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NEW TECHNOLOGIES

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A pilot study of short-term hemodynamic effects of negative pressure ventilation in chronic obstructive pulmonary disease assessed using electrical cardiometry

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Abstract

Background: Pulmonary rehabilitation combined with negative pressure ventilation (NPV) demonstrated benefits in patients with chronic obstructive pulmonary disease (COPD). The effect of NPV remains unknown. This study aims to clarify the short-term response of the hemodynamic outcome of NPV in patients with COPD undergoing pulmonary rehabilitation program by electrical cardiometry.

Methods: This is an observational retrospective study of COPD patients who had been treated in a pulmonary rehabilitation unit with NPV between January 2018 and December 2019 that were enrolled to analyze the hemodynamic outcomes.

Results: Thirty patients with COPD that were undergoing a pulmonary rehabilitation program and were regularly receiving NPV were enrolled. Cardiac output ($p < .001$) and heart rate ($p < .001$) showed a significant decrease after NPV. Stroke volume did not demonstrate significant change ($p = .15$). There was a significant decrease in thoracic fluid content ($p = .016$) and a significant increase in stroke volume variation ($p = .038$) systemic vascular resistance ($p < .001$) and left ventricular ejection time ($p < .001$). Other hemodynamic parameters were all comparable before and after NPV.

Conclusions: Negative pressure ventilation demonstrated an impact on hemodynamics in patients with chronic obstructive pulmonary disease undergoing pulmonary rehabilitation. Electrical cardiometry is a feasible method of determining the hemodynamic effects of negative pressure ventilation. Thoracic fluid content significantly decreased immediately after the NPV.

KEYWORDS

cardiac output, chronic obstructive pulmonary disease, electrical cardiometry, negative pressure ventilation, pulmonary rehabilitation

1 | INTRODUCTION

Negative pressure ventilation (NPV), a type of noninvasive ventilation, was first described in 1928 by Drinker and colleagues (Drinker & Shaw, 1929). Two negative pressure ventilator types are available, the cuirass ventilator and the tank ventilator, which is also known as the iron lung (Thomson, 1997). NPV decreases pleural and alveolar pressure and contributes to lung expansion by creating a pressure gradient called transpulmonary pressure (Shneerson, 1991). Both NPV and positive pressure ventilation (PPV) have an effect on hemodynamics. NPV

with the cuirass ventilator increases venous return, leading to ventricular filling and increased cardiac output (CO) (Shekerdemian et al., ,1996, 1999; Skaburskis et al., 1987). In contrast to NPV, the effect of PPV in most situations is decreased venous return, leading to decreased CO (Cherpanath et al., 2013; Pinsky, 1990; Soni & Williams, 2008). NPV increases pulmonary vessel resistance when overdistended, which is similar to the action of PPV (Cherpanath et al., 2013). A report from Grasso et al. indicated that negative pressure ventilation resulted in better oxygenation that was associated with increased recruitment of atelectatic lung (Grasso et al., 2008). However, Engelberts et al. found no difference in lung mechanics between NPV and PPV when conditions were strictly controlled. The effects of hemodynamics were not investigated in their study (Engelberts et al., 2012). NPV improves ventilation (Gorini et al., 2001; Vitacca et al., 2000) and breathing in patients with chronic obstructive pulmonary disease (COPD) (Gutierrez et al., 1988). Pulmonary rehabilitation can enhance the quality of life and improve clinical outcomes in patients with COPD (Hopkinson, 2017). A 5-year observation study revealed the benefits of maintenance pulmonary rehabilitation combined with NPV for patients with COPD (Huang et al., 2016). A study demonstrated the efficiency of NPV with tank respirators in patients with COPD (Corrado et al., 2009). Electrical cardiometry noninvasively measures CO, stroke volume (SV), thoracic fluid content (TFC), and other hemodynamic parameters through four electrocardiogram electrodes. TFC is calculated as the reciprocal of the total thoracic impedance (1/Zo) across the thorax (Narula et al., 2017). TFC is designed to quantify the changes in the thoracic fluid, including intravascular and extravascular fluid, rather than to measure the absolute fluid levels. TFC has been demonstrated to have a favorable correlation with fluid management (Kang et al., 2012; Sanidas et al., 2009; van de Water et al., 2005; Yancy & Abraham, 2003). Modalities of monitoring hemodynamic changes noninvasively have been growing and are used widely (Chen et al., 2014; van de Water et al., 2003). The American College of Cardiology and the American Heart Association supported the application of noninvasive evaluation for the diagnosis and treatment of chronic heart failure (Hunt, 2005). The hemodynamic effects of NPV have been extensively studied in postoperative patients (Chaturvedi et al., 2008; Shekerdemian, Bush, Lincoln, et al., 1997; Shekerdemian et al., ,1996, 1997, 1999). However, the hemodynamic effects of NPV in patients with COPD are unknown. This study aims to demonstrate the short-term response of a pulmonary rehabilitation program on hemodynamic outcomes in patients with COPD undergoing NPV through electrical cardiometry.

