Role of Non- Invasive Continuous Positive Airway Pressure in Management of Acute Cardiogenic Pulmonary Edema

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ABSTRACT

Purpose of the sudy; this work was conducted to invesitgate the effect of non- invasive continuous positive airway pressure (CPAP); as adjunct tool, in management of acute cardiogenic pulmonary edema(ACPE). Methods and Results:Forty patients with ACPE were randomly assigned into two groups : group I who received conventional therapy and group II who received conventional therapy in addition to mask face CPAP for an hour. There was a sginifcant differences in blood gases values (PaO_2, $PaCO_2$ HCO_3 and pH), in favour to CPAPgroup. No patient needed invasive mechanical ventilation in group II, while 30% of patients in group I needed intubation. Conculsion: CPAP is a usful tool in mamgement of ACPE. Its administeration by face mask improve blood gases, and reduce the need for mechanical intubated ventilation.

INTRODUCTION

Acute cardiogenic pulmonary edema (ACPE) is a common cause of acute respiratory failure presenting at emergency department. Conventional treatment of such conditions includes the use of oxygen therapy, opiates, diuretic and vasodilators[7]. Failure of these treatment leads to intubation and mechanical ventilation that improve oxygenation and reduce respiratory muscle fatigue. However these was associated with high risk of morbidity (e.g. airway trauma, nosocomial pneumonia and sinusitis), in addition endotracheal intubation may prolong intensive care unit and hospital stay as addition time may be necessary for weaning from ventilation and treatment complication[3,18].

The use of continuous positive airway pressure (CPAP) delivered through a tight fitting face mask in patients with ACPE significantly reduces the needs for endotracheal intubation however; no survival benefit was demonstrated by the individual trial[13,16].

The possible benefits of CPAP includes improve oxygenation, increase in functional residual capacity , a decrease in right to left intrapulmonary shunt, reduced atelectasis, and an increase in cardiac output via decreases in left ventricular after load. Furthermore CPAP may reduce the work of breathing by improving pulmonary compliance. Also few authors stated that CPAP might be improved arterial oxygen tension PaO_2 and decrease carbon dioxide arterial tension $PaCO_2$ [2].

Despite this finding the utilization of CPAP requires a specialized gas-delivery circuit and more supervision that traditional oxygen mask, it use remains sporadic. Moreover, CPAP has not been shown to affect the need for mechanical ventilation or mortality from cardiogenic pulmonary edema[14].

Therefore the aims of this study was (1) was to assess the effect of the CPAP on blood gas, and (2) to determines whether the use of

CPAP would reduce the need for intubation and mechanical ventilation in patients with ACPE.

SUBJECTS AND METHODS

Subjects

Forty patients with ACPE had been recruited from emergency department at national Heart Institute and were randomly assigned into two equal groups; Group I who received conventional oxygen and Group II who relieved conventional oxygen plus CPAP therapy. The ACPE diagnosed according to criteria of Remme and Swedberg[12] when the patients complained from dyspnea, orthopnea, forthy expectoration, basal crepitation bilateral diffuse interstitial or alveolar edema as confirmed by portable X-ray. All patients were in class IV New York Heart Association (NYHA). The patients were excluded if they had a diagnosis of myocardial infarction, systolic blood pressure below 90mmHg; sever stenotic valvular disease, chronic obstructive pulmonary disease and abundant secretion. Patients were also excluded from the study if they were unresponsive agitated, and unable to cooperate, or if had any conditions that precluded application of the face mask (e.g. recent facial trauma).

Procedure

The standard medical treatment (oxygen therapy, nitroglycerin, frusemide and morphine) was administrated for all patients. These patients complained from dyspnea, with use of accessory respiratory muscles or paradoxical abdominal motion, orthopnea, forthy expectoration, and basal crepitation by auscultation at the time of admission.

