

# The impact of pulmonary functions on outcome of intubated patients with chronic obstructive pulmonary disease

Ahmed Metwally<sup>a</sup>, Khaled Hussein<sup>a</sup>, Ashraf Zein El-Abdeen<sup>a</sup>, Ahmed Hamed<sup>a</sup>, Azaa Ez-Eldeen<sup>b</sup>

**Background** Assessment of lung mechanics and respiratory muscle determinants is considered to be useful for adjustment of ventilator settings to minimize dynamic hyperinflation in patients with chronic obstructive pulmonary disease (COPD).

**Objective** The aim of this study is to assess the impact of pulmonary functions on the outcome of mechanically ventilated COPD patients.

**Patients and methods** This study was carried out in the respiratory ICU, chest department, Assiut University Hospital. A total of 47 mechanically ventilated COPD patients were recruited into the study. The patients were divided into two groups on the basis of outcomes: successful group (36 patients) and failed group (11 patients). Analyses of different lung mechanics and respiratory muscle determinants were carried out for both groups.

**Results** There was no significant difference in age between both the groups. Arterial blood gases analysis showed a significantly decreased pH, PaO<sub>2</sub>, and SaO<sub>2</sub> in the failed group and a significantly increased PaCO<sub>2</sub> in the failed group after 1, 24, and 48 h of mechanical ventilation. Values of lung mechanics in the failed group, compared with the successful group, showed a significantly lower respiratory compliance (29.18 ± 1.47 vs. 45.92 ± 4.08 ml/cm H<sub>2</sub>O), a significantly higher respiratory resistance (24.73 ± 1.19 vs. 22.39 ± 1.10 cm H<sub>2</sub>O/L/S),

and a significantly higher intrinsic positive end expiratory pressure (8.36 ± 0.67 vs. 7.58 ± 0.94 cm H<sub>2</sub>O). Values of respiratory muscle determinants in the failed group showed a significantly lower negative inspiratory force (-17.18 ± 2.82 vs. -26.44 ± 3.32 cm H<sub>2</sub>O), a significantly lower vital capacity (382.27 ± 102.75 vs. 810.00 ± 133.03 ml), and a significantly higher occlusion pressure P0.1 (6.09 ± 1.04 vs. 1.72 ± 0.66 cm H<sub>2</sub>O).

**Conclusion** Pulmonary functions in mechanically ventilated COPD patients including lung mechanics and respiratory muscle determinants had an important impact on outcome. A significant decrease in respiratory compliance, negative inspiratory force, and vital capacity with a significant increase in respiratory resistance, intrinsic positive end expiratory pressure, and occlusion pressure P0.1 was observed in the COPD group with failed extubation. *Egypt J Broncho* 2015 9:125–132  
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*Egyptian Journal of Bronchology* 2015 9:125–132

**Keywords:** intrinsic positive end expiratory pressure, lung mechanics, respiratory muscle determinants

Departments of <sup>a</sup>Chest <sup>b</sup>Clinical Pathology, Faculty of Medicine, Assiut University, Assuit, Egypt

Correspondence to Khaled Hussein Ahmed, Chest Department, Assiut University Hospital, Assuit 71111, Egypt  
Tel: +201006184912;  
e-mail: khaldhussein@yahoo.com

**Received** 13 November 2014 **Accepted** 16 January 2015

## Introduction

Chronic obstructive pulmonary disease (COPD) has become a major epidemic with an increasing incidence worldwide, particularly in developing countries [1], and is now one of the leading causes of death and disability, with a prevalence of more than 10% in individuals above 40 years of age in most countries [2].

COPD is a disease characterized by poorly reversible airflow limitation of the airways that is usually progressive and associated with an abnormal inflammatory response in the lung [3]. The abnormal inflammatory response is usually triggered by smoking [4] or exposures to other environmental irritants [5] that interact with genetic factors [6], leading to both airway and systemic inflammation, resulting in airway injury and lung damage.

COPD is a disease characterized by airway narrowing that produces a critical increase in airway resistance,

especially during exhalation. Breathing through narrowed airways imposes an additional load on the respiratory muscles [7].

In the presence of high resistance to expiratory flows and short expiratory times, the respiratory system cannot return to its resting volume at the end of exhalation. As a result, a new resting state is established, such that there is a positive recoil pressure [(intrinsic positive end expiratory pressure (PEEP<sub>i</sub>))] at the end of expiration [8].

