because of the lack of appropriate equipment for NIV, variability of ventilation modes and settings, and difficulty in providing a patient-ventilator synchrony. Moreover, the most appropriate ventilatory mode for NIV in AHRF has not been clearly established yet. Pressure support ventilation (PSV) with positive end-expiratory pressure (PEEP), a flow-cycled mode, is widely used in many centers but the presence of mask leaks engenders prolonged inspiratory times (T_{insp}), expiratory distress and eventually patient-ventilator dysynchrony, particularly in patients with COPD. Assisted pressure-controlled ventilation (APCV) is a time-cycled mode widely used in patients who basically are in need of invasive mechanical ventilation. The major advantages of APCV is the preferable decelerating flow pattern as seen in PSV and the opportunity for setting the T_{insp}. We hypothesized that if we could use APCV for NIV and set the T_{insp} to the maximum of 1 s, we probably would achieve a better patient-ventilator synchrony, leading to an effective CO_2 elimination despite mask leaks. On the other hand, the effect of IMV in producing deleterious changes in cardiac functions and hemodynamics was previously well established, but whether NIV (neither PSV nor APCV) similarly affects cardiac functions or not still remains controversial [12–14].

Therefore, we conducted a prospective randomized study to determine whether noninvasive APCV, a time-cycled mode, could be used as an alternative to PSV in COPD patients with AHRF who encounter expiratory distress due to mask leaks. Another aim of this study was to establish the hemodynamic effects of NIV in COPD patients and compare the 2 modes from this point of view.

Materials and Methods

Patient Selection and Inclusion Criteria

Thirty-four consecutive adult male COPD patients with respiratory failure requiring NIV support were included in this prospective randomized study approved by the institutional review board. Inclusion criteria were as follows:

1 diagnosis of COPD on the basis of clinical history, findings of physical examination, chest radiograph and/or previous pulmonary function test data, and receiving conventional medical treatment consisting of inhaled bronchodilators, systemic corticosteroids, antibiotics and supplemental oxygen therapy;
2 respiratory rate (RR) >24 breaths/min, partial arterial carbon dioxide pressure ($PaCO_2$) >50 mm Hg and respiratory acidosis (pH ≤7.35);
3 normal level of consciousness or moderate signs of respiratory encephalopathy (drowsiness, confusion, flapping tremor);
4 absence of pneumonia (as a cause of exacerbation), systemic diseases, cardiac instability, chest wall deformities and immediate need for endotracheal intubation;
5 written or informed consent obtained from the patients or their relatives.

Study Design

The study was initiated in the first 24 h of hospital admission. The neurologic status was assessed by the Glasgow Coma Scale and patients with a score of 10 or greater were selected for NIV. Patients were consecutively and randomly assigned to 2 groups for NIV ventilation, either APCV or PSV performed via the same ICU ventilator (PB-7200ae; Puritan-Bennett Corp., Carlsbad, Calif., USA) and mask for at least 1 h. Throughout the entire protocol, each patient received NIV in a semirecumbent position using a full-face bilevel positive airway pressure mask (KOO Medical Equipment Co. Ltd., Shanghai, China). Patients in whom NIV failed (who required endotracheal intubation in the first 1 h) were excluded from the study.

Ventilator Settings and Respiratory Data

Arterial oxygen saturation (SaO_2) and electrocardiogram (Life Scope Bsm-2301; Nihon Kohden Corp., Tokyo, Japan) were monitored continuously during the ventilation period. In APCV mode, inspiratory pressure levels (P_{insp}) were set to 15 cm H_2O at baseline and increased in increments of 2 cm H_2O to achieve an RR lower than 30 breaths/min. T_{insp} was set to a maximum of 1 s for APCV mode. Similarly, in PSV mode pressure support ($P_{support}$) levels were titrated according to the patient's tolerance, starting at 10 cm H_2O and increasing up to 16–20 cm H_2O. In order to compensate for mask leaks, a low level of PEEP (3–5 cm H_2O) was added in both modes. P_{insp} for PSV was defined as the sum of $P_{support}$ and PEEP. Inspired oxygen fraction (FiO_2) levels were titrated to obtain an SaO_2 greater than 90%. The flow trigger was used for inspiratory triggering in both modes and was set as low as possible, avoiding autotriggering. The humidifier was set off during the trial in order to decrease the workload needed to overcome the circuit resistance. Radial artery catheterization was performed in every patient in order to monitor the mean arterial pressure (MAP) and to allow arterial blood sampling. Arterial blood gas analysis results, RR, expired tidal volume (V_T) and minute ventilation (V_E) were recorded at baseline and after 1 h of NIV.