2 | METHOD

2.1 | Population

This center-based study was conducted in the pulmonary rehabilitation unit of Fu Jen Catholic University Hospital and was approved by the local institutional review board (C107137). We investigated the hemodynamic effects of NPV in patients with COPD who underwent the pulmonary rehabilitation program for 1 month. Patients who had exacerbation within 3 months, a tracheostomy tube, did not use NPV, required oxygen supplement during NPV, and were diagnosed with a neuromuscular disease were excluded.

2.2 | Study design

This single-arm observational retrospective study identifies patients with COPD who were treated in a pulmonary rehabilitation unit using NPV from January 1, 2018, to December 31, 2019. Hemodynamics were assessed using electrical cardiometry (ICON, Osypka Medical, Berlin, Germany), and pressure measurements were represented by atmospheric pressure. Patients with COPD in the pulmonary rehabilitation program were asked to perform supervised exercise regimens on a stationary ergometer and received a passive pulmonary expansion treatment by NPV at least once per week for a 12-week course.

2.3 | Protocol of NPV in the pulmonary rehabilitation program

In the setup of our pulmonary rehabilitation unit, a time-triggered NPV (RTX, Hayek, London, United Kingdom) was provided to patients in a sitting position for 20 min using the cuirass ventilator. NPV was set to deliver an inspiratory pressure level of −20 cm H_2O that was expelled at atmospheric pressure during the expiratory phase. The frequency of intermittent negative pressure ranged from 14 to 18 b/min with the inspiratory/expiratory ratio ranging from 1:2 to 1:4. No oxygen was provided during the NPV treatment. Hemodynamics' data were routinely monitored during each NPV treatment session while patients were in a sitting position.

2.4 | Data collection

Baseline characteristics, including age, sex, body mass index, history of smoking, pulmonary function test, the Global Initiative for Chronic Obstructive Lung Disease stage, and history of comorbidities, of all the patients. Hemodynamic data (CO, cardiac index, SV, stroke index, index of contractility [ICON], systolic time ratio, preejection period, stroke volume variation, corrected flow time, TFC, systemic vascular resistance [SVR], and left ventricular ejection time) were analyzed.

2.5 | Statistical analysis

Continuous data, including baseline characteristics, physiological parameters, and hemodynamics' data, are expressed as a mean ± standard deviation. The parametric paired *t* test was used to compare the change in physiological parameters. To adjust the

heterogeneous history of cardiac disease on hemodynamic outcomes, generalized estimating equation linear regression models were used to assess the effects of NPV. The level of significance was set at 0.05. Data were analyzed using SPSS (version 20.0 for Windows, Chicago, IL, USA).

TABLE 1 Baseline characteristic of the patients ($n = 30$)

Demographic data	
Male sex, *n*, %	26 (86.7)
Age, years	69.6 (9)
BMI, kg/m^2	24.5 (4.4)
Lung function	
FEV_1, % predict	67.4 ± 18.3
FVC, % predict	87.9 ± 14.4
FEV_1/FVC, %	56.2 ± 9.9
TLC, % predict	91.5 ± 17.5
RV, % predict	131 ± 54
GOLD stage	
Stage I, *n*, %	12 (40)
Stage II, *n*, %	14 (46.7)
Stage III, *n*, %	3 (10)
Stage IV, *n*, %	1 (3.3)
Smoking history and comorbidities	
Current or former smoker, *n* %	25 (83.3)
Cardiac disease, *n* %	14 (46.7)
Diabetes mellitus, *n* %	6 (20)
Osteoporosis, *n* %	1 (3.3)
Psychological disorders, *n* %	2 (6.7)

Note: Data presented as mean ± *SD*, and number (%).

