

The prognostic value of the dead-space fraction and other physiological parameters in the weaning process of mechanical ventilation in patients with obstructive air flow

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Background Patients with obstructive air flow including chronic obstructive lung diseases and bronchial asthma use a substantial proportion of mechanical ventilation (MV) in the ICU, and their overall mortality with ventilator support can be significant. From the pathophysiological standpoint, they have increased airway resistance, pulmonary hyperinflation, and high pulmonary dead space, leading to an increased work of breathing. MV is an integral part of the treatment for acute respiratory failure.

Aim of the work The present study aimed to demonstrate the prognostic value of ventilatory parameters including that of the dead-space fraction (DSF), end-tidal carbon dioxide (ETCO₂), lung mechanics, and gas exchange during the application of MV.

Patients and methods Forty consecutive patients admitted to the ICU with acute respiratory failure due to chronic obstructive lung diseases and acute severe asthma were enrolled in the study. Lung mechanics (compliance and airway resistance), DSF, ETCO₂, and arterial blood gases were measured at the following times: on admission to the ICU, initially, and finally before extubation.

Results Successfully weaned and survivors represent 60% ($n = 24$) of all patients included in this study. They had a

lower MV duration at a mean of 3.75 days \pm 1.8 SD. Logistic regression analysis revealed a significant association between the MV duration, pH more than 7.32, and dynamic compliance on the one hand and extubation failure on the other, but no significant association was found between the DSF and extubation failure, with odds ratio equal to 2.08 (95% confidence interval: 0.05–85.78, $P = 0.7$).

Conclusion We concluded that DSF is not an influential predictor of extubation failure in patients with obstructive air flow, whereas dynamic compliance plays a strong prognostic role in the weaning process.

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Introduction

Chronic obstructive airway disease includes not only chronic obstructive lung diseases (COPD) and chronic asthma, but also disorders such as bronchiectasis, pneumoconiosis, and post-tuberculosis lung disease. COPD is a major health and economical problem of growing prevalence; in the year 1990, it represented the sixth most common cause of deaths worldwide, and by 2020, the disease is expected to become the third most common cause of death. The mortality associated with asthma is also considerable, with an estimated one to eight deaths per 100 000 inhabitants/year. Adequate triage or screening for admission to the ICU is therefore essential among patients at risk [1].

Although the lung mechanics are similar under both conditions (asthma and COPD), there are some physiopathological differences that should be mentioned. In effect, COPD is characterized by greater airway collapse and a loss of elastic rebound of the lungs [2], whereas asthma is characterized by hypertrophy of the airway walls secondary to inflammation, lesser airway collapse despite the considerable decrease in airway

caliber (central involvement vs. peripheral involvement in COPD), and a generally reversible obstruction that can be minimal or lacking in long-evolving asthma [3].

If the ventilator demand exceeds the capacity of the respiratory muscles, acute respiratory failure results [4]. Although a general indication for almost all conditions, patients of this kind should not receive invasive mechanical ventilatory support, because the mortality increases significantly as a result. However, the failure of noninvasive mechanical ventilation (MV) (effective in at least 75% of the cases, particularly in COPD) can lead to a fatal outcome [5].

Measurements of respiratory mechanics are simple to perform and provide useful and relevant information for severity assessment and ventilator management.

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They are really reliable only under passive conditions of ventilation, in which plateau pressure (P_{plat}) monitoring is essential for adequate ventilatory management [6]. Resistance of the airways is described as an obstruction to airflow provided by the conducting airways, resulting mainly from the larger airways (down to division 6–7). Airway resistance to flow is present during both inspiration and expiration and the energy required to overcome it represents the actual work of breathing [7].

The expiratory capnogram provides qualitative information on the wave form patterns associated with MV and quantitative estimation of the end-tidal carbon dioxide ($ETCO_2$). Dead-space fraction (DSF) can be calculated easily from the Enghoff modified caution of the Bohr equation using the arterial partial pressure of carbon dioxide ($PaCO_2$) with the assumption that $PaCO_2$ is similar to alveolar PCO_2 . DSF is increased in COPD, pulmonary embolism, decreased cardiac output, and hypovolemia. A high DSF represents an impaired ability to excrete CO_2 because of any kind of V/Q mismatch [6,8].

Few studies have been conducted to assess the value of physiological factors as a weaning predictor. The present work aimed to study the ventilatory parameters including of $ETCO_2$, lung mechanics, DSF, and gas exchange in COPD, and bronchial asthma patients during the application of MV to assess whether the length of MV in these patients can be predicted by the measurement of these parameters.

Patients and methods

Patients

The present study included 40 patients aged between 45 and 78 years, who were admitted to the ICU in El-Minia University Hospital during a period from July 2012 to August 2013 for the management of acute respiratory failure due to COPD and acute severe asthma.

The protocol was approved by the institutional ethics committee, and informed consent was obtained from the patients or their next of kin. Exclusion criteria were the association of pulmonary edema, hemodynamic instability, and the presence of intrathoracic drainage. Data were collected in all patients requiring MV; criteria used were similar to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) and the ATS guidelines for intubations [9]. The following data were recorded: age, sex, smoking history, comorbidity, and pulmonary function tests.

