Average volume-assured pressure support ventilation mode in the management of acute hypercapnic respiratory failure

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Background Although average volume-assured pressure support (AVAPS) mode has been studied in chronic respiratory failure, studies evaluating its efficacy in acute hypercapnic respiratory failure (AHRF) are limited.

Objective The aim of this study was to investigate the benefits of spontaneous timed AVAPS (ST/AVAPS) mode in delivering noninvasive ventilation (NIV) for patients with AHRF compared with the conventional ST/BiPAP (ResMED, San Diego, California, USA) mode.

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Introduction

Mechanical ventilation is considered an effective management for patients with acute respiratory failure (RF). However, it is associated with many hazards to the patient when the tube is in place or after its removal. In recent years, noninvasive ventilation (NIV) has been developed to improve ventilation and oxygenation without the need for an endotracheal intubation and has proved its effectiveness in the management of RF [1–3].

BiPAP (ResMED, San Diego, California, USA) therapy as a type of pressure-limited noninvasive positive pressure ventilation (NPPV) mode has gained interest for its variability of administered pressure between inspiration and expiration. That variable pressure can decrease the amount of pressure against which the patient exhales and thus decreases excessive respiratory effort during the expiratory cycle. Moreover, it provides less peak inspiratory pressure [4]. On the other hand, volume-limited NPPV prevents fluctuation of tidal volume (VT) in the presence of changes in patient effort, chest wall compliance, or airway resistance [5]. Recently, hybrid modes that combine the benefit of pressure-targeted and volume-targeted ventilation have been developed in the treatment of acute hypercapnic respiratory failure (AHRF). Average volume-assured pressure support (AVAPS) is one of those newly developed modes [6]. AVAPS is a mode of NIV that estimates the patient's VT over several breathes and calculates the variations in inspiratory positive airway pressure (IPAP) needed to achieve the patient's target VT aiming for patient safety and comfort [7]. Although this mode has been studied in chronic RF, studies evaluating its efficacy in AHRF are limited.

Objective

The aim of this study was to investigate the benefits of the new ST/AVAPS mode in delivering NIV for patients with AHRF compared with the conventional ST/BiPAP mode.

Patients and methods

This randomized parallel study was conducted over one year from November 2014 to November 2015 at respiratory ICU (RICU) and wards of Chest Department, Assiut University Hospital. We programmed to include 30 patients in each of the study group, either the conventional ST mode (group I) or the new ST/AVAPS mode (group II). Patients eligible for study were randomized using the random assignment technique formally prepared by a computer generator program. The computer randomizing program assigns the case numbers in each group. Informed consent was obtained from the patients or their relatives. The study was approved by the Faculty of Medicine Ethics Committee, Assiut University.

Inclusion and exclusion criteria

This study included adult patients with AHRF with pH less than 7.35 and PCO_2 more than 6.5 kPa (i.e. >48.8 mmHg) when persisting after the initial standard medical therapy (bronchodilators and oxygen therapy) [8]. Diagnosis of the diseases causing hypercapnic RF, such as chronic obstructive pulmonary disease (COPD), obesity hypoventilation syndrome, and overlap syndrome (COPD

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and obstructive sleep apnea), was made clinically, radiologically, and based on the criteria of diagnosis of these disorders [9–11]. The reasons for ARF were evaluated and recorded as acute exacerbation of COPD [9] or obesity hypoventilation syndrome [10], pneumonia [12], acute heart failure [13], or pulmonary embolism [14]. Patients with pH more than 7.15, disturbed conscious level attributed to causes other than RF (hepatic or ureamic encephalopathy and neurological diseases), and Glasgow Coma Scale (GCS) less than 8 or those who had absolute or relative contraindications to NIV were excluded from the study [8].

Noninvasive device

A NIV, ResMED (S9 VPAP ST; ResMED, San Diego, California, USA), was used in this study. The device consists of two major parts, the S9 device unit and the H5i (ResMED, San Diego, California, USA) humidifier unit. The S9 device unit comprises the compressor that compresses room air into pressurized air to deliver positive airway pressure. The H5i humidifier unit comprises the humidifier into which distilled sterile water is poured and filling is guided by a scale of three grades minimum, medium, and maximum. On the inner side of S9 device unit, an air outlet is present, thus delivering humidified air to the patient through the air tube connected to the mask.