This state of air trapping or dynamic hyperinflation is common in patients with COPD. Initially, hyperinflation tends to keep the airways open, reduces airway resistance, increases elastic recoil, and tends to improve expiratory flow. However, hyperinflation exerts several deleterious effects. The positive pressure within regions of hyperinflated lung increases the mean intrathoracic pressure and causes the inspiratory

muscles to operate at a higher than resting lung volume. Thus, dynamic hyperinflation places the respiratory muscles at a considerable mechanical disadvantage and further impairs respiratory muscle function [9].

PEEP<sub>i</sub> also imposes a huge inspiratory threshold load because a negative intrapleural pressure equal to the level of PEEP<sub>i</sub> has to be generated before inspiratory flow begins within alveoli [10].

In emphysema, although static compliance is high because of the overall effect of loss of elastic recoil, dynamic compliance is often low on account of the airways obstruction and because of loss of traction of elastic tissue on the airways [11]. Pulmonary hyperinflation along with an increase in the work of breathing that occurs in COPD are the main contributing factors to respiratory muscle dysfunction [12].

During mechanical ventilation, a study to minimize dynamic hyperinflation is the main goal to improve the weaning outcome. Assessment of lung mechanics and respiratory muscle determinants is considered to be useful for adjustment of ventilator settings to minimize dynamic hyperinflation in patients with COPD.

### Patients and methods

This study was carried out in the respiratory intensive care unit, chest department, Assiut University Hospital. The study included mechanically ventilated COPD patients with respiratory failure who fulfilled the following inclusion criteria.

#### Inclusion criteria

- (1) Disturbed conscious level because of hypercapnia.
- (2) pH less than 7.25 with increased partial pressure of arterial carbon dioxide (PaCO<sub>2</sub>).

#### Exclusion criteria

- (1) Age: younger than 20 years or older than 80 years.
- (2) Pregnancy.
- (3) Disturbed conscious level because of metabolic causes such as renal and hepatic disorders.
- (4) Patients with neurological deficit.
- (5) Patients with pulmonary vascular diseases.
- (6) Patients with primary cardiac diseases.

#### All patients at admission to the respiratory intensive care unit were subjected to the following:

##### Arterial blood gases

These were obtained by blood samples from the radial artery and analyzed using a blood gases analyzer (Rapid lab850; CHIRON/Diagnostics; critical care systems), and included pH, PaCO<sub>2</sub>, bicarbonate level,

partial pressure of arterial oxygen (PaO<sub>2</sub>), and arterial oxygen saturation (SaO<sub>2</sub>).

##### Laboratory assessment

- (1) Complete blood picture.
- (2) Serum urea and creatinine.
- (3) Serum albumin.
- (4) Specific tests include high-sensitivity C-reactive protein and tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ).

#### During mechanical ventilation, patients were subjected to the following:

- (1) Continuous monitoring of heart rate, blood pressure (systolic and diastolic), respiratory rate, and arterial oxygen saturation using a pulse oximeter (SpO<sub>2</sub>).
- (2) Arterial blood gases were assessed for all patients 30 min, 24, and 48 h after mechanical ventilation.
- (3) Ventilatory parameters were determined and included both setting parameters and monitored parameters.

##### Setting parameters

- (1) Tidal volume: 6–8 ml/kg of ideal body weight.
- (2) Machine rate: 8–10 breath/min.
- (3) Fraction of inspired oxygen (FiO<sub>2</sub>).
- (4) PEEP: We started with PEEP 5 cm H<sub>2</sub>O, and then we increased PEEP to equal 80% of PEEP<sub>i</sub> if it was still high despite the prolongation of the *I-E* ratio.
- (5) *I-E* ratio: 1–4 at least.

##### Monitored parameters

- (1) Peak airway pressure.
- (2) Plateau pressure.
- (3) Exhaled tidal volume: in liters.
- (4) Respiratory rate: breath/min.
- (5) Minute ventilation: liters/min.
- (6) Graphic display included pressure, flow, and volume scalars.

#### Mechanics of breathing

Mechanics of breathing were recorded after 24 and 48 h of mechanical ventilation.