Arterial cannula was inserted into radial or pedal artery to draw blood sample for blood gases analysis. The blood was drawn in heparin rinsed plastic syringe and was analyzed immediately. The acid –base analyzer (Model ABL 3075R, radiometer A/S Copenhagen) was used to detect the level of (PaO_2, $PaCO_2$ pH, HCo_3 and SaO_2) at the blood sample. It was performed on the entry of the study (pre) and after 30 minutes (post).The ECG (HP; Italy) allows continuous observation and mentoring of the patients through a modified chest lead, to provide optimal information regarding changes in rhythm and heart rate (HR). The blood pressure can measure directly from an automatic syphagmoamnometer that strapped around the brachial region and the monitor display systolic and diastolic blood pressure, and thereby ensure close patients monitoring. All patients or their relative gave written consent. All patients were placed in semi setting position with back support. The patients were administrated to non invasive CPAP (Respi, Care, Drager; London) through tight fitting face mask with pressure of (5, 7,9,11 cm H_2O), that was increased in incremental steps for 15 minutes at each level with total duration of one hours. Care was taken to ensure that patient could tolerate applied pressure and could breathe consistently with a closed mouth. Electrocardiogram (ECG) were monitored continuously through out CPAP and oxygen therapy. During treatment the resolution of ACPE was defined as evident of clinical improvement with a respiratory rate of less than 30 breathe per minutes, and oxygen saturation of more than 96%. While the predetermined criteria for intubation and mechanical ventilation were clinical deterioration (loss of consciousness, airway obstruction) and either a fall in arterial oxygen tension to less than 70 mmHg, with patients breathing 100% oxygen, by mask, or arise in arterial carbon dioxide to more than 55mmHg.

Data Analysis

Base line demographic data and physiological measurements of both groups were expressed as (mean ± SD), for parametric data and as number and percentage for nonparametric data. The student t-test for paired measurements was used to detect significant differences within groups, while unpaired t-test was use to detect significant difference between both groups. The rate of success expressed as percentage. The level of significant was assumed at (P<0.0%) at two tiled test.

RESULTS

At the time of the study entry, age, sex ,causes of disease and NYHA classification were similar between two groups of the study and revealed no significant differences (P>0.05) table (1).

At study entry; the patient in both groups had sever respiratory failure, the most common feature of which were tachypnea as the mean value of respiratory rate were (35±5versus 39±8 br/m), for control versus CPAP groups. The mean value of heart rate was (113 ± 21 versus 107 ± 23 b/min) for control versus CPAP. There was a mixed respiratory and metabolic acidosis as the mean value of pH was (7.29±0.9 versus 7.25±1) for control and CPAP groups while the mean value of HCO3 was 24±3 versus 23 ±1.8 mmol/L).the mean value of SBP was (124± 19.5 versus 128.5±23mmHg) while for DBP it was (85±8.6 versus 87±11mmHg) for control versus CPAP groups respectively.

The mean value of PaO_2 for control group was 79±13.8mmHg, while it was 76±8mmHg for CPAP and this value showed no significant different between both groups at entry of the study and demonstrated that all patient in both group suffering from hypoxemia. The mean value of SaO2% was (89 ± 3.8 versus 90 ± 2.6) for control and CPAP groups. The mean value of $PaCO_2$ for control group was 48±5mmHg and 46±8 mmHg for CPAP group and reflect that all patient were hypercapnea, with no significant differences between them (P>0.05), table (2).

After 30 minutes; the patient receiving oxygen therapy plus CPAP had significant increase of PaO_2 from pre treatment value of (Pre) 76±8mmHg to 97.6±10mmHgfor post (after 30 minutes) this associated with simaler increase of SaO_2 form 90± to 98.2± and this showed significant increase of both Pao2and SaO_2 (P<0.05), while the mean value $PaCO_2$ was 46±8mmHg form pretreatment (Pre) to 32.8±4.5 mmHg with stastical significant difference (P<0.05) table (3). For control group there were significant increase in PaO_2 and SaO_2, while for $PaCO_2$, there significant increase from pretreatment observation (P<0.05), table (4).

The percentage of improvement (increased) for PaO_2 was (14.6 versus 28.4) for control versus CPAP. While for $PaCo_2$ the mean percentage values was (18.7 versus 28.7), while SaO_2 had percentage of increase of 4.5 for control group and 9.2% for CPAP, table (3, 4) fig. (1,2). Comparing the mean values of PaO_2 & SaO_2 after 30 minutes between both control and CPAP groups showed significant increase in favoring CPAP therapy (P<0.05), while for $PaCo_2$, there was significant reduction for both group and favoring CPAP therapy, table (5), fig. (3).

Table (1): Patients' demographic characteristic at the entry of the study.