Hemodynamic and O_2 Transport Variables

Pulmonary capillary wedge pressure (PCWP), mean pulmonary artery pressure (MPAP), central venous pressure (CVP) measurements and mixed venous blood saturation (SvO_2) sampling was done through a 7F triple-lumen thermodilution balloon-tipped Swan-Ganz catheter (Edwards Swan Ganz®; Baxter Healthcare Corp., Irvine, Calif., USA) inserted into the internal jugular or subclavian vein by the Seldinger technique. MAP, MPAP, PCWP and CVP values were measured via a pressure transducer connected to the bedside monitor (Life Scope p BSM-4103; Nihon Kohden Corp.) and recorded at baseline and after 1 h of NIV. Cardiac output (CO) was determined by the thermo-

dilution technique as described before [15]. Three consecutive measurements were performed and mean values were recorded. Cardiac index [CI = CO(l/min)/BSA(m^2)], systemic vascular resistance index [SVRI = 80 × (MAP – CVP)/CI], pulmonary vascular resistance index [PVRI = 80 × (MPAP – PCWP)/CI], arterial oxygen content [CaO_2 = (0.0138 × Hgb × SaO_2) + 0.0031 × partial arterial oxygen pressure (PaO_2)], venous oxygen content [CvO_2 = (0.0138 × Hgb × SvO_2) + 0.0031 × PvO_2], oxygen delivery index (DO_2I = CaO_2 × CO ×10) and oxygen consumption index [VO_2I = ($C_{(a-v)}O_2$ × CI × 10)] values were recorded from the digital readout of a cardiac output monitor (Explorer; Baxter Edwards Critical Care, Irvine, Calif., USA) which establishes the calculations by using the standard formulas defined above.

Statistical Analysis

A software package (SPSS) was used for statistical analysis. Comparisons with baseline values within each group were done by the Wilcoxon signed-ranks test. The 2 NIV modes were compared with each other in terms of the changes in respiratory and hemodynamic variables (the amounts of increase or decrease in each group after the implementation of NIV) by the Mann-Whitney U test. A value of $p < 0.05$ was accepted as the level of statistical significance.

Results

Of the 34 patients included in the study, 7 patients were not evaluated because they could not tolerate NIV and were intubated orotracheally for IMV support. From those who failed, 2 from the PSV group and 1 from the PCV group showed claustrophobic symptoms and refused to use the mask. These patients underwent IMV support because of severe respiratory acidosis. The other 4 patients (2 each from the APCV and PSV groups) showed worsening hemodynamic and respiratory status (severe hypotension, arrhythmia, ineffective triggering and severe hypoxemia) and were intubated during the first hour of NIV. Demographic data of the remaining 27 patients and the mean pressures needed to achieve the target RR are shown in table 1. At baseline, there were no

Table 1. General clinical and demographic baseline data

	APCV (n = 14)	PSV (n = 13)	p
Age, years	61 ± 4	63 ± 5	NS
FEV_1, l	0.91 (33)	0.89 (30)	NS
FEV_1/FVC, %	43	41	NS
P_{insp}, cm H_2O	18.3 ± 2.6	18.9 ± 2.2	NS
PEEP, cm H_2O	4 ± 1	4 ± 1	NS

Figures in parentheses are percent predicted. FEV_1 = Forced expiratory volume at 1 s; FEV_1/FVC = forced expiratory volume at 1 s/forced vital capacity ratio.