- (1) Respiratory compliance: This was assessed by pressing the inspiratory pause button of the ventilator. This is automatically calculated and displayed as the ratio between the exhaled tidal volume and the difference between plateau pressure and the unoccluded PEEP.
- (2) Respiratory resistance: This was assessed by pressing the inspiratory pause button of the

ventilator. Resistance was automatically calculated and displayed by dividing the plateau pressure subtracted from peak pressure by flow.

- (3) Intrinsic positive end expiratory pressure: This is displayed automatically by pressing the expiratory pause button of the ventilator.

### Respiratory muscle determinants

Respiratory muscle determinants were recorded after 48 h of mechanical ventilation.

- (1) Negative inspiratory force (NIF): We measured NIF three times and the most negative result was taken. A negative force that was weaker than  $-20$  cm H<sub>2</sub>O implies respiratory muscle weakness and difficult extubation (Fig. 1).
- (2) Vital capacity (VC): We measured VC three times and the average of the results was obtained.
- (3) Occlusion pressure (P0.1): We measured P0.1 five times over a period of 60–90 s and the average of these measurements was obtained.

### Medical treatment

Medical treatment included nebulized salbutamol and ipratropium bromide, which were administered through a piece connected to the ventilator circuit near the mouth. Intravenous hydrocortisone, 100 mg/12 h, was administered to all COPD patients until discharge from the ICU. Theophylline was administered intravenously 6 mg/kg over 20–30 min, followed by a continuous infusion of 0.6 mg/kg/h. Antibiotics were administered as combination therapy with cefepime 1 g or ceftazidime 1 g/12 h plus levofloxacin 500 mg/24 h or amikacin 500 mg/12 h as all our patients had bacterial infection.

According to the weaning outcome, we classified patients into two groups: successful group and failed group.

### Statistical analysis

Data were recorded to statistical package for social science, version 16. Data were described as mean  $\pm$  SD

Fig. 1



Measurement of negative inspiratory force during MV.

and frequencies for nominal data. The correlation between continuous variables was determined using Spearman's correlation coefficient. *P* less than 0.05 was considered significant.

### Results

Figure 2 shows the outcome of mechanically ventilated COPD patients and indicated that 76.6% of patients had successful outcome, whereas 23.4% of patients had failed outcome.

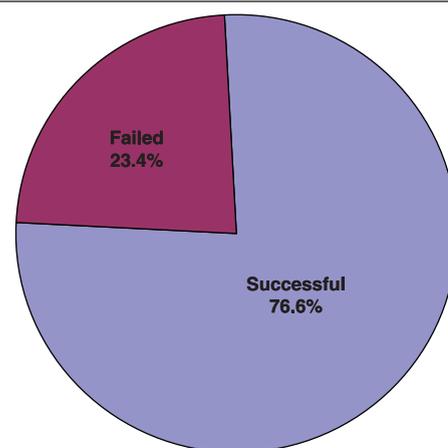
Table 1 shows the demographic and historical data of both groups of COPD patients and indicated a significantly increased smoking index in the failed group. Also, there was a significantly increased number of exacerbations last year, previous hospitalizations, and incidence of previous mechanical ventilation in the failed group.

Table 2 shows the baseline clinical and gasometric data. A significantly decreased pH was found in the failed group, with no significant difference in the other baseline clinical and gasometric data in both groups of COPD.

As shown in Table 3, from baseline laboratory data in both groups of COPD, it is clear that there was a significantly increased TNF- $\alpha$  and high-sensitivity C-reactive protein in the failed group, whereas there was a significantly decreased serum albumin in the failed group.

Figure 3a–d shows follow-up of arterial blood gases in both groups of COPD patients, with significantly ( $P < 0.01$ ) decreased pH, PaO<sub>2</sub>, and SaO<sub>2</sub> in the failed group and a significantly ( $P < 0.001$ ) increased PaCO<sub>2</sub> in the failed group after 1, 24, and 48 h of mechanical ventilation.

Fig. 2



Outcome of mechanically ventilated COPD patients. COPD, chronic obstructive pulmonary disease.