Variables	Group I Oxygen therapy N = 20 patients	Group II Oxygen therapy+ CPAP N = 20 patients	P-Value
Age (years)X±SD	56±4	55±6	0.6
Sex (M/F)	8/12	7/13	0.8
Diagnosis IHD ICM	 8 12	 9 11	0.8
NYHA classification	IV	IV	1

Table (2): The physiological measurements at the entry of the study for both groups.

Variables	Group I Oxygen therapy N = 20 patients	Group II Oxygen therapy + CPAP N = 20 patients	P-Value
Respiratory rate(br/m)	35±5	39±8	0.9*
Heart rate (b/m)	113±21	107±23	0.9*
Systolic blood pressure (mmHg)	124±19.5	128.5±23	0.2*
diastolic blood pressure (mmHg)	85±8.6	87±11	0.5*
PaO_2 (mmHg)	79±13.8	76. ±8	0.8*
$PaCO_2$ (mmHg)	48±9	46±8	0.4*
SaO_2 %	89±3.8	90±2.6	0.2*
Arterial HCO3(mmol/l)	24.3±3	23.2±1.8	0.3*
Arterial pH	7.29±0.9	7.25±1.2	0.5*

Table (3): Statistical analysis of the mean values of blood gases (PaO_2, $PaCO_2$, and SaO_2) at the entry of study (Pre), and after30 minutes of treatment (Post) for group II (oxygen therapy and CPAP).

Variables	PaO_2		$PaCO_2$		SaO_2	
Statistics	Pre	Post	Pre	Post	Pre	Post
Mean	76	97.6	46	32.8	90	98.2
±SD	8	10	8	4.5	2	1.8
MD	-21.6		13.2		-8.2	
P-value	0.03		0.04		0.05	
%of improvement	28.4		28.7		9.2	

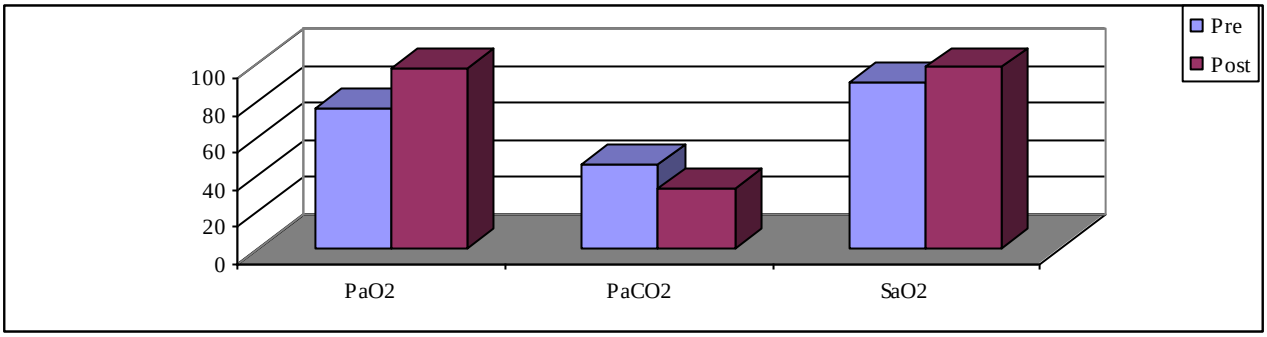

Fig. (1): The mean value of blood gases ((PaO_2, $PaCO_2$, and SaO_2) at entry of the study (Pre) and after 3o minutes (Post) of CPAP therapy.

Table (4): Statistical analysis of the mean values of blood gases analysis (PaO_2, $PaCO_2$, and SaO_2) at the entry of study (Pre), and after 30 minutes of treatment (Post) for group I (oxygen therapy).

Variables / Statistics	PaO2		$PaCO_2$		SaO_2	
	Pre	Post	Pre	Post	Pre	Post
Mean	75	86	48	39	89	93
±SD	13.8	16.1	9	7.6	2.8	2.1
MD	-11		9		-4	
P-value	0.05		0.04		0.05	
%of improvement	14.6		18.7		4.5	