Table 2. Respiratory and hemodynamic baseline data

	APCV (n = 14)	PSV (n = 13)	p
pH	7.29 (7.17–7.34)	7.32 (7.27–7.33)	0.44
$PaCO_2$, mm Hg	85 (61–114)	72 (65–99)	0.14
PaO_2/FiO_2	228 (139–315)	208 (189–236)	0.31
SaO_2, %	86 (74–98)	85 (72–94)	0.35
SvO_2, %	71 (33–83)	69 (59–79)	0.56
CO, l/min	6.8 (4.6–9.5)	6.3 (5.1–8.9)	0.77
CI, l/min/m^2	4 (2.7–5.5)	3.5 (2.8–5.1)	0.70
MAP, mm Hg	87 (71–110)	91 (71–105)	0.53
MPAP, mm Hg	38 (24–60)	31 (21–50)	0.38
PCWP, mm Hg	12 (5–17)	11.5 (7–16)	0.56
CVP, mm Hg	10 (7–16)	9.5 (6–16)	0.80
SVI ml/m^2/beat	36 (20–69)	45 (31–50)	0.56
SVRI, dyn.s/cm^5/m^2	1,527 (1,196–2,633)	1,884 (1,075–2,337)	0.38
PVRI, dyn.s/cm^5/m^2	432 (219–869)	414 (256–577)	0.56
CaO_2, ml/dl	17 (10–21)	16 (9–25)	0.56
CvO_2, ml/dl	14 (6–19)	13 (8–17)	0.92
DO_2I, ml/min/m^2	660 (336–872)	594 (394–855)	0.44
VO_2I, ml/min/m^2	127 (39–426)	101 (7–245)	0.44

Values are expressed as medians with ranges in parentheses. SVI = Stroke volume index.

Table 3. Respiratory parameters at baseline and after NIV

	APCV baseline	APCV after NIV	PSV baseline	PSV after NIV
V_E, l/min	9.3 (7.3–10.5)	11.6 (9.7–14.1)*	9.7 (7.8–11.4)	11.7 (9.4–14)$^+$
RR, breaths/min	30 (26–35)	23 (17–27)*	31 (27–38)	25 (19–30)$^+$
V_T, ml	318 (278–363)	547 (411–670)*	321 (281–383)	511 (389–641)$^+$

Values are expressed as medians with ranges in parentheses. * $p < 0.05$ compared with APCV baseline; $^+$ $p < 0.05$ compared with PSV baseline.

Table 4. Median changes in the respiratory and hemodynamic variables after 1 h of NIV

	APCV (n = 14)	PSV (n = 13)	p
pH	0.07 (0.29 to –0.01)	0.02 (0.17 to –0.03)	0.53
$PaCO_2$, mm Hg	–13.2 (–2 to –46)	–4.5 (12 to –18)	0.12
PaO_2/FiO_2	38 (77 to –29)	10 (133 to –48)	0.77
SaO_2, %	4 (22 to –5)	10 (25 to –4)	0.31
CO, l/min	–1.31 (0.17 to –4.19)	–1.47 (–0.83 to –2.86)	0.84
CI, l/min/m^2	–0.82 (0.1 to –2.3)	–0.87 (–0.4 to –1.7)	0.92
SvO_2, %	–2 (16 to –33)	–0.5 (11 to –11)	0.96
MAP, mm Hg	0 (3 to –14)	–4.5 (16 to –19)	0.62
PCWP, mm Hg	6 (7 to –7)	1 (7 to –10)	0.56
MPAP, mm Hg	–1 (6 to –10)	1 (9 to –7)	0.33
CVP, mm Hg	2 (4 to –3)	3 (7 to –3)	0.11
SVI, ml/m^2/beat	–8.6 (0.9 to –19.3)	–5.6 (–2 to –17.4)	0.87
SVRI, dyn.s/cm^5/m^2	314 (2,167 to –265)	300 (616 to –102)	0.71
PVRI, dyn.s/cm^5/m^2	96 (240 to –110)	139 (379 to –29)	0.31
CaO_2, ml/dl	0.87 (5.6 to –0.8)	2.1 (4.4 to 0.7)	0.26
CvO_2, ml/dl	0.59 (7.41 to –7.6)	–0.46 (3.3 to 1.54)	0.56
DO_2I, ml/min/m^2	–67 (165 to –281)	–49 (21 to –196)	0.56
VO_2I, ml/min/m^2	38 (144 to –147)	32 (135 to –51)	0.56

Data are calculated by the extraction of pre-NIV values from post-NIV values and are expressed as medians with ranges in parentheses. SVI = Stroke volume index.

significant differences between the APCV and PSV groups regarding the respiratory, hemodynamic and O_2 transport variables (table 2).