Figure 4a–f show ventilator parameters in both groups of COPD patients. There was a significant ( $P < 0.01$ )

**Table 1 Demographic and historical data of both groups of COPD**

	Successful (n = 36)	Failed (n = 11)	P-value
Sex: M/F	30/6	10/1	0.894
Age (mean ± SD)	64.53 ± 7.15	68.73 ± 10.76	0.139
Smoking [N (%)]	29 (80.6)	10 (90.9)	0.733
Smoking index (mean ± SD)	34.76 ± 6.98	62.30 ± 2.26	0.000
Exacerbation last year (mean ± SD)	3.58 ± 1.08	5.27 ± 0.90	0.000
Hospitalization last year (mean ± SD)	1.72 ± 0.88	2.45 ± 0.82	0.018
Previous MV [N (%)]	7 (19.4)	10 (90.9)	0.000

COPD, chronic obstructive pulmonary disease.

**Table 2 Baseline clinical and gasometric data**

	Successful (n = 36)	Failed (n = 11)	P-value
Respiratory rate	28 ± 4.2	29 ± 5.3	0.145
Heart rate	130 ± 9.1	131 ± 9.8	0.231
pH	7.23 ± 0.04	7.18 ± 0.03	0.001
PaCO <sub>2</sub>	85.64 ± 11.77	88.73 ± 9.95	0.435
PaO <sub>2</sub>	42.67 ± 3.69	42.00 ± 2.68	0.582
SaO <sub>2</sub>	71.25 ± 7.63	67.09 ± 6.91	0.113

increase in the respiratory rate, minute ventilation, peak pressure, and plateau pressure in the failed group. However, there was a significant ( $P < 0.01$ ) decrease in the exhaled tidal volume and PaO<sub>2</sub>/FiO<sub>2</sub> in the failed group.

Table 4 shows the follow-up of lung mechanics in both groups of COPD patients. A significantly decreased respiratory compliance and a significantly increased respiratory resistance and PEEP<sub>i</sub> were found in the failed group.

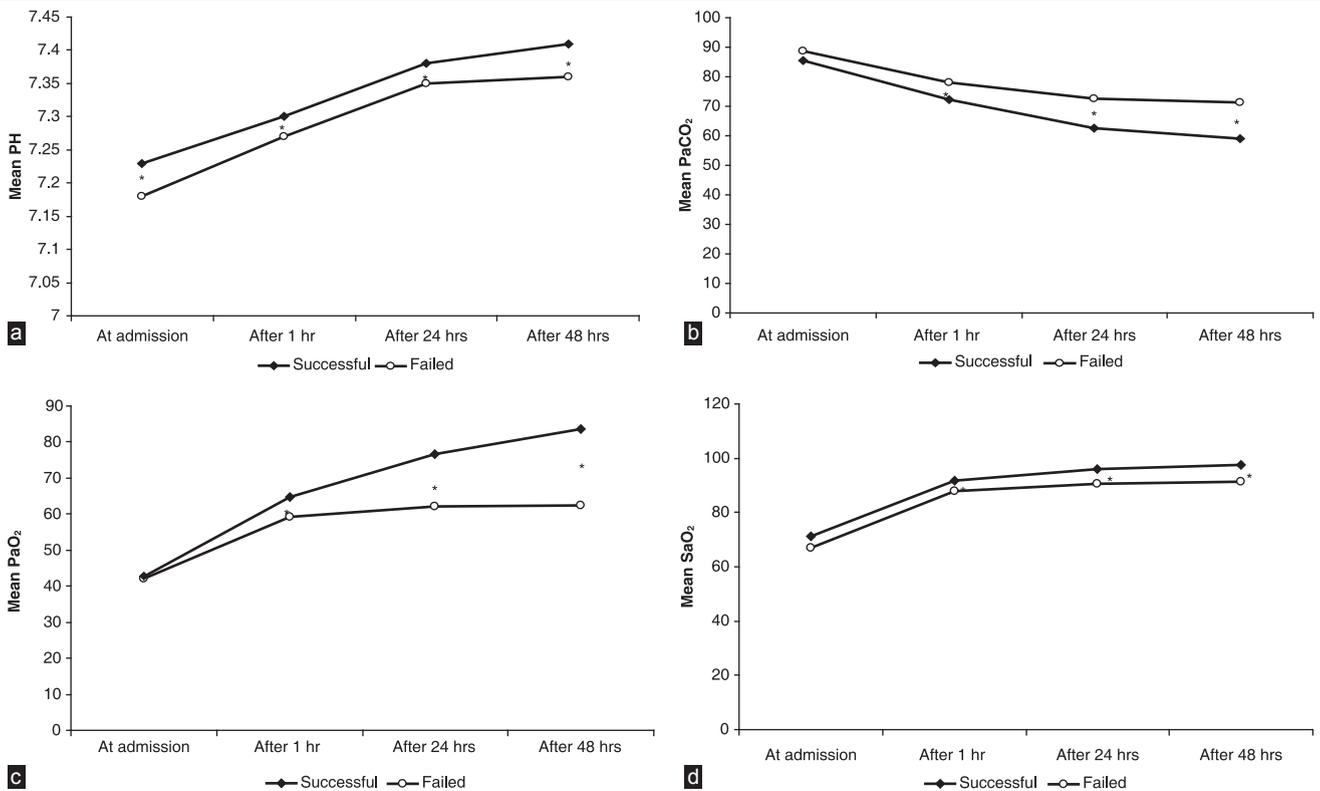
Table 5 shows the respiratory muscle determinants in both groups of COPD patients. Significantly decreased NIF and VC, and a significantly increased P0.1 were found in the failed group.