Abbreviations: BMI, body mass index; FEV_1, forced expiratory volume in one second; FVC, forced vital capacity; GOLD, The Global Initiative for Chronic Obstructive Lung Disease; RV, residual volume; TLC, total Lung Capacity.

3 | RESULTS

Data of 30 patients with COPD who regularly received the NPV treatment in the pulmonary rehabilitation program were collected for analysis. Demographic characteristics at baseline are provided in Table 1. No significant change in respiratory rate ($p = .071$) and oxygen saturation ($p = .313$) was observed after NPV treatment compared with the resting state (Table 2). No significant change in SV was observed ($p = .168$); however, a significant decrease in HR was observed (by 4.8 bpm; $p < .001$; Table 2), resulting in a significant decrease in CO (by 0.33 L/min; $p < .001$; Table 3). Parameters, namely stroke index, ICON, systolic time ratio, pre-ejection period, and corrected flow time, were comparable before and after NPV treatment (Table 3). A significant increase in stroke volume variation, SVR, and left ventricular ejection time were noted (by 3.13%, $p = .038$, 201 dynes/sec/cm^5/m^2, $p < .001$ and 11.7 ms, $p < .001$, respectively). And a significant decrease in TFC was observed (by 0.37; $p = .016$; Table 3).

4 | DISCUSSION

The present study demonstrated the feasibility of using electrical cardiometry to measure hemodynamic change during NPV treatment in patients with COPD in a pulmonary rehabilitation unit. Almost half of the patients had heart disease-related comorbidities. Lockhat et al. revealed that CO was greater during continuous external NPV compared with PPV (2.9 vs. 2.6 L/min, $p = .02$) (Lockhat et al., 1992). Studies have detailed similar results of an increase in CO in dogs with normal lungs, injured lungs, and left heart failure (Krumpe et al., 1977; Skaburskis et al., 1987, 1990). A research team from the United Kingdom demonstrated that compared with PPV, continuous external NPV significantly improves CO in children after right heart surgery (Shekerdemian et al., 1996) and repair of tetralogy of Fallot (Shekerdemian et al., 1999). Chaturvedi et al. revealed that in patients who underwent coronary artery bypass graft surgery,

TABLE 2 Physiological parameter

			Mean change (end minus rest)		
	Rest	End of NPV		95% CI	*p*-value
Resp. rate, b/m	18.2 ± 3.4	17.3 ± 2.1	−0.8 ± 2.4	−1.71 to 0.04	.071
SpO_2, %	96.9 ± 2	97.2 ± 2.5	0.3 ± 1.6	−0.27 to 0.87	.313
HR, b/m	80.5 ± 15.8	75.7 ± 14.7	−4.8 ± 6.9	−7.27 to −2.33	<.001***
sBP, mm Hg	125.6 ± 17.3	120.0 ± 12.9	−5.6 ± 11.6	−9.74 to −1.46	.013*
dBP, mm Hg	74.9 ± 10.7	73.0 ± 9.5	−1.97 ± 7.67	−4.71 to 0.78	.171
MAP, mm Hg	91.8 ± 11.3	88.6 ± 8.7	−3.18 ± 6.77	−5.6 to −0.76	.016*

Note: Data presented as mean ± *SD*.

Paired *t* tests were used for the quantitative measurements.

Abbreviations: dBP, diastolic pressure; HR, heart rate; NPV, negative pressure ventilation; RR, respiratory rate; sBP, systolic blood pressure; SpO_2, oxygen saturation.

**$p < .01$.

*$p < .05$.