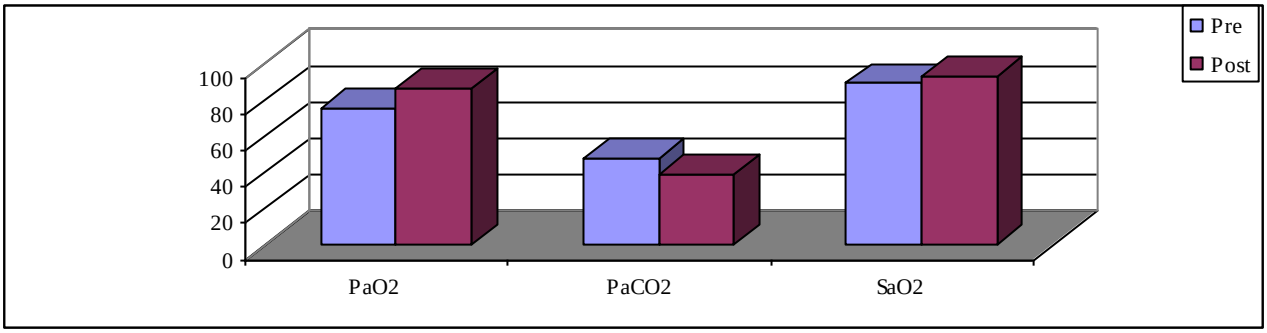

Fig. (2): The mean value of blood gases ((PaO_2, $PaCO_2$, and SaO_2) at entry of the study (Pre) and after 3o minutes (Post) of control group.

Table (5): Comparative analysis of the mean values of blood gases (PaO_2, $PaCO_2$, and SaO_2) after 30 minutes of treatment (Post) for both groups (I& II).

Variables / Statistics	PaO_2,		$PaCO_2$		SaO_2	
	Group I	Group II	Group I	Group II	Group I	Group II
Mean	86	97.6	39	32.8	93	98.2
±SD	16.1	10	7.6	4.5	2.1	1.8
MD	-11.6		6.2		-5.2	
P-value	0.03		0.04		0.04	

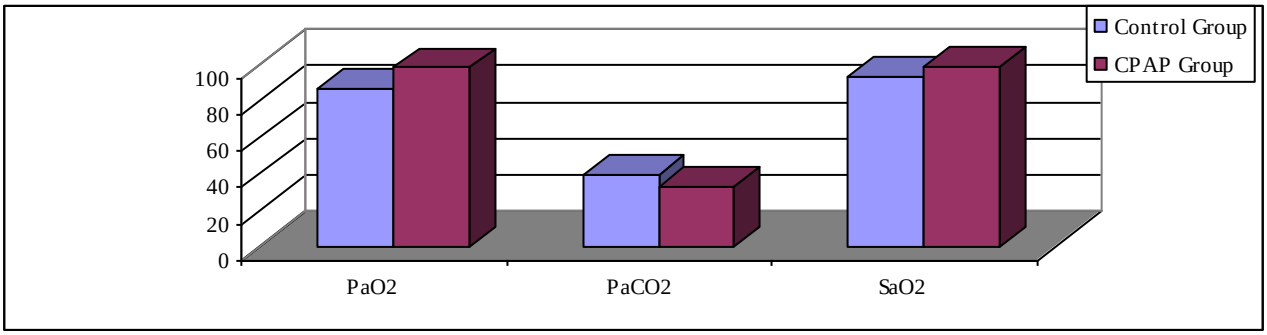

Fig. (3): The mean value of blood gases ((PaO_2, $PaCO_2$, and SaO_2) after 3o minutes (Post) for both CPAP therapy group and control group.

DISCUSSION

The results of present study demonstrated that the continuous positive airway pressure delivered through face mask has several advantages over supplemental oxygen alone in the management of sever cardiogenic pulmonary edema. Continuous positive airway pressure resulted in more rapid improvement in oxygenation, ventilation and fewer patients required mechanical ventilation (5%) versus (30%) in control group.

This is in agreement with Bersten et al.,[1] who investigate 39 patients with ACPE and found no needs for mechanical ventilation in CPAP group while rate of intubation (35%) that was smaller to our finding.

The effect of (CPAP), on blood gases PaO2, $PaCO_2$, SaO_2, HCO_3, PH), as well as hemodynamic variables (HR, RR, SBP, and DBP) were studied in patients with acute pulmonary edema secondary to congestive heart failure.

The results of the current study revealed that there was continuous and significant reduction of percentage of relative changes of the hemodynamic variables (HR, RR, SBP and DBP) (9.79 to 12. 09, 14.49 to 19.51, 11.9 to 6.42 and 8.25 to 12.38 %) respectively. This was reduced and maintained from immediate values to 30 minutes after discontinuation of CPAP therapy.