**Table 3 Baseline laboratory data**

	Successful (n = 36)	Failed (n = 11)	P-value
WBC (10 <sup>6</sup> /ml)	13.9 ± 6.7	16.0 ± 8.3	0.190
Hemoglobin (g/dl)	15.5 ± 2.1	16.4 ± 2.2	0.223
Creatinine (mg/dl)	1.5 ± 0.7	1.7 ± 1.2	0.270
Serum albumin	28.53 ± 5.47	22.73 ± 3.41	0.002
hsCRP	73.61 ± 8.15	106.91 ± 16.47	0.000
TNF-α	121.53 ± 31.45	262.09 ± 36.77	0.000

hsCRP, high-reactive protein; TNF-α, tumor necrosis factor-α; WBC, white blood cells.

**Fig. 3**



(a) Follow-up of the mean pH in both groups of COPD. (b) Follow-up of the mean PaCO<sub>2</sub> in both groups of COPD. (c) Follow-up of the mean PaO<sub>2</sub> in both groups of COPD. (d) Follow-up of the mean SaO<sub>2</sub> in both groups of COPD. COPD, chronic obstructive pulmonary disease.

Figure 5 shows that there was a significant positive correlation ( $r = 0.958, P = 0.000$ ) between NIF and VC in both groups of COPD patients.