Initiation of noninvasive ventilation

The patients were gently placed in the sitting position and simply were explained the technique of the device. Patients were then fitted with oronasal mask ultamirage II mask (ResMed) connected to the device, which was chosen on individual basis. Patients were randomized to receive NIV using either BiPAP-ST mode (group I) or BiPAP-ST/AVAPS (group II).

Ventilatory settings

Expiratory positive airway pressure (EPAP) was adjusted at 3 cm H_2O. In patients with known obstructive sleep apnea, EPAP was initially adjusted at 4–5 cm H_2O. IPAP was adjusted at 15 cm H_2O (20 cm H_2O if pH from 7.15–7.25) and then up titrate IPAP to 20–30 to achieve adequate thoracic and abdominal effort and slow RR. Backup rate was set at 16–20 rate/min and inspiratory–expiratory (I : E) ratio at 1 : 2 or 1 : 3 in COPD patients, whereas in other disorders it was adjusted at 1 : 1. Inspiratory time was set at 0.8–1.2 s in COPD patients, whereas in other disorders it was adjusted at 1.2–1.5 s [8].

Parameters for BiPAP-ST/AVAPS mode were adjusted similar to the conventional mode besides including a setting of patient's height, target VT (6–8 ml/kg of ideal body weight/min in COPD patients and 6 ml/kg ideal body weight/min in other disorders), and minimum and maximum pressure support to provide the required IPAP and EPAP ranges.

Severity of illness assessment

Severity of illness on admission was assessed using the Acute Physiology And Chronic Health Evaluation II [15], the Modified Sequential Organ Failure Assessment Score [16], and the [17]. GCS was applied for evaluating the patient's conscious level [18]. Oxygen was administered through the mask to maintain oxygen saturation from 88 to 92%. Standard medical treatments including inhalational bronchodilators, intravenous corticosteroids, xanthenes, antibiotics, diuretics, or vasopressors were given in addition to NIPPV. Exhaled VT, respiratory rate (RR), heart rate, arterial blood pressure, and arterial blood gas (ABG) were recorded before initiation of NIV and at 1, 12, 48, and 72 h following therapy. Moreover, length of hospital stay (started from the first day of use of NIV until discharge from hospital) and duration of NIV use until obtaining the recommended outcome were recorded.

Defining outcome

Successful therapy was considered when the objective criteria showed a decrease of at least 20% in RR compared with spontaneous breathing, an improvement in ABGs with pH more than 7.35, a decrease in $PaCO_2$ of at least 15% compared with spontaneous breathing while maintaining a SaO_2 (with or without oxygen) 88–92% or when the subjective criteria showed improvement in the patient as regards both dyspnea and comfort despite persistent respiratory acidosis [i.e. the inability to obtain a clinically significant decrease in $PaCO_2$ of $\geq$15% (compared with the initial $PaCO_2$ value under spontaneous breathing) or increase in pH>7.30 after 2 h of therapy] [19].

NIV failure was considered when one major criterion was present at any time, or when two minor criteria persisted after 6 h of NIV. The major criteria included respiratory arrest, respiratory pauses or bradycardia (<50 breath/min) with loss of consciousness, hypotension with systolic arterial blood pressure below 70 mmHg, and refractory hypoxemia with inability to maintain a SaO_2 more than 90% despite high FiO_2 more than 60%. The minor criteria included tachypnea over 35 breath/min or an increase in the RR compared with its value at admission, pH less than 7.30 and decreased compared with its initial value or a decrease in conscious level compared with its initial value [20].

Statistical analysis

Statistical package for the social sciences (SPSS), version 20 (produced by IBM SPSS statistics for Windows, version 20; IBM Corp., Armonk, New York, USA) software was used for analysis of results. Using tests of normality (the Shapiro–Wilk and Kolmogorov–Smirnov tests), data of duration of hospitalization and duration of NIV were detected to be nonparametric. They were presented in median and interquartile range and analyzed using the Mann–Whitney *U*-test for comparison between the two study groups. Other results in this study were presented as mean±SD or number and percentage. The qualitative data were compared between the two groups using the χ^2-test and the quantitative data were compared using Student's *t*-test. Changes in clinical and gasometrical parameters over time among the two groups were analyzed using the one-way analysis of variance test. *P*-value less than 0.05 was considered significant.