Respiratory Variables

When compared with baseline, NIV provided a significant decrease in RR and increase in V_T and V_E ($p < 0.05$) within each group (table 3), but these changes (median changes after NIV) did not differ between the 2 groups. There were significant improvements in pH and $PaCO_2$ values in the APCV group ($p < 0.05$ and $p < 0.01$, respectively; fig. 1). The improvements in SaO_2 were also significant within each group when compared with baseline ($p < 0.05$) but showed no difference between PSV and APCV (table 4). We also observed an improvement in the PaO_2/FiO_2 ratio in both groups, but this was not statistically significant when compared with baseline values; the improvement rates did not differ between the 2 groups (fig. 1; table 4).

Hemodynamic and O_2 Transport Variables

NIV resulted in significant falls in the CO, CI and stroke volume index (SVI) levels within each group compared with the baseline values ($p < 0.05$; fig. 2). The amounts of decrease were similar when the 2 modes were compared with each other (table 4). These alterations in

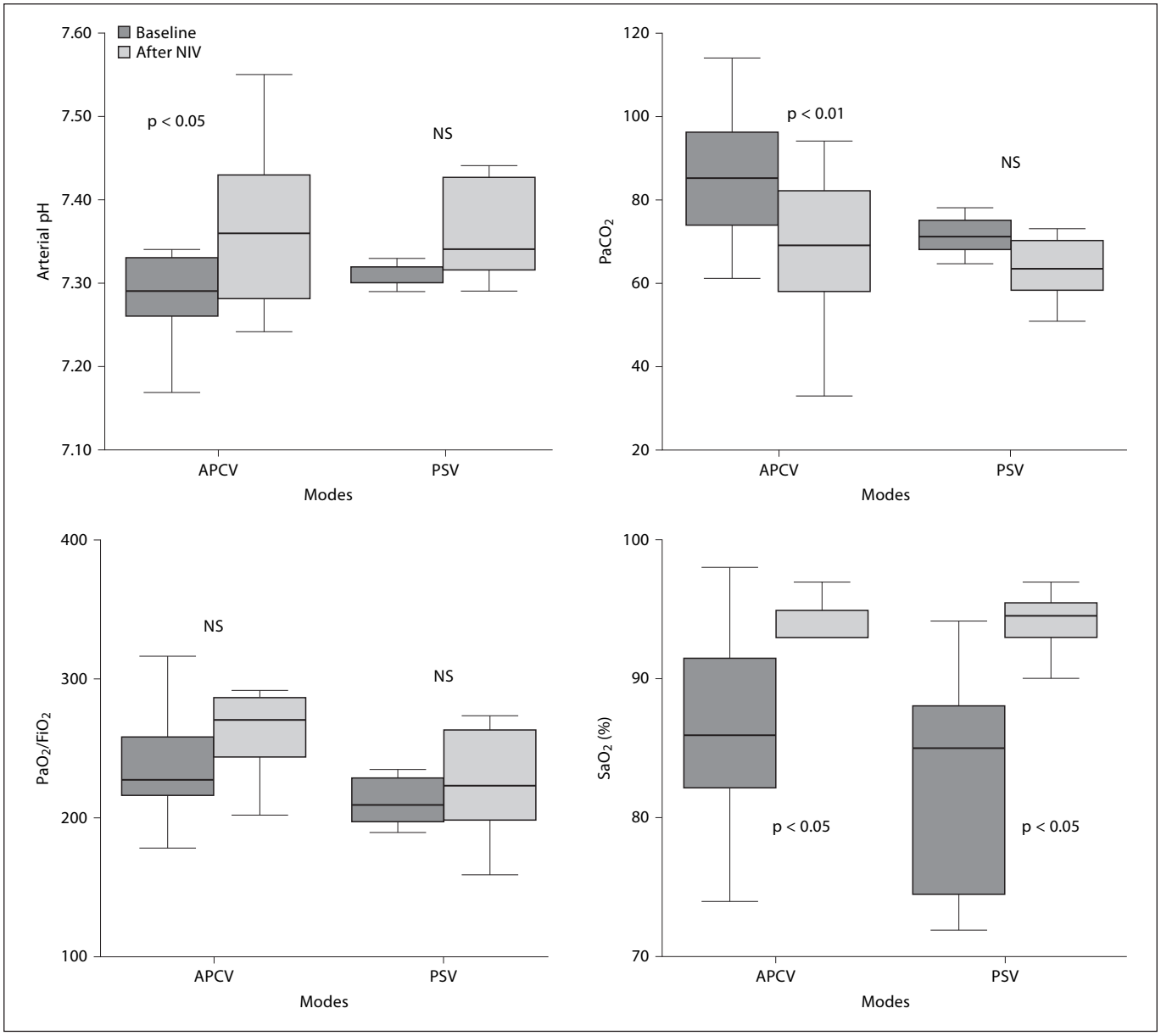

Fig. 1. Box plots of the respiratory parameters at baseline and after 1 h of NIV for each mode. The line in the middle of the box indicates the median, the upper and lower end of the box indicate the upper and lower quartile, and the upper and lower end of the error bar indicate the maximum and minimum. NS = Not significant.

