REUE | Original Article

Correlation between tidal volume and Pco_2 clearance in patients under noninvasive mechanical ventilation

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OBJECTIVE. To analyze the relation between tidal volume (V_t) and alveolar ventilation measured by changes in Pco_2 in patients on noninvasive mechanical ventilation (NIV).

METHODS. Prospective observational study of patients treated in the emergency department of Hospital General Universitario Reina Sofia. We recruited patients with hypercapnic respiratory insufficiency (indicated by a ratio of Pco_2 to fraction of inspired oxygen [Fio_2] < 300, pH < 7.35, and Pco_2 > 45 mmHg) for whom NIV was indicated. Variables were recorded at the start of NIV and after 1 and 3 hours.

RESULTS. Ninety-two patients with a mean (SD) age of 77.67 (10.86) years were included. We observed significant decreases in Pco_2 levels over time, from 72.8 (15.06) mmHg at baseline to 68.1 (18.9) mmHg at 1 hour and to 66.4 (17.45) mmHg at 3 hours (P = .001). Mean Pco_2 clearance went from 4.73 (12.4) mmHg/h at 1 hour to 1.68 (8.4) mmHg/h at 3 hours. V_t increased from 400.64 (143.22) mL at 1 hour to 430.81 (156.27) mL at 3 hours. The relation between the V_t calculated per ideal body weight (IBW) and Pco_2 at 1 hour was significant at 0.309 (P = .003). Initial Fio_2, initial expiratory positive airway pressure, and V_t per IBW at 1 hour were significant predictors according to logistic log regression (R^2, 0.458; P < .001).

CONCLUSIONS. The increase in V_t per IBW in patients with acute hypercapnic respiratory failure under NIV shows a weak to moderate positive correlation with alveolar ventilation measured by means of Pco_2 clearance.

Key words: Noninvasive ventilation. Acute hypercapnic respiratory failure. Tidal volume. Pulmonary ventilation.

Correlación entre el volumen tidal y la tasa de aclaramiento de pCO_2 en los pacientes sometidos a ventilación mecánica no invasiva

OBJETIVO. Analizar la relación entre el volumen tidal (Vt) y la ventilación alveolar, medida a través del cambio en la pCO_2, en pacientes sometidos a ventilación mecánica no invasiva (VMNI).

MÉTODOS. Estudio observacional prospectivo realizado en el servicio de urgencias del Hospital General Universitario Reina Sofía. Se incluyeron pacientes con diagnóstico de insuficiencia respiratoria hipercápnica (pO_2/FiO_2 < 300, pH < 7,35 y pCO_2 > 45 mm Hg) con indicación de VMNI. Se estableció un seguimiento al inicio, 1ª y 3ª hora.

RESULTADOS. Se incluyeron 92 pacientes. La media de edad fue de 77,67 ± 10,86 años. Los niveles de la pCO_2 descendieron progresivamente: 72,8 ± 15,06 mm Hg basal, 68,1 ± 18,9 mm Hg a la hora y 66,4 ± 17,45 mm Hg a las 3 horas (p = 0,001). Media de aclaramiento de pCO_2: 4,73 ± 12,4 mm Hg en la 1ª hora y de 1,68 ± 8,4 mm Hg/hora a las 3 horas. El Vt aumentó de 400,64 ± 143,22 ml en la primera hora a 430,81 ± 156,27 ml en la 3ª hora. La relación entre el Vt por peso ideal y el aclaramiento de la pCO_2 en la 1ª hora fue de 0,309 (p = 0,003). Las variables independientes predictoras para el aclaramiento de pCO_2 en la 1ª hora fueron la FiO_2 inicial, la presión positiva espiratoria en vía aérea (EPAP) inicial, Vt por peso ideal y la EPAP a la hora (R^2: 0,458; p < 0,001).

CONCLUSIONES. Un aumento en el Vt por peso ideal, en los pacientes con insuficiencia respiratoria hipercápnica sometidos a VMNI, presenta una correlación positiva débil-moderada, en la ventilación alveolar, medida a través del aclaramiento de pCO_2.