Results

A total of 60 patients were included in this study. After randomization, 30 patients were enrolled in group I (ST mode) and 30 patients in group II (ST/AVAPS mode). The two groups were comparable as regards the age, sex, and BMI ($P>0.05$). There were also no significant differences in the underlying cause of RF and cause of admission between the two groups ($P>0.05$) (Table 1). The baseline clinical and gasometrical data as well as disease severity assessment scores (Acute Physiology And Chronic Health Evaluation II, Simplified Acute Physiologic Score II, Modified Sequential Organ Failure Assessment Score) were comparable in both groups (Table 2).

Table 1 Demographic data of the study group (*n*=60)

Variables	Group I (*n*=30)	Group II (*n*=30)	*P*-value
Age	56.7±9.8	56.1±10.7	0.812
Sex			
Male	21 (70)	20 (66.7)	0.876
Female	9 (30)	10 (33.3)	0.817
BMI (kg/m^2)	31.3±7	29.4±9.2	0.366
Underlying cause of RF			
COPD	15 (50)	16 (53.3)	0.857
Overlap syndrome	10 (33.3)	10 (33.3)	1.000
OHS	5 (16.7)	4 (13.3)	0.848
Cause of admission			
Infection exaccerbation	26 (86.7)	28 (93.3)	0.785
Heart failure	3 (10)	2 (6.7)	0.655
Pulmonary embolism	1 (3.3)	0 (0)	0.675
Disease severity scores			
APACHE II Score	11±3.3	10.4±3.6	0.503
SAPS II	28.6±9.2	25.7±7.6	0.194
M SOFA Score	3.3±1.6	3.4±1.4	0.932

Data are presented as frequency [*n*(%)] or mean±SD. APACHE II, Acute Physiology And Chronic Health Evaluation II; COPD: chronic obstructive pulmonary disease, overlap; COPD and obstructive sleep apnea; M SOFA, Modified Sequential Organ Failure Assessment; OHS, obesity hypoventilation syndrome; RF, respiratory failure; SAPS II, Simplified Acute Physiologic Score II.

Table 2 Baseline clinical and gasometric parameters of the study groups (*n*=60)

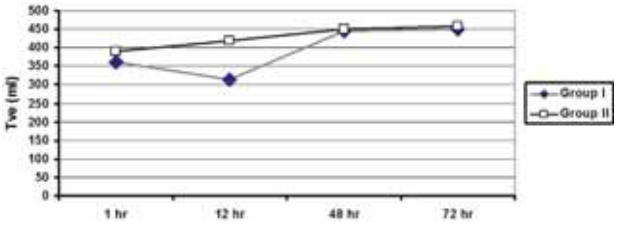
Variables	Group I (*n*=30) (mean±SD)	Group II (*n*=30) (mean±SD)	*P*-value
Clinical			
GCS	14.47±0.68	14.67±0.66	0.253
RR (breath/min)	28.23±7.06	26.03±7.22	0.238
HR (rate/min)	127.3±14.1	122±15.6	0.170
SBP (mmHg)	80.7±8.3	77±8.8	0.100
DBP (mmHg)	77.6±6.9	76.7±8.9	0.132
ABG			
pH	7.32±0.08	7.31±0.08	0.606
$PaCO_2$	75.6±17.46	74.13±16.8	0.741
PaO_2	65.53±14.77	64.1±15.79	0.718
SaO_2	88.9±7.3	84.23±17.51	0.183

ABG, arterial blood gas; DBP, diastolic blood pressure; GCS, Glasgow Coma Scale; HR, heart rate; $PaCO_2$, partial arterial carbon dioxide tension; PaO_2, partial arterial oxygen tension; RR, respiratory rate; SaO_2, arterial oxygen saturation; SBP, systolic blood pressure.