cardiac functions did not lead to any significant change in systemic arterial pressures. In the PSV group, both CVP and SVRI levels increased significantly compared to baseline, while in the APCV group only SVRI levels did so ($p < 0.05$; fig. 2). CaO_2 also increased after 1 h of NIV in both groups when compared with the baseline levels ($p < 0.05$), but these data were comparable between the 2 groups (fig. 2; table 4).

Discussion

This prospective randomized study suggests that the time-cycled APCV mode can be as effective and safe as PSV in eliminating CO_2 and improving pH with comparable cardiac side effects in COPD patients with expiratory distress.

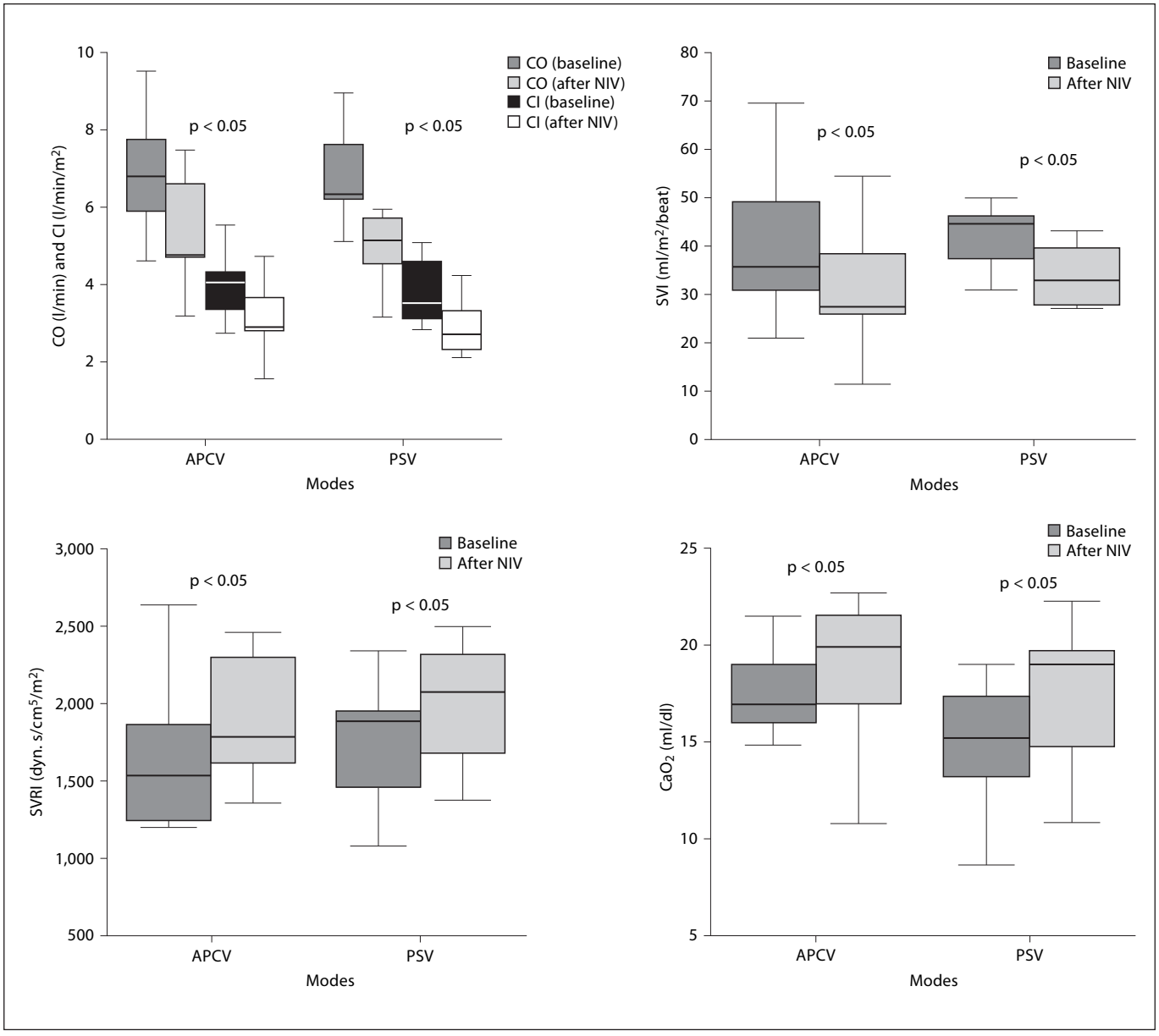

Fig. 2. Box plots of the hemodynamic and O_2 transport parameters at baseline and after 1 h of NIV for each mode. The line in the middle of the box indicates the median, the upper and lower end of the box indicate the upper and lower quartile, and the upper and lower end of the error bar indicate the maximum and minimum.

In the literature, there are few prospective randomized studies comparing the ventilation modes used for NIV. Vitacca et al. [16] performed the first prospective randomized trial that compared the clinical usefulness of both PSV and assisted volume-controlled ventilation in 29 patients, and reported no significant clinical difference in terms of the success rate between the 2 NIV modes. However, a better patient compliance with fewer side effects was found in PSV than in assisted volume-controlled ventilation. In another study, Girault et al. [17] compared the respiratory physiological effects and comfort with assisted volume-controlled ventilation and PSV performed via nasal mask in AHRF. They reported that both ventilation modes provided respiratory muscle rest

and similarly improved breathing pattern and gas exchange. They also observed that the inspiratory effort was lower during assisted volume-controlled ventilation than during PSV but patients, interestingly, felt more comfortable with PSV. The common finding of these 2 studies was the better tolerance with PSV owing to the lower peak pressures occurring in the mask. These findings suggest that COPD patients can presumably tolerate pressure-targeted modes much better than volume-targeted modes.