Palabras clave: Ventilación mecánica no invasiva. Insuficiencia respiratoria hipercápnica. Volumen tidal. Ventilación pulmonar.

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Introduction

Acute respiratory failure (ARF) is one of the most common medical problems encountered in emergency departments (EDs).[1,2] ARF is not a disease per se, but rather the final common consequence of a wide variety of specific conditions, not only of respiratory origin but also cardiologic, neurologic, toxic, and traumatic.

Classically, ARF is defined as:[2-4]

– Partial respiratory failure (type I or hypoxemic): arterial pO_2 < 60 mm Hg while breathing room air (FiO_2 21%), at rest and at sea level.[4,5]

– Global respiratory failure (type II or hypercapnic ARF): in addition to the previous criterion, arterial blood gas values must include elevated pCO_2 (≥ 45 mm Hg) and low pH (< 7.35).

Focusing on ARF, one frequent cause is alveolar hypoventilation. Alveolar ventilation (VA) is the difference between minute ventilation (tidal volume – Vt – × respiratory rate) and dead space ventilation, with Vt being the most relevant factor. Thus, an increase in Vt leads to an increase in VA and consequently a decrease in arterial pCO_2.[6,7]

In emergency medicine—both in prehospital and hospital settings—noninvasive mechanical ventilation (NIV) has become the treatment of choice for respiratory diseases presenting with hypercapnic ARF, particularly in severe exacerbations of chronic obstructive pulmonary disease (COPD),[6,8,9] reducing mortality and orotracheal intubation rates.[6,8,10-12]

In general, NIV uses pressure-targeted, spontaneous modes in which the difference between inspiratory and expiratory pressure—called pressure support (PS)—is directly proportional to Vt. Clinical practice guidelines recommend starting with low PS levels and increasing them progressively according to patient tolerance while ensuring a Vt between 6–8 mL/kg. However, these Vt values have been extrapolated from invasive mechanical ventilation (IMV).[8,12-14] Currently, no studies focused specifically on NIV have evaluated the true effectiveness of Vt.

The objective of this study was to analyze the relationship between Vt in patients undergoing NIV and VA as measured by change in pCO_2. Our hypothesis was that patients with hypercapnic ARF treated with NIV would show a greater decrease in pCO_2 when Vt is higher.

Method

We designed a prospective observational analytical study carried out in the ED of Hospital General Universitario Reina Sofía (HGURS) in Murcia (Spain), with a reference population of 202,000 inhabitants. The study was conducted from January 18th, 2015 through and May 3rd, 2017.

This study adhered to current regulations governing research projects and fully complied with the ethical principles of the Declaration of Helsinki and Good Clinical Practice guidelines. It was also approved by the Clinical Research Ethics Committee of HGURS.

We used a non-probabilistic convenience sampling strategy, consecutively including all patients older than 18 years diagnosed with hypercapnic ARF (defined as blood gas values with pO_2/FiO_2 < 300, pH < 7.35, and pCO_2 > 45 mm Hg) and with an indication for NIV in the ED. Exclusion criteria were:

– Need for urgent orotracheal intubation.

– Extreme dyspnea, exhaustion, or respiratory fatigue (paradoxical abdominal breathing).

– Lack of cooperation or severe agitation.

– Pneumothorax.

– Patients undergoing renal replacement therapy.

– Patients with uncontrolled air leaks.

– Glasgow Coma Scale < 8.

– Use of a ventilator other than the Trilogy 202 Ventilator (Philips Respironics®).