We tackled the progress of the patients clinically and using repeated ABG analysis at 1, 12, 48, and 72 h. At 1 h, there was no significant change in GCS, RR, PH, or $PaCO_2$ in either groups; however, a significant improvement was observed in PaO_2 in both groups I and II and in oxygen saturation in group II (P=0.007) (Table 3). At 12 h, a significant improvement was observed in GCS (15±0 vs. 14.67±0.66, P=0.010), RR (22.7±4.21 vs. 26.03±7.22, P=0.033), and pH (7.37+0.06 vs. 7.31+0.08, P=0.001) with a sustained improvement in PaO_2 and SaO_2 in group II. Meanwhile, in group I, only RR showed a significant improvement at that time (24.17±3.34 vs. 28.23±7.06 P=0.006). Moreover, group I could not maintain the previously detected improvement in oxygen tension with further decrease in oxygen tension at 12 h (Table 4). At 48 h, (Table 5) there was a significant improvement in GCS, RR, pH, $PaCO_2$, and SaO_2 in group I. In group II, a significant improvement in $PaCO_2$ started to appear, along with maintenance of the previously acquired improvement in other parameters. At 72 h of follow-up, maintenance of improvement in the monitored parameters continued in both groups (Table 6).

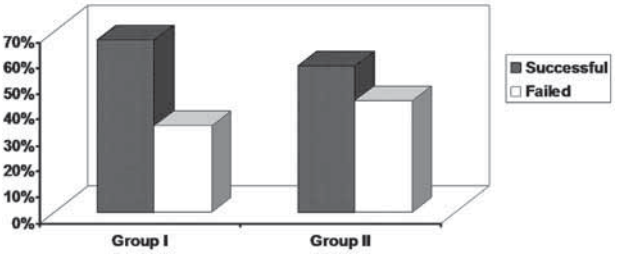
Figure 1 demonstrates exhaled VT over 72 h in both study groups in which we observed fluctuation in VT in group I. Figure 2 shows the estimated outcome for both groups. In group I, the number of successful cases was

Figure 1



Exhaled tidal volume monitoring over 72 h in both study groups. TVe, exhaled tidal volume.

Figure 2



Estimated outcomes of BiPAP therapy in both study groups. The number of successful cases was 20 (66.7%) in group I and 17 (56.7%) in group II (P=0.426).

Table 3 Changes in Glasgow Coma Scale, respiratory rate, and arterial blood gases at 1 h

	Group I (n=30) (mean±SD)			Group II (n=30) (mean±SD)		
	At admission	At 1 h	P-value	At admission	At 1 h	P-value
Clinical						
GCS	14.47±0.68	14.63±0.67	0.343	14.67±0.66	14.87±0.35	0.149
RR (breath/min)	28.23±7.06	26.8±6.04	0.402	26.03±7.22	24.43±4.14	0.297
ABG						
pH	7.32±0.08	7.34±0.08	0.319	7.31±0.08	7.35±0.09	0.709
$PaCO_2$	75.6±17.46	74.3±15.14	0.600	74.13±16.8	69.87±16.24	0.321
PaO_2	65.53±14.77	75.9±17.94	0.018*	64.1±15.79	75.87±21.93	0.020*
SaO_2	88.9±7.3	91.87±8.53	0.153	84.23±17.51	93.63±6.22	0.007*

ABG, arterial blood gas; GCS, Glasgow Coma Scale; $PaCO_2$, partial arterial carbon dioxide tension; PaO_2, partial arterial oxygen tension; RR, respiratory rate; SaO_2, arterial oxygen saturation. *Significant.

Table 4 Changes in Glasgow Coma Scale, respiratory rate, and arterial blood gases at 12 h

	Group I (n=30) (mean±SD)			Group II (n=30) (mean±SD)		
	At admission	At 12 h	P-value	At admission	At 12 h	P-value
Clinical						
GCS	14.47±0.68	14.63±0.81	0.392	14.67±0.66	15±0	0.010*
RR (breath/min)	28.23±7.06	24.17±3.34	0.006*	26.03±7.22	22.7±4.21	0.033*
ABG						
pH	7.32±0.08	7.35±0.07	0.148	7.31±0.08	7.37±0.06	0.001*
$PaCO_2$	75.6±17.46	74.2±17.64	0.757	74.13±16.8	67.07±12.3	0.071
PaO_2	65.53±14.77	70.5±16.3	0.219	64.1±15.79	75.87±14.5	0.004*
SaO_2	88.9±7.3	91.9±5.9	0.086	84.23±17.51	92.7±6.1	0.017*

ABG, arterial blood gas; GCS, Glasgow Coma Scale; $PaCO_2$, partial arterial carbon dioxide tension; PaO_2, partial arterial oxygen tension; RR, respiratory rate; SaO_2, arterial oxygen saturation. *Significant.