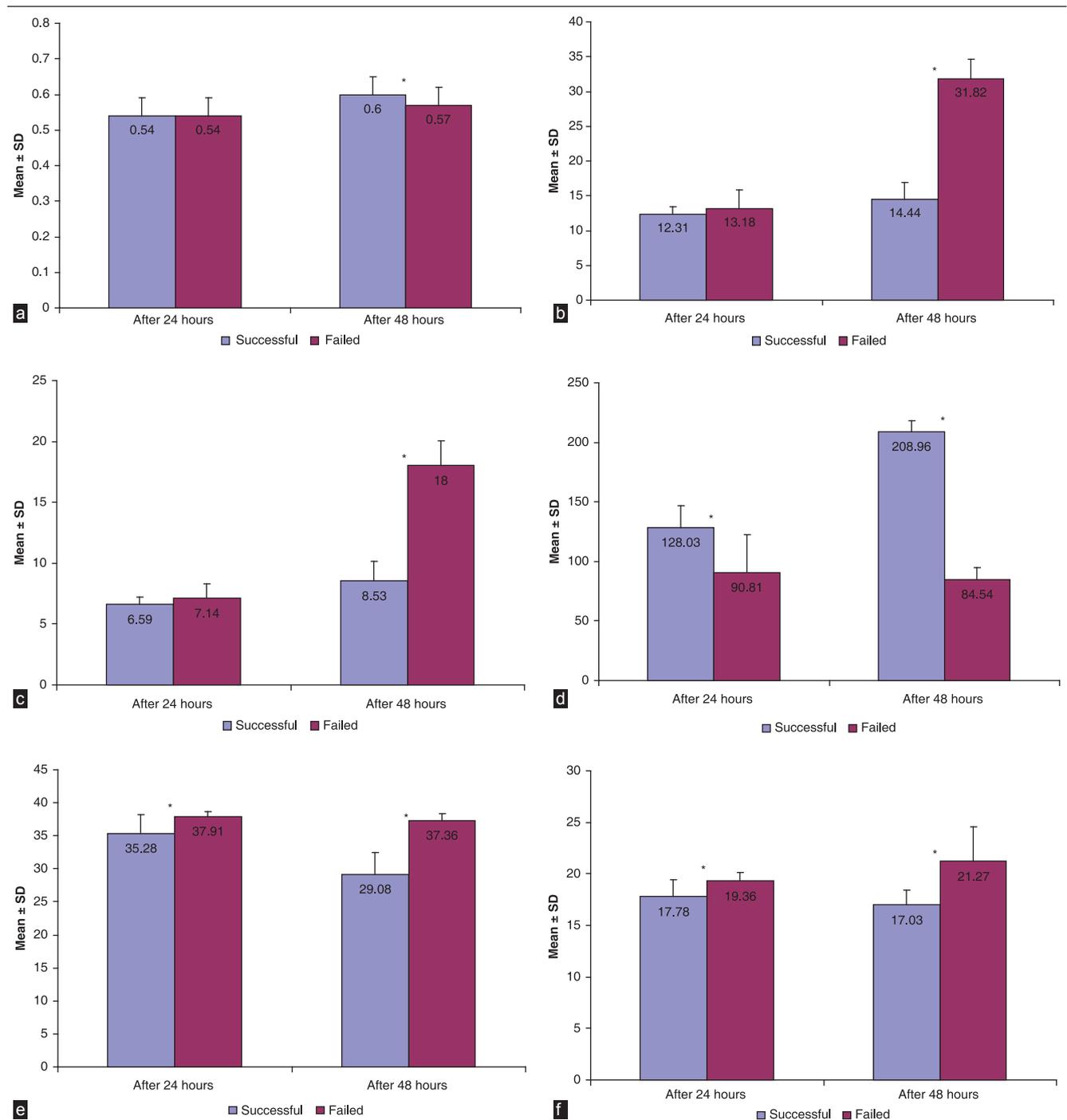
Figure 6 shows that there was a significant negative correlation ( $r = 0.970, P = 0.000$ ) between NIF and duration of MV.

Figure 7 shows that there was a significant positive correlation ( $r = 0.441, P = 0.002$ ) between peak airway pressure and respiratory resistance.

### Discussion

Increased number of exacerbations in the last year, more frequent hospitalizations, and previous mechanical

**Fig. 4**



(a) Follow-up of the mean exhaled tidal volume in both groups of COPD. (b) Follow-up of the mean respiratory rate in both groups of COPD. (c) Follow-up of the mean minute ventilation in both groups of COPD. (d) Follow-up of the mean PaO<sub>2</sub>/FiO<sub>2</sub> in both groups of COPD. (e) Follow-up of the mean peak pressure in both groups of COPD. (f) Follow-up of the mean plateau pressure in both groups of COPD. COPD, chronic obstructive pulmonary disease.

**Table 4 Follow-up of lung mechanics of both groups of COPD**

	Successful (n = 36) (mean ± SD)	Failed (n = 11) (mean ± SD)	P-value
Compliance (24 h)	45.92 ± 4.08	29.18 ± 1.47	0.000
Compliance (48 h)	55.42 ± 4.21	30.82 ± 3.03	0.000
Resistance (24 h)	22.39 ± 1.10	24.73 ± 1.19	0.000
Resistance (48 h)	16.72 ± 1.14	19.36 ± 1.21	0.000
PEEPi (24 h)	7.58 ± 0.94	8.36 ± 0.67	0.0014
PEEPi (48 h)	1.72 ± 0.64	2.91 ± 0.30	0.000

COPD, chronic obstructive pulmonary disease; PEEPi, intrinsic positive end expiratory pressure.