***$p < .001$.

TABLE 3 Hemodynamic outcomes

	Rest	End of NPV	Mean change (end minus rest)		
				95% CI	*p*-value
CO, L/min	3.88 ± 0.95	3.55 ± 0.9	−0.33 ± 0.4	−0.47 to 0.18	<.001***
CI, unit	0.062 ± 0.018	0.057 ± 0.018	0 ± 0.01	−0.01 to 0	.002**
SV, ml	49.3 ± 11.6	48.2 ± 11.2	−1.09 ± 4.21	−2.59 to 0.42	.168
SI, unit	0.78 ± 0.19	0.76 ± 0.19	−0.02 ± 0.07	−0.04 to 0.01	.251
ICON, unit	32.3 ± 15	30.7 ± 14	−1.85 ± 5.49	−3.81 to 0.11	.097
STR, unit	0.45 ± 0.08	0.45 ± 0.1	−0.003 ± 0.05	−0.02 to 0.015	.774
PEP, ms	123 ± 16.4	125 ± 16.7	2.13 ± 11	−1.82 to 6.08	.299
SVV, %	13.97 ± 6.61	17.1 ± 6.11	3.13 ± 8.19	0.14 to 6.14	.05
FTC, ms	318 ± 13	320 ± 13.2	1.73 ± 14.5	−3.46 to 6.93	.518
TFC, unit	17.4 ± 3.92	17.1 ± 4.22	−0.37 ± 0.85	−0.67 to −0.06	.026*
SVR, dynes·sec/cm^5/m^2	1,969 ± 453	2,170 ± 581	201 ± 297	94.9 to 308	.001**
LVET, ms	278 ± 27.3	290 ± 26	11.7 ± 17.7	5.38 to 18.2	.001**

Note: Data presented as mean ± *SD*.

Paired *t* tests were used for the quantitative measurements.

Abbreviations: CI, cardiac index; CO, Cardiac output; FTC, corrected flow time; ICON, index of contractility; LEVT, left ventricular ejection time; PEP, pre-ejection period; SI, stroke index; STR, systolic time ratio; SV, stroke volume; SVR, systemic vascular resistance; SVV, stroke volume variation; TFC, thoracic fluid content.

*$p < .05$.

**$p < .01$.

***$p < .001$.

CO, and SV increased immediately in the postoperative period (Chaturvedi et al., 2008). In patients with COPD who received the NPV treatment, CO significantly decreased at the end of treatment. The decrease in HR directly resulted in a decrease in CO. However, compared with previous studies, the sitting position in the present study might have been an influencing factor that resulted in opposite outcomes. For NPV treatment, the sitting position is the most common in the pulmonary rehabilitation unit for outpatients. Reduction in CO can be a potential risk factor for low cardiac output syndrome (Massé & Antonacci, 2005). In the present study, no significant difference in SV and ICON but a significant increase in SVR was noted. SVR is often referred to as afterload. We inferred that NPV increased the preload because SV is regulated by preload, afterload, and contractility. TFC had a significant correlation with pulmonary capillary wedge pressure (Malfatto et al., 2012) and cardiopulmonary exercise testing parameters of ventilatory efficiency in patients with congestive heart failure (Tereno Valente et al., 2011). In the present study, a significant decrease in TFC was observed, which indicated that NPV improved the lung expansion and decreased total fluid in the thoracic cavity.

This study has some limitations. To our knowledge, this is the first study to investigate the effects of NPV on the hemodynamics of patients with COPD in a pulmonary rehabilitation program. However, in contrast to our expectation, hemodynamics were found to be affected by the sitting position. Comorbidities such as cardiovascular disease are common among patients with COPD. Thus, a potential bias in this report is that we were unaware of whether patients with COPD and cardiovascular disease took medication before NPV treatment. In the present study, NPV was provided at a mandatory rate with zero end-expiratory pressure, whereas most related studies have used continuous external NPV. We did not perform the pulmonary function test routinely before and after NPV. Therefore, we have not presented lung function after NPV in this report. Further studies are warranted to determine the change in hemodynamics of patients with COPD who received NPV treatment in a different mode of ventilation, delivery pressure, and position during the treatment.

5 | CONCLUSION

NPV affected hemodynamics in patients with COPD undergoing pulmonary rehabilitation. Electrical cardiometry was applicable for determining the effects of NPV on hemodynamics. TFC significantly decreased immediately after NPV. Further investigation on changes in hemodynamics of patients with COPD who received NPV is necessary to clarify further details.

CONFLICT OF INTEREST

All authors have declared the conflict of interest. All authors certify that no funding or grant has been received for the conduct of this study and/or preparation of this manuscript.

AUTHOR CONTRIBUTIONS

K.Y.C and Y.N. conceptualized and designed the study. K.Y.C. carried out the experiment. K.Y.C. performed the analytic calculations and verified the data. K.Y.C. drafted the initial manuscript. Y.N. reviewed and revised the manuscript. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

ETHICAL APPROVAL

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the Helsinki declaration.

DATA AVAILABILITY STATEMENT

Data available on request due to privacy/ethical restrictions.

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