20 (66.7%) and the number of failed cases was 10 (33.3%). Failure rate was reported in patients with infection exacerbation in 70% of cases and heart failure in 30% of cases. In group II, the number of successful cases was 17 (56.7%) and that of failed cases was 13 (43.3%), in which the main cause of failure was reported in patients with infection exacerbation; however, the difference between the two groups was not significant (P=0.426). Although, no significant difference was found between the two groups as regards length of hospital stay (P=0.960), the duration spent on NIV was significantly shorter in group II than in group I [1 (1–1.25) vs. 2 (1–3), P=0.049] (Table 7).

Discussion

When $PaCO_2$ is increased as in patients with acute hypercapnic RF, the patient has to increase minute ventilation to reduce hypercapnia. In acute hypercapnic RF, the respiratory muscles are failing to generate sufficient alveolar ventilation leading to hypoventilation and progressive hypercapnia. Thus, the means to improve this patient is to increase alveolar ventilation and reduce work of breathing [21]. Pressure-limited modes of NIV could decrease the amount of pressure against which the patient exhales, thus decreasing work of breathing without increasing peak inspiratory pressure [4]. On the other hand, volume-limited NPPV has the advantage to maintain adequate VT in the presence of changes in patient effort, chest wall compliance, or airway resistance [5]. Recently, hybrid modes that combine the benefit of both pressure-targeted and volume-targeted ventilation could be of great benefit in patients with hypercapnic RF. This study aimed to investigate the benefits the new ST/AVAPS mode in delivering NIV for patients with AHRF.

When monitoring patients with RF over 72 h, this study showed that, at 12 h, a significant improvement was observed in GCS, RR, and PH with a sustained improvement in PaO_2 and SaO_2 in group II; however, these parameters significantly improved at 48 h in group

Table 5 Changes in Glasgow Coma Scale, respiratory rate, and arterial blood gases at 48 h

	Group I (n=30) (mean±SD)			Group II (n=30) (mean±SD)		
	At admission	At 48 h	P-value	At admission	At 48 h	P-value
Clinical						
GCS	14.47±0.68	14.97±0.18	<0.001*	14.67±0.66	15±0	0.010*
RR (breath/min)	28.23±7.06	24.37±3.44	0.010*	26.03±7.22	23.03±1.69	0.034*
ABG						
pH	7.32±0.08	7.4±0.06	<0.001*	7.31±0.08	7.4±0.04	<0.001*
$PaCO_2$	75.6±17.46	66.47±13.29	0.026*	74.13±16.8	64.37±13.25	<0.001*
PaO_2	65.53±14.77	72.2±13.15	0.070	64.1±15.79	75.97±12.09	0.015*
SaO_2	88.9±7.3	93.07±4.35	0.010*	84.23±17.51	93.67± 3.14	0.007*

ABG, arterial blood gas; GCS, Glasgow Coma Scale; $PaCO_2$, partial arterial carbon dioxide tension; PaO_2, partial arterial oxygen tension; RR, respiratory rate; SaO_2, arterial oxygen saturation. *Significant.