Study protocol

All patients were sedated and mechanically ventilated using a Puritan Bennett 840 microprocessor ventilator (Germany). Initially, the patients were placed on volume-control constant flow mode and ventilated with a tidal volume (V_t) ranging from 6 to 8 ml/kg body weight and a respiratory rate from 10 to 12/min supplied by volume control mode (controlled mandatory ventilation), and initial inspired fraction (FiO_2) 1.0 (100%). The extrinsic best positive end-expiratory pressure (PEEP) is the maximum amount of PEEP that can be applied without increasing the peak airway pressure during volume preset MV [10]. For all patients, the started parameters were usually changed, according to each situation after the first arterial blood gases (ABGs) analysis. All patients were under cardiac and respiratory monitoring (heart rate, respiratory rate, O_2 saturation, and $ETCO_2$). When passive ventilation was obtained and respiratory muscle activity was resumed (i.e. spontaneous breathing trial), the patient started to trigger the ventilator at his or her usual rate. Then, a spontaneous breathing trial using a T tube was performed during a 30-min period. Criteria for passing the spontaneous breathing trial were those defined by the Sixth International Consensus Conference on Intensive Care Medicine [11]. Weaning was decided when the following weaning criteria were met [11–13].

- (1) Reversal of the cause of MV.
- (2) Hemodynamic stability: that is, no clinically important hypotension and no requirement for vasopressors or a requirement only for low-dose vasopressor therapy (e.g. dopamine or dobutamine <5 l/kg/min).
- (3) Patient capable of initiating an inspiratory effort.
- (4) No electrolyte disturbances and no sedation or narcotics.
- (5) Good nutritional status and no clinically evident myopathy or neuropathy.
- (6) Corrected reversible causes of weaning failure such as sepsis or heart failure.

The patients were assessed for the following outcomes:

- (1) Weaning success was defined as an absence of tachypnea more than 35, tachycardia more than 120, PaO_2/FiO_2 more than 150, FiO_2 less than 0.4–0.5, pH more than 7.32, and the patient is not reintubated and ventilated within 48 h of extubation, rapid shallow breathing index (respiratory rate/ V_t) less than 105 [11].
- (2) Days of MV.
- (3) The length of ICU stay.

Extubation failure was defined as reintubation or noninvasive ventilation within 48 h after extubation [14].

The arterial sample was analyzed immediately in the blood gas analyzer ALB 30 (Radiometer, Copenhagen, Denmark) present in the respiratory ICU. ABG was performed initially and before extubation. Follow-up by ABGs after MV was carried out till the discharge of the patient.

The ETCO_2 was measured using the noninvasive cardiac output monitor (DS5000A Patient Monitor; Digital Science Technology, 2535 W. 237th Street, Suite 108, Torrance, CA 90505, USA) on the expiratory side of the circuit's endotracheal tube connector. After proper calibration and an equilibration time of 20 min with stable hemodynamic and respiratory variables, ETCO_2 were determined and the highest reading was recorded.

Estimation of the alveolar DSF using arterial and ETCO_2 : $\text{PaCO}_2 - \text{ETCO}_2 / \text{PaCO}_2$ was ~59.5% of $\text{VD}_{\text{alv}} / \text{Vt}_{\text{alv}}$ (Bohr-Fowler) [15–17].

Lung mechanics (resistance and elastance) were measured by the technique of rapid airway (end-inspiratory and end-expiratory) occlusion using standard formulas [18].

This produces a rapid decrease in the peak pressure, and after 3–5 s, the pressures at the ventilator and the alveoli equilibrate at which point the pressure curve plateaus off. The difference between the peak pressure and the P_{plat} yields the total resistance of the respiratory tract.

Measuring of airway resistance

The resistance to airflow during inspiration was determined by dividing the peak inspiratory flow rate (V_{insp}) into the pressure needed to overcome the resistance to airflow ($\text{PIP} - \text{P}_{\text{plat}}$). $\text{R}_{\text{insp}} = \text{PIP} - \text{P}_{\text{plat}} / \text{V}_{\text{insp}}$, where R_{insp} is the airway resistance during inspiration and V_{insp} is the peak inspiratory flow rate.

This resistance represents the summed resistance of the connector tubing, the tracheal tube, and the airways. However, changes in R_{insp} should represent changes in the airway resistance as long as the inspiratory flow rate and the size of the tracheal tube and the connector tubing are constant.

Measurement of compliance during MV

Compliance was calculated during MV by dividing the volume of air delivered to the patient through the ventilator by the obtained pressure.

Static compliance is easily calculated as the ratio between Vt and the P_{plat} minus PEEP. Elastance is the reverse of compliance (how much pressure we need for a given volume). A low compliance-high elastance reflects mainly a small aerated lung available for ventilation [6].

The Vt delivered had to be corrected by the subtraction of the noncompressible volume of the tubing system that connects the patient to the mechanical ventilator. We can assess auto-PEEP by airway