The underlying physiological mechanisms by which NIV provides an improvement in arterial blood gases and pH levels are still not well established. Diaz et al. [13] performed the only clinical trial investigating the short-term effects of NIV on pulmonary gas exchange and hemodynamics in COPD patients with AHRF. They concluded that the improvements in $PaCO_2$ and PaO_2 levels after 15 and 30 min of NIV were found to be associated with the increase in overall alveolar ventilation (resulting from decrease in RR and increase in V_T). Moreover, the authors also concluded that the total gas exchange capacity of the lungs remained unchanged since NIV did not result in recruitment of nonventilated or poorly ventilated alveoli. Thus, implementation of high pressure-support levels in order to provide alveolar recruitment was not useful as a short-term procedure, at least for this group of patients [13]. All of this available information suggests that the most important goal of NIV is to improve the respiratory pattern by increasing the alveolar ventilation and V_E [18].

In PSV, after the patient triggers the inspiration, the ventilator provides a gas flow according to the preset pressure and then cycles into expiration when the inspiratory flow decays below 25% of the initial peak flow or to an absolute flow of 5 liters/min as in the ventilator we used for this study. Most ventilators have a backup means of terminating the inspiratory phase such as an overpressure of 1.5 cm H_2O, or if the total T_{insp} exceeds 3 s or 80% of 1 breathe cycle [19]. In addition to T_{insp}, RR, inspiratory flow, V_T and V_E are also determined by the patient. One specific problem in the implementation of NIV with PSV mode is the difficulty in terminating the inspiratory phase in case of leaks occurring in the mask. This problem causes a prolonged T_{insp}, leading to a difficulty in cycling into expiration, to auto-PEEP and eventually to patient-ventilator dysynchrony. APCV, a time-cycled mode, gives the opportunity to set the T_{insp} so that each breath can last equally. Consequently, when T_{insp} is set to a duration of about 1 s, a better patient-ventilator synchrony can be achieved especially in hypercapnic COPD patients who are in need of high inspiratory flow rates due to expiratory distress.