Clinical, analytical, diagnostic, and therapeutic data collected during the care process were obtained through a specific form and the electronic ED health record. Ventilator data (FiO_2, inspiratory positive airway pressure —IPAP—, expiratory positive airway pressure —EPAP—, PS, and Vt) were obtained from the Trilogy 202 Ventilator (Philips Respironics®). The ventilatory mode used was S/T.[15] According to institutional protocol, an oronasal interface with a passive circuit was used, air leak control was set at < 15 L/min, backup respiratory rate between 12–14 breaths/min, and inspiratory time of 1.2 seconds. Ventilator parameters were programmed by the attending physician based on clinical criteria and institutional protocol. Ideal body weight was calculated using the Lorentz formula, one of the most widely used methods because it incorporates height and sex as corrective factors.[16,17]

Once NIV was initiated, baseline clinical and analytical variables were recorded. Patients were monitored during the first 3 hours of NIV (time points at 1 hour and 3 hours). Clinical, analytical, and blood gas variables were collected at 1 hour and 3 hours. The Vt at the first hour was calculated as the mean of all Vt measurements between the start and the end of the first hour of NIV. The Vt between the second and third hour was calculated as the mean of all Vt measurements between the beginning of the second hour and the end of the third hour. To better interpret Vt, it was analyzed globally (unadjusted), adjusted to ideal body weight, and adjusted to actual body weight.

VA was measured by the hourly pCO_2 clearance rate. The first-hour analysis was the difference between pCO_2 at the first hour and baseline. The clearance rate at the third hour was calculated as the difference between pCO_2 at the third hour and at the first hour, divided by 2.

For sample size estimation, an 80% power, a type I error of 0.05, and a minimum correlation of 0.7 were considered, resulting in a required final sample size of 75 patients.

The statistical analysis was performed using IBM SPSS Statistics, version 20. Qualitative variables were analyzed using frequency distributions with percentages, and for quantitative variables the mean, standard deviation, standard error, median, and interquartile range were calculated. To assess the association between qualitative variables, the chi-square test was used when more than 75% of expected frequencies were greater than 5, or Fisher's exact test when low cell counts recommended it; risk was evaluated using contingen-

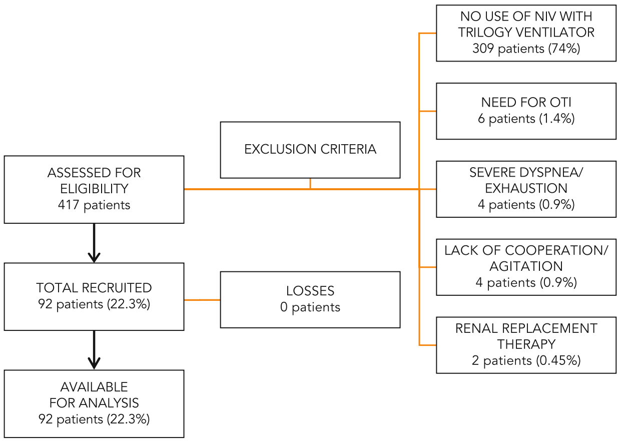

Figure 1. Flow diagram of patients included in the study. OTI: orotracheal intubation.

cy tables. Normality of quantitative variables was assessed using the Kolmogorov–Smirnov test when > 50 observations were available, or the Shapiro–Wilk test when < 50 observations were available. Means were compared using the Student t test when quantitative variables were normally distributed, or the Mann–Whitney U test when distributions were non-parametric. Similarly, to determine correlation between quantitative variables, Pearson correlation was used for normally distributed variables, and Spearman correlation for the remainder. To identify real predictor variables of pCO_2 clearance, a simple linear regression model was used. All factors with $P < .10$ in univariate analysis were included, along with variables that could be related to the model, such as pressure support (PS) and age. A value of $P < .05$ was considered statistically significant.

Results

A total of 417 patients were screened for eligibility. Of these, 309 (74.1%) were excluded due to the use of a ventilator other than the Trilogy 202 Ventilator, 6 (1.4%) due to the need for urgent orotracheal intubation, 4 (0.9%) due to extreme dyspnea or exhaustion, 4 (0.9%) due to agitation or lack of cooperation, and 2 (0.45%) because they were undergoing renal replacement therapy. Finally, 92 patients (22.3%) were enrolled. No patient was lost to follow-up during the study period (Figure 1).