**Table 5 Respiratory muscle determinants of both groups of COPD**

	Successful (n = 36) (mean ± SD)	Failed (n = 11) (mean ± SD)	P-value
NIF	-26.44 ± 3.32	-17.18 ± 2.82	0.000
VC	810.00 ± 133.03	382.27 ± 102.75	0.000
P0.1	1.72 ± 0.66	6.09 ± 1.04	0.000

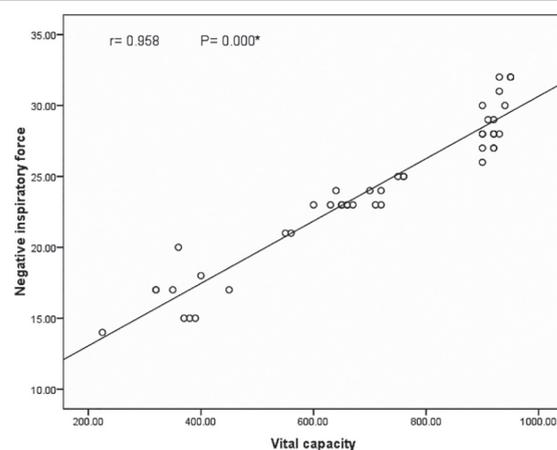
COPD, chronic obstructive pulmonary disease; NIF, negative inspiratory force; P0.1, occlusion pressure; VC, vital capacity.

ventilation are associated with an increased risk of failed extubation [13]. This is consistent with our results.

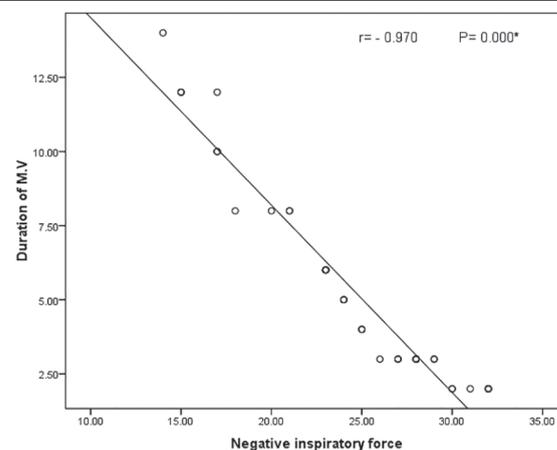
Our results indicated a significantly increased smoking index in the failed group; these results are in agreement with those of Bakr *et al.* [14], who reported that there was a significant difference in the smoking index in both groups of COPD: 38 ± 33.5 packs/years in the successful group and 62.82 ± 29.68 packs/years in the failed group.

Follow-up of arterial blood gases was performed in both groups of COPD patients. Significantly decreased pH, PaO<sub>2</sub>, and SaO<sub>2</sub> and a significantly increased PaCO<sub>2</sub> were found in the failed group; these results are in agreement with those of Ucgun *et al.* [15], who reported that COPD patients with failed extubation were characterized by a significantly decreased PaO<sub>2</sub> (52.8 ± 7.0 mmHg), a significantly decreased pH (7.20 ± 0.97), and a significantly increased PaCO<sub>2</sub> (75.8 ± 10.1 mmHg). Also, Bakr *et al.* [14] reported an increase in mortality with derangement of all arterial blood gases items in patients with COPD, with mean pH (7.24 ± 0.03 and 7.20 ± 0.01), mean PaCO<sub>2</sub> (54.8 ± 6.39 and 76.1 ± 11.1 mmHg), and mean PaO<sub>2</sub> (54.8 ± 7.1 and 50.2 ± 4.9 mmHg) for survivors and nonsurvivors, respectively; this was statistically significant ( $P < 0.05$ ).

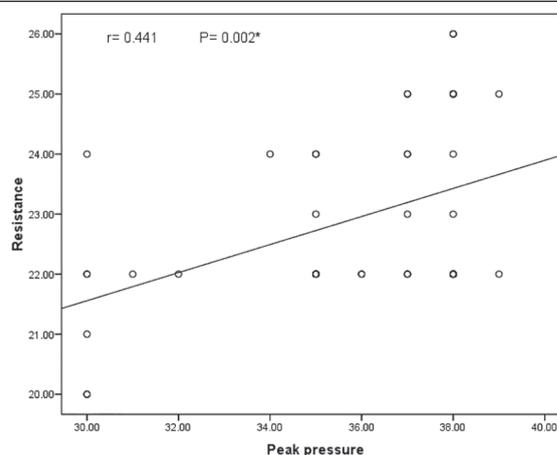
There was a statistically significant decrease in PaO<sub>2</sub>/FiO<sub>2</sub> in the failed group; these results are in agreement with those of Park and Koh [16], who reported a significantly decreased PaO<sub>2</sub>/FiO<sub>2</sub> in the failed group.