In accordance with the results of several studies published previously [2–12, 16, 17], we observed significant improvements in breathing patterns after NIV. These improvements were probably due to the reduction in RR and increase in V_T which led to an enhancement in alveolar ventilation and V_E as reported in the study by Diaz et al. [13]. The median increase in V_E was 2.3 liters in the APCV group and 2 liters in the PSV group, showing no statistically significant difference. Both the APCV and PSV groups showed similar improvements in pH and $PaCO_2$ levels. The significance observed within the APCV group might be due to the greater number of patients with higher $PaCO_2$ levels, who can have a greater response to therapy. Another possibility may be the easier cycling into the expiratory phase, avoiding auto-PEEP and providing better patient-ventilator synchrony in APCV, since the increases in V_E were comparable in both groups. In PSV mode, we observed severe prolonged T_{insp} up to 3 s and backup termination of the inspiratory phase leading to expiratory distress, especially when mask leaks occurred.

Hemodynamic effects of positive pressure ventilation performed either with a mask or through an endotracheal tube can vary according to the fluid status and cardiac functions of the patient [20, 21]. The most important hemodynamic alteration seen in patients with normal cardiac functions is the fall in cardiac output in relation with the decrease in ventricular preload and venous return. Nevertheless, NIV is generally tolerated much better than IMV, owing to lower inflation pressures. There are also some data supporting the beneficial effects of NIV in COPD patients with mild arrhythmias [22]. All in all, every mechanical ventilation procedure must be performed very carefully, especially in patients with hemodynamic instability or accompanying cardiac disease. In a randomized controlled trial in patients with acute cardiogenic pulmonary edema, after the implementation of bilevel ventilation, Summers et al. [14] reported an increased risk of myocardial infarction due to early systemic hypotension. Diaz et al. [13] have also confirmed that NIV causes a significant decrease in CO without a change in SvO_2 in COPD patients with acute respiratory failure. The negative effect of the decrease in CO on SvO_2 is probably compensated by the increase in the total ventilation/perfusion ratio of the lungs [21].

We similarly observed significant decreases in CO, CI and SVI without any significant change in SvO_2 levels. There were no significant differences in the decrease

rates of these variables when APCV and PSV groups were compared with each other. These findings were probably related to not only the decrease in venous return but also the decrease in oxygen consumption and work of breathing, because the patients enrolled in the study did not have any primary cardiac pathology. Since the changes in MAP and CVP were not as evident as the fall in CO, the increase in SVRI can also be assumed to be a direct result of the significant fall in CO and CI.

NIV resulted in a significant increase in SaO_2 in both the APCV and PSV groups. The PaO_2/FiO_2 ratio also increased, but this change was not significant. The increase in CaO_2 was possibly due to the augmentation of SaO_2, regardless of the unchanged hemoglobin values of the patients.

To our knowledge, there are no published clinical or physiological data concerning the specific effects of NIV on oxygen delivery and consumption. Only Diaz et al. [13] reported a significant decrease in oxygen delivery after 15 and 30 min of NIV in COPD patients with AHRF. Our results also showed a moderate but insignificant decrease in oxygen delivery after NIV. The effect of positive pressure ventilation on oxygen delivery is expected to be an increase due to the increase in the CaO_2. However, since oxygen delivery is also dependent on CO, this decrease may be explained by the significant fall in CO and CI after NIV.

There are some limitations and weaknesses in our study that are worth noting. APCV seems to be more effective than PSV in some patient groups in terms of eliminating CO_2, but we do not have any specific data available to support this assumption. Although baseline $PaCO_2$ and pH levels were not significantly different between the 2 groups, there were more hypercapnic patients in the APCV group that might have had a greater response to therapy. Moreover, due to technical limitations we did not have the chance to monitor the T_{insp} for each patient in PSV and leak volumes for either mode or any better indices of energy expenditure and respiratory muscle activity (such as esophageal pressure, the pressure time product, work of breathing or diaphragmatic electromyogram). Also, some of the statistical data could probably be more informative if the sample size of our study was bigger. Further studies in patients with more similar $PaCO_2$ and pH values are needed to clarify these hypotheses and obtain useful clinical indications about the choice of ventilation modality.

From our results, it can be concluded that APCV can be an alternative mode to PSV in COPD patients who are in need of NIV due to AHRF. Patients who require higher inspiratory flow rates might cycle into expiration more easily and manage a more effective CO_2 elimination under APCV in the case of mask leaks, since it is a time-cycled mode. Moreover, its hemodynamic side effects do not differ considerably from those of PSV, although it is an assisted controlled mode of ventilation. Larger randomized prospective trials are needed to establish the short- and long-term effects and advantages of the noninvasive APCV mode in acute hypercapnic COPD patients.

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