Table 1 illustrates the descriptive analysis of the baseline variables. The patients' mean age was 77.67 ± 10.86 years; 50 (54.3%) were men and 42 (45.7%) were women. A total of 37% were on chronic home oxygen therapy. The most frequent diagnosis was acute pulmonary edema (52.2%), followed by COPD exacerbation (33.7%). A total of 20.7%of patients had less common diagnoses. Regarding treatments administered in the ED, the most widely used were inhaled ipratropium bromide (74; 80.2%), systemic corticosteroids (71; 76.9%), and inhaled salbutamol (69; 74.7%).

Table 2 illustrates the variation in blood gas values and ventilatory parameters over time.

During the first hour, a drop in pCO_2 was observed, from a baseline mean of 72.8 ± 15.06 mm Hg down to 68.1 ± 18.9 mm Hg ($P = 0.001$). The mean pCO_2 clearance within the first hour was 4.73 ± 12.4 mm Hg. The mean Vt during the first hour was 400.64 ± 143.22 mL. The correlation between pCO_2 clearance and Vt was 0.319 ($P = .002$), 0.309 ($P = .003$), and 0.327 ($P = .001$) for the global value, adjusted to ideal weight, and adjusted to actual weight, respectively (Figure 2).

At 3 hours, the mean pCO_2 was 66.4 ± 17.45 mm Hg ($P = .02$ between hour 1 and hour 3). During this 2-hour interval, the mean reduction in pCO_2 was 3.36 ± 16.8 mm Hg, resulting in a pCO_2 clearance rate of 1.68 ± 8.4 mm Hg per hour. The mean Vt at 3 hours was 430.81 ± 156.27

Table 1. Baseline distribution of patients

Demographic and clinical data	N = 92 n (%)
Age, years [Mean (SD)]	77.67 (10.86)
Male sex	50 (54.3)
COPD	37 (40.2)
Hypoventilation syndrome	26 (28.3)
Home oxygen therapy	34 (37.0)
CPAP	14 (15.2)
BiPAP	9 (9.8)
Height (cm) [Mean (SD)]	159.5 (9.4)
Actual weight (kg) [Median (IQR)]	76.5 (70.0-86.0)
Ideal weight (kg) [Median (IQR)]	57.5 (51.8-62.0)
BMI [Median (IQR)]	30.67 (26.66-33.7)
SBP (mmHg) [Mean (SD)]	131.3 (25.2)
DBP (mmHg) [Mean (SD)]	70.0 (14.3)
HR (bpm) [Median (IQR)]	81.0 (70.2-95.0)
RR (rpm) [Median (IQR)]	20.0 (17.0-24.0)
Temperature (°C) [Median (IQR)]	36.4 (36.1-36.7)
Sat O_2/FiO_2 [Mean (SD)]	283.9 (88.5)
pO_2/FiO_2 [Mean (SD)]	145.5 (127.3)
Glasgow Scale [Median (IQR)]	15.0 (14.0-15.0)
Creatinine (mg/dL) [Median (IQR)]	1.17 (0.82-1.48)
ProBNP (pg/mL) [Median (IQR)]	2442.0 (518.5-6901)
Main diagnosis	
COPD exacerbation	31 (33.7)
Acute pulmonary edema	48 (52.2)
Other diagnoses	19 (20.7)
Intrinsic asthma	4 (4.3)
Restrictive lung disease	3 (3.3)
BZD/opioid intoxication	3 (3.3)
Community-acquired pneumonia	2 (2.2)
Bronchoaspiration	2 (2.2)
Obesity–hypoventilation syndrome	2.02 (2.2)
Bronchiectasis	1 (1.1)
CO intoxication	1 (1.1)
Acute respiratory distress syndrome	1 (1.1)
Treatments administered in the emergency department	
Ipratropium bromide	74 (80.2)
Systemic corticosteroids	71 (76.9)
Salbutamol	69 (74.7)
Furosemide	58 (63.0)
Nebulized corticosteroids	51 (54.9)
Morphine chloride	25 (27.2)
Nitroglycerin infusion	14 (15.2)

COPD: chronic obstructive pulmonary disease; CPAP: continuous positive airway pressure; BiPAP: bilevel positive airway pressure; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate; RR: respiratory rate; Sat O_2: oxygen saturation; FiO_2: fraction of inspired oxygen; pO_2: arterial oxygen pressure; ProBNP: pro–B-type natriuretic peptide; BZD: benzodiazepines; CO: carbon monoxide; SD: standard deviation; IQR: interquartile range.