**Fig. 5**

A significant positive correlation between NIF and VC. NIF, negative inspiratory force; VC, vital capacity.

**Fig. 6**

A significant negative correlation between NIF and duration of MV. NIF, negative inspiratory force.

**Fig. 7**

A significant positive correlation between peak pressure and resistance.

Also, Madkour and Adly [17] reported that the mean PaO<sub>2</sub>/FiO<sub>2</sub> in COPD patients with failed extubation

was significantly lower than that in the group of patients with successful extubation.

Measurement of lung mechanics indicated a significantly decreased respiratory compliance and significantly increased respiratory resistance and PEEP<sub>i</sub> in the failed group. These results are in agreement with those of Jubran and Tobin [18], who reported that COPD patients who failed the weaning trial of mechanical ventilation developed worsening of lung mechanics compared with patients who tolerated the weaning trial. Also, Lin *et al.* [19] reported that patients who had better underlying lung mechanics (higher respiratory compliance and lower PEEP<sub>i</sub>) had better chances of weaning from mechanical ventilation.

Nava *et al.* [20] reported that respiratory compliance measured in the first 24 h from intubation enabled good differentiation between the patients who were weaned successfully and those who failed.

Our results indicated a significant increase in resistance and PEEP<sub>i</sub> in the failed group. These results are in agreement with those of Broseghini *et al.* [21], who reported that patients with acute exacerbation of COPD were characterized by high respiratory resistance and severe pulmonary hyperinflation, with 'intrinsic' PEEP (PEEP<sub>i</sub>) up to 22 cm H<sub>2</sub>O. Increased respiratory resistance and pulmonary hyperinflation were associated with increased incidence of failed extubation.

Increased respiratory resistance in patients with COPD can be explained by the fact that in advanced COPD, the alveolar attachments that normally keep the smaller airways open by radial traction are lost [22], increased airway collapsibility occurs because of destruction of the lung parenchyma, and there is loss of lung elastic recoil [23].

In respiratory muscle determinants, significantly decreased NIF and VC, and a significantly increased P0.1 were found in the failed group; these results are in agreement with those of Graham and Nausherwan [24], who reported that patients with obstructive pulmonary disorders had P0.1 (2.52 ± 1.03 cm H<sub>2</sub>O). The obstructive group was divided into a successful weaning group, P0.1 (2.01 ± 1.21 cm H<sub>2</sub>O), and a failed weaning group, P0.1 (6.35 ± 2.95 cm H<sub>2</sub>O). Thus, the results of this study showed that increased P0.1 value was linked to a poor outcome in the weaning trial (all patients who had P0.1 greater than 6.4 cm H<sub>2</sub>O failed to wean), whereas decreased P0.1 value was linked to a successful outcome in the weaning trial.

Also, Nava *et al.* [20] reported that patients with COPD had P0.1 (4.1 ± 1.2 cm H<sub>2</sub>O), the group of COPD patients with successful weaning had P0.1 (3.5 ± 1.3 cm H<sub>2</sub>O), and the group of COPD patients in whom weaning failed had P0.1 (5.2 ± 0.5 cm H<sub>2</sub>O).

Li *et al.* [25] documented that NIF among the group of COPD patients who failed extubation was -13 ± 3 cm H<sub>2</sub>O, whereas NIF in the group of COPD patients with successful extubation was -21 ± 4 cm H<sub>2</sub>O.

There was a significant positive correlation between NIF and VC; this result is in agreement with that of Prigent *et al.* [26], who reported that there was a positive correlation between NIF and VC. NIF can be used to evaluate patients with hypoventilation because of inspiratory muscle weakness, whereas VC is limited by weakness of both inspiratory and expiratory muscles and by compliance of the respiratory system.

Lawn *et al.* [27] reported that decreased VC less than 15 ml/kg and NIF less than -30 cm H<sub>2</sub>O may be predictors for invasive mechanical ventilation.

## Conclusion

Pulmonary functions in mechanically ventilated COPD patients including lung mechanics and respiratory muscle determinants had an important impact on outcome. A significant decrease in respiratory compliance, NIF, and VC, with a significant increase in respiratory resistance, intrinsic PEEP, and occlusion pressure P0.1, was observed in the COPD group with failed extubation.

## Acknowledgements

Conflicts of interest  
None declared.

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