The relief of tachypnea, toward normal breathing rate seen in the present study may occur as a result of reduced stimulation of pulmonary irritant receptors due to decrease in the interstitial pulmonary edema[15]. A significant reduction in HR and blood pressure may reflect the evidence that the hemodynamic response to CPAP is due to decrease in circulatory stress mediated by sympathyo-adrenergic drive in ACPE. As CPAP induced lung inflation and stimulated pulmonary vagal afferent that led to increase parasympathetic tone and reflexively reduced cardiac sympathetic outflow, as well as a decrease in intrathoracic aortic transmural pressure by CPAP that tend to decrease baroreceptor discharge[11].

For blood gases the results of the present study revealed that, there were significant increased in (PaO_2, SaO_2), after the end of CPAP therapy with return to base line values after 30 minutes of discontinuation of CPAP. Also HCO_3, showed increase immediately after CPAP, but was non-significant and then returned to base line values, while the PH remained higher but non-significant. There was significant reduction in ($PaCO_2$), from immediate, to next 30 minutes after discontinuation, of CPAP therapy. Our results confirmed a liner increase in PaO_2, SaO_2, and HCO3, immediately after and after 30 minutes of the end of CPAP, at incremental pressure of (5, 7, 9, and 11 cmH_2O), with return to a base line value when CPAP was removed. For ($PaCO_2$), it remained relatively lower than their base line level and pH remained relatively higher than base line level measurements. This finding demonstrates that the changes in blood gases were due to the effect of CPAP rather than other factors such as medication.

The study results showed significant increase in PaO_2, and decrease in $PaCO_2$, which may be introduced by increasing the end- expiratory volume with recruitment of alveoli, and redistribution of fluid but not by decreasing lung water. All of these resulted in a greater surface area where gas exchanges occurs[14].

In current study; the significant increase in SaO_2. According to oxyhemoglobine dissociation curve, may be attributed to an increase in PaO_2, also the SaO_2 measurement is affected by pH and $PaCO_2$ as increase in pH

and decrease in $PaCO_2$ shift the curve to the left, resulting in increased oxygen affinity for hemoglobin at the same PaO_2 value[17].

A decrease in RR when coupled with improved oxygenation and a lower $PaCO_2$ is however consider as indirect objective evidence of decrease work of breathing and dyspnea. Several investigators mentioned reduction of respiratory work, as an advantage of CPAP therapy, although no definite proof of such effect exist[5,6,7].

The present results were in agreement with Chadda et al.,[4] who demonstrated significant improvement in PaO_2, $PaCO_2$, SaO_2, with reversal of acidosis (pH>7.35) as measured within 30 minutes and one hour of administration of 10 cmH_20 CPAP. Also hemodynamic variables showed significant fall in RR at 40 minutes of CPAP therapy. However there were no significant changes in HR, SBP, and DBP, which contradicted with the results of present study, that may be attributed to the use of different pressure value and all patients at the entry of the study suffered from acidosis (pH< 7.35).

The present study was supported by the study of Kelly et al.,[7] who showed that the CPAP is an effective treatment for acute cardigenic pulmonary edema (ACPE). As they reported more rapid improvement in RR, HR, and pH at one hour of treatment with 7.5 cm H_2O CPAP. More over there were no treatment failure in the CPAP group as well as non-of the patients reported adverse effects from CPAP other than minor discomfort from the facial mask. Also the main clinical benefits of CPAP occurred within the first hour, suggesting that treatment should be commenced as soon as possible after admission. Treatment should ideally commenced within the emergency department.

The present results coincided with results of early study by Remm et al.,[12] who reported that CPAP therapy via face mask with 10 cm H_2O in patients with acute cardiogenic pulomnary edema was useful. They found a reduction of HR, RR, SBP, and DBP, after 10 minutes and during 3 hours of investigation period of CPAP therapy. As well as the $PaCO_2$ was decreased while PaO_2, and pH was increased after 10 minutes and continued over entire of three hours period, with only exception that the $PaCO_2$ remained unchanged. They were attributed the improvement in oxygenation to decrease in intrapulomnary shunting and ventilation to perfusion mismatch.

The current study confirmed with, Lin et al.,[9] stated that CPAP delivered through a facemask at serial of (2.5, 5, 7, and 10 cmH_2O) causes significant improvement in hemodynamic variables such as (HR, RR, and SBP). Also the PaO_2 was significantly increased and this result was significant when compared with control group who received oxygen therapy alone.