Table 2. Blood gas values and ventilatory parameters

Blood gas and ventilatory variables	Baseline [Mean (SD)]	> 1 h of NIV [Mean (SD)]	*P* (Baseline vs. 1 h)	> 3 h of NIV [Mean (SD)]	*P* (1 h vs 3 h)
pH	7.24 (0.075)	7.28 (0.07)	< .001	7.29 (0.07)	.002
pCO_2 (mmHg)	72.8 (15.06)	68.1 (18.9)	.001	66.4 (17.45)	.02
HCO_3^- (mmol/L)	30.36 (6.20)	30.96 (6.06)	.100	31.17 (6.03)	.255
IPAP (cm H_2O)	16.5 (1.47)	17.0 (1.55)	< .001	17.48 (1.89)	.02
EPAP (cm H_2O)	6.14 (1.12)	6.30 (1.17)	.010	6.47 (1.22)	.054
PS (cm H_2O)	10.35 (1.08)	10.69 (1.17)	.010	11.0 (1.46)	.110
FiO_2 (%)	35.9 (12.66)	33.27 (7.76)	.001	31.5 (6.8)	.001
Vt (ml)	–	400.64 (143.2)	–	430.81 (156.2)	< .001
Vt/PI (ml)	–	6.98 (2.24)	–	7.54 (2.57)	< .001
Vt/PR (ml)	–	5.26 (2.19)	–	5.66 (2.36)	< .001

NIV: non-invasive mechanical ventilation; SD: standard deviation; IPAP: inspiratory positive airway pressure; EPAP: expiratory positive airway pressure; PS: pressure support; FiO_2: fraction of inspired oxygen; Vt: tidal volume; IBW: ideal body weight; ABW: actual body weight.

mL. The correlation between pCO_2 clearance and Vt from hour 1 to hour 3 was 0.097 (P = .355), 0.127 (P = .227), and 0.126 (P = .233) for the global value, ideal weight, and actual weight, respectively.

During the first hour, the correlations between IPAP, EPAP, PS, and FiO_2 and global Vt were –0.101 (P = .588), –0.196 (P = .290), 0.163 (P = .380), and –0.335 (P = .066), respectively. Between hour 1 and hour 3, the Spearman rho values were –0.173 (P = .351), –0.065 (P = .730), –0.160 (P = .389), and –0.237 (P = .199) for IPAP, EPAP, PS, and FiO_2, respectively (Table 3).

Finally, the predictors of pCO_2 clearance during the first hour that remained significant in the simple linear regression model were initial FiO_2, initial EPAP, Vt adjusted to ideal weight, and EPAP at 1 hour, with an R^2 of 0.458 (P < .001) (Table 4).

Discussion

The use of NIV in the management of hypercapnic ARF is common practice in emergency departments (EDs), aiming to increase AV.[13] By analogy with IMV, Vt is the main respiratory parameter for improving AV.6,7 Our study focuses on patients frequently treated in EDs and is the

Table 3. Relationship between tidal volume and ventilatory parameters

Variable	Spearman Rho	*P*	Variable	Spearman Rho	*P*
IPAP at 1 hour (cm H_2O)	–0.101	.588	IPAP 3 h (cm H_2O)	–0.173	.351
EPAP at 1 hour (cm H_2O)	–0.196	.290	EPAP 3 h (cm H_2O)	–0.065	.730
PS at 1 hour (cm H_2O)	0.163	.380	PS 3 h (cm H_2O)	–0.160	.389
FiO_2 at 1 hour (%)	–0.335	.066	FiO_2 3 horas (%)	–0.237	.199