Table 6 Changes in Glasgow Coma Scale, respiratory rate, and arterial blood gases at 72 h

	Group I (n=30) (mean±SD)			Group II (n=30) (mean±SD)		
	At admission	At 48 h	P-value	At admission	At 48 h	P-value
Clinical						
GCS	14.47±0.68	14.93±0.37	0.002*	14.67±0.66	15±0	0.010*
RR (breath/min)	28.23±7.06	23.87±3.49	0.004*	26.03±7.22	21.47±1.66	0.002*
ABG						
pH	7.32±0.08	7.4±0.06	0.000*	7.31±0.08	7.41±0.03	<0.001*
$PaCO_2$	75.6±17.46	66±14.35	0.023	74.13±16.8	66.8±0.13.09	0.020*
PaO_2	65.53±14.77	73.5±14.46	0.039	64.1±15.79	76.17±9.13	0.001*
SaO_2	88.9±7.3	93.7±7.2	0.002*	84.23±17.51	94.17±2.2	0.004*

ABG, arterial blood gas; GCS, Glasgow Coma Scale; $PaCO_2$, partial arterial carbon dioxide tension; PaO_2, partial arterial oxygen tension; RR, respiratory rate; SaO_2, arterial oxygen saturation. *Significant.

Table 7 Effect of BiPAP therapy on length of hospital stay and duration of noninvasive ventilation on both groups (n=60)

Variables	Group I (n=30)	Group II (n=30)	P-value
LoH stay (days)	13.5 (9–19)	13 (8–21)	0.960
Duration of NIV (days)	2 (1–3)	1 (1–1.25)	0.049*

Data are presented as median (interquartile range). LoH, length of hospital; NIV, noninvasive ventilation. *Significant.

I even with fluctuation in oxygen tension and saturation. As in our study, Claudett *et al.* [22] and Hussein and colleagues [23] observed a rapid and significant improvement in ABGs and consciousness (GCS) in both groups; however, patients treated with BiPAP S/T+AVAPS improved much faster compared with patients treated with the conventional strategy. Moreover, we showed improvement in $PaCO_2$ at 48 h in group II, whereas it appeared at 72 h in group I. Battisti *et al.* [24] compared manually adjusted pressures with self-adjusting pressure support in patients with acute RF, which produced a decrease in $PaCO_2$ levels in the latter group. In chronic patients with obstructive sleep apnea and alveolar hypoventilation syndrome, some authors reported a rapid improvement in $PaCO_2$ and sleep quality using VAPS [7,25,26], whereas others reported no difference between AVAPS and the conventional ST mode [7]. VAPS was studied for stable hypercapnic COPD patients in a limited number of previous recent clinical trials. Ekkernkamp *et al.* [27] compared noninvasive VAPS mode and high-intensity pressure support in 40 patients and revealed that there was a greater decrease in transcutaneous partial pressure of CO_2 during VAPS. However, other studies demonstrated no advantage of AVAPS versus pressure support in chronic stable COPD patients [28,29]. The ability of AVAPS mode to maintain the exhaled VT compared with BiPAP S/T mode alone as observed in our study could explain the faster improvement in oxygenation and hypercapnia consequently with improvement in alveolar ventilation.Overall outcome in our study showed that, in group I, the number of successful cases was 20 (66.7%) and the number of failed cases was 10 (33.3%). In group II, the number of successful cases was 17 (56.7%) and the number of failed cases was 13 (43.3%). Outcome results were variable in previous studies. Intubation rate was reported to exceed 20% in a group of hypercapnic patients [30]. Plant *et al.* [31] reported an overall intubation rate of only 15% in patients receiving NIV in respiratory wards, but this rate reached 36% in patients with a pH less than 7.30. A recent study reported a rate of NIV failure of only 11% in severe COPD patients admitted in a specialized RICU [32]. Our results could not be compared with the previous studies due to variation in patients' population, patients' age, and the setting of the study (RICU, respiratory monitoring unit in a respiratory ward, general ICU, and both hospital ward and ICU). The relatively higher rate of failure in group II than in group I could be attributed to the fact that AVAPS mode was applied in more patients with infection exacerbation compared with BiPAP/ST mode. However, we reported a significantly shorter duration on NIV in group II compared with group I. Thus, AVAPS mode could be cost-effective on patients with AHRF.

Conclusion

Both ST/BiPAP and AVAPS modes are effective in the management of patients with AHRF. However, AVAPS modes showed more rapid and steady improvement in clinical parameters and shorter duration on NIV.

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The manuscript has been read and approved by all authors, that the requirements for authorship as stated earlier in this document have been met, and that each author believes that the manuscript represents honest work, if that information is not provided in another form.

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Conflicts of interest

There are no conflicts of interest.

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