It was showed significant reduction in RR, HR, SBP, DBP, at 30 minutes after CPAP therapy as well as there were an improvement in $PaCO_2$ with increased PaO_2 and pH. While HCO_3 remained unchanged and these findings are kept consistent and similar to present finding, those confirm short-term effect of CPAP therapy[1].

On the other hand the results of current study contradicted with the early study by Lenique et al.,[8] who studies the effect of CPAP at 0, 5 & 10 cmH_2O in nine patients with acute heart failure. While the pressure used in current study was 5, 7, 9, 11 cmH_2O. They found no decrease in RR, and $PaCO_2$, as well as HR, decreased slightly but not significantly, although PaO_2 was increased, these variation results may be attributed to sample selection, and different level of pressure values used.

The present study contradicted with results of Mehta et al.,[10] who failed to demonstrate significant effect of CPAP at 10cmH_2O on $PaCO_2$ PH, HR as well as dyspnea scores within 30 minutes of initiation of treatment. Although statistical significant improvement observed only in RR, when compared with patients who were receiving Bi-level positive airway pressure. This improvement continued at 60 minutes of CPAP therapy but did not reach statistical significant that due to small sample and different pressure used. Despite this finding that highlights the bi-level positive pressure over CPAP, the rate of developing myocardial infarction was twice as those with CPAP. This rapid improvement occurring with Bi-level positive pressure most likely related to the higher inspiratory than expiratory pressure enabling bi-level positive pressure to actively assist inspiration, augment tidal volume and decrease work of breathing. By this mechanism Bi-level positive pressure enhanced, improvements in gases exchange, vital singes and dyspnea[15].

More over the present results contradicted with Chadda et al.,[4] who study the cardiac and respiratory effect of CPAP and Bi-level non-invasive positive pressure ventilation (Bi-NPPV) in acute cardiogenic pulmonary edema (ACPE). They failed to demonstrate significant changes in HR, blood pressure during CPAP at 5 and 10 cmH_2O, and NPPV. However NPPV and 10 CmH_2O produce a reduction in right and left ventricular preload, which suggested an improvement in cardiac performance. This contradiction raised form small simple (six patients) and different techniques of application, as well as duration of application. As patients were monitored during five separate study periods, every period lasted 20 minutes. The first and the fifth period were period of spontaneous breathing, CPAP of 5, 10 and Bi-NPPV were administered in between.

It is seem that the CPAP has a beneficial effect in improving gas exchanges, and hemodynamic variables and may reduce the need for mechanical ventilation in CHF patients with ACPE. Because of its safety simplicity and reversibility, CPAP therapy bridge the gap between the oxygen mask and mechanical ventilation and may be used as physical therapy modality in emergency department.

Conclusion

The results of this study confirmed the following conclusion: the CPAP is safe – well tolerated adjunctive therapy in congestive heart failure patients with acute cardiogenic pulmonary edema. It produces rapid improvement in hemodynamic variables (HR, RR, SBP, and DBP) as well as blood gases (PaO_2, $PaCO_2$, SaO_2, HCO_3, and pH) in these patients within first hour of its application in emergency setting. In addition, the CPAP as physical therapy modalities reduce the need for mechanical intubated ventilation.

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الملخص العربى

دور جهاز التنفس الإيجابى المستمر الغير تداخلى فى علاج الارتشاح الرئوى الحاد

أجريت هذة الدراسة لمعرفة امكانية استخدام جهاز التنفس الإيجابى المستمر الغير تداخلى فى علاج الإرتشاح الرئوى الحاد المصاحب لفشل عظلة القلب . اشتملت الدراسة على أربعون مريض فى وحدة الطوارئ بمعهد القلب القومى بحيث خضع عشرون مريض للعلاج التقليدى العشرون الآخرين للعلاج التقليدى فضلاً عن استخدام جهاز التنفس الإيجابى المستمر الغير تداخلى لمدة ساعة وتم تقيم المريض من خلال قياس غازات الدم وديناميكية الدم. تشير النتائج إلى امكانية استخدام هذه الطريقة فى علاج هؤلاء المرضى وأن استخدامه يؤدى إلى تقليل الاحتياج إلى استخدام جهاز التنفس الاختراقى .