NIV: non-invasive mechanical ventilation; SD: standard deviation; IPAP: inspiratory positive airway pressure; EPAP: expiratory positive airway pressure; PS: pressure support; FiO_2: fraction of inspired oxygen; Vt: tidal volume; IBW: ideal body weight; ABW: actual body weight.

first to analyze the relationship between Vt in patients undergoing NIV and AV. Our results confirm the importance of Vt in NIV for improving AV. However, the correlations identified were weak to moderate. Therefore, a discrepancy exists between the relevance of Vt in IMV and its role in NIV.

In IMV, the most widely used mode is volume-controlled ventilation. Thus, the interaction between the ventilator and the patient depends only on the programmed settings, airway resistance, and pulmonary compliance. In Emergency and Critical Care, it is common to maintain diaphragmatic relaxation to avoid interference with the programmed respiratory pattern.[14]

In NIV, the approach is essentially the opposite. By definition, the patient is in spontaneous ventilation, so the interaction between the patient and the ventilator is more complex and may even lead to failure of the technique due to poor patient–ventilator synchrony. Furthermore, NIV incorporates a clearly differentiating factor—the use of a noninvasive interface—which introduces varying degrees of system leak.[8,11,13] This patient–ventilator interaction, together with leak, likely contributes to the differences observed between Vt in IMV and Vt in NIV.

This divergence may also be associated with 3 specific aspects of NIV in relation to Vt. First, Vt is not directly set; it is the result of the programmed PS.[8,11] Second, because the patient is breathing spontaneously, Vt varies with each respiratory cycle. Third, in NIV the measurement of Vt is taken during expiration, unlike IMV.

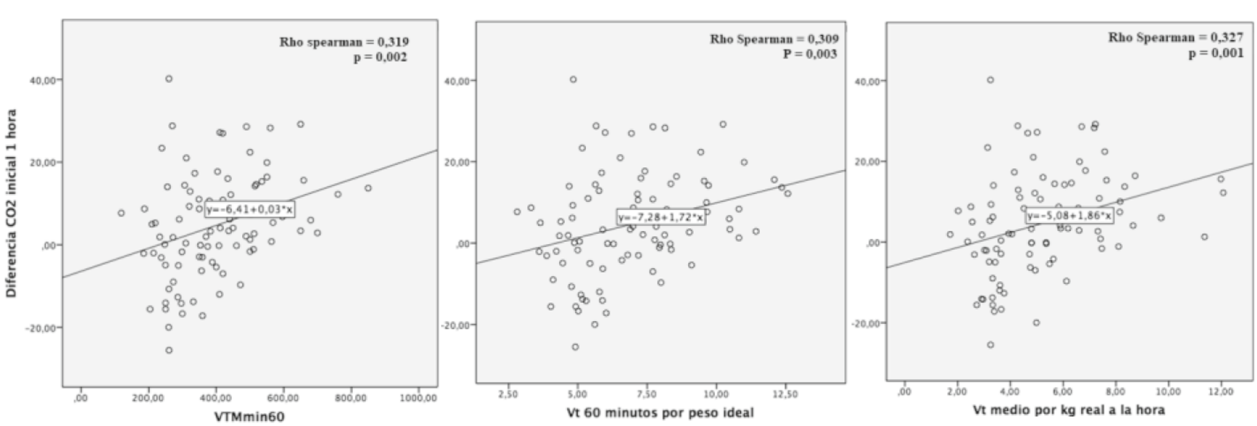

Figure 2. Relationship between tidal volume and pCO_2 clearance rate.
VTMmin60: tidal volume at 1 hour; Vt: tidal volume. Initial CO_2 difference at 1 hour: difference between pCO_2 at the first hour and baseline pCO_2.

Table 4. Simple linear regression for pCO_2 clearance and ventilatory parameters

Variable	Beta	*P*	95% CI for Beta	
Constant	–4.968	.545	–21.229	11.294
Initial FiO_2 (%)	–0.230	.021	–0.425	–0.035
Initial EPAP (cm H_2O)	5.749	.008	1.544	9.954
Vt/IBW at 1 hour (mL)	1.472	.008	0.390	2.554
EPAP at 1 hour (cm H_2O)	–4.378	.034	–8.419	–0.337

FiO_2: fraction of inspired oxygen; EPAP: expiratory positive airway pressure; Vt: tidal volume; IBW: ideal body weight.

In our study, no significant correlation was observed between PS and Vt during any time period. This finding challenges the classic association "higher PS –> higher Vt,"[7,14] which forms the basis of volume-assured pressure support modes such as AVAPS (average-volume-assured pressure support).[18] Tuggey *et al.*[19] titrated PS in patients with chronic respiratory failure and found an increase in minute ventilation when PS increased; however, this increase was not linear. With PS values < 10 cm H_2O, minute ventilation rose noticeably. With PS between 10 and 15 cm H_2O, the increase was smaller and even negligible in patients with COPD. With PS >20 cm H_2O, minute ventilation again increased significantly, but leak also increased, thus limiting effectiveness. In our study, PS values ranged between 10 and 12 cm H_2O. Therefore, a "minimum PS" likely is needed to achieve an adequate Vt and reduce the work of breathing. Our findings do not contradict the use of bilevel pressure modes but indicate that a PS between 6 and 10 cm H_2O is required.

In our view, other ventilatory factors—either individually or in combination—play an essential role in improving ventilation in hypercapnic patients. In the simple linear regression at 1 hour, the independent variables positively associated with improved pCO_2 clearance were initial EPAP and Vt adjusted to ideal body weight. In contrast, initial FiO_2 showed a negative correlation, underscoring the importance of appropriately setting FiO_2 at the beginning of NIV.

A particularly important indicator was Vt adjusted to ideal body weight. Our study evaluated the contribution of global Vt, Vt per ideal body weight, and Vt per actual body weight. In the univariate analysis, all three showed similar behavior regarding pCO_2 clearance. However, after logistic regression, Vt per ideal body weight remained in the final model.

Consensus from the British Thoracic Society,[12] Masip *et al.*,[13] Artacho *et al.*,[8] Rialp Cervera *et al.*,[9] and Davidson *et al.*[14] have emphasized the importance of using ideal body weight. Our results are consistent with these recommendations, identifying Vt per ideal body weight as the optimal target parameter. In patients with hypercapnic ARF, the target oxygen saturation is 88%–92%; therefore, FiO_2 must be titrated early—within the first hour—to achieve this goal.[20]

Of note, pCO_2 clearance showed a progressive decline over time, with a greater reduction within the first hour and a lower rate between hours 1 and 3. This does not necessarily indicate reduced ventilation or the need for increased PS. Rather, as respiratory acidosis improves, the need for pCO_2 clearance diminishes until minimal—especially in patients with gasometric and ventilatory stabilization. In parallel, Vt increased from the first to the third hour. In this context, authors such as Confalonieri *et al.*,[21] Duan *et al.*,[22] and Nava[23] emphasize the importance of close monitoring during the first hour of NIV initiation, which is consistent with our results.

This study has several limitations. First, various etiologies of hypercapnic ARF were grouped together rather than analyzed individually. Second, leak was not recorded, so Vt measurements may be biased; however, no patient was excluded due to uncontrolled leak, so the values are likely reliable. Third, the institutional protocol includes increasing PS until achieving a minimum Vt of 5 mL/kg. Finally, the convenience sampling method introduces inherent bias. Nonetheless, this study represents an initial step and a basis for future research into the relationship between Vt and improved ventilation measured through pCO_2 clearance.

In conclusion, an increase in Vt adjusted to ideal body weight in patients with hypercapnic ARF undergoing NIV shows a weak-to-moderate positive correlation with alveolar ventilation, measured through pCO_2 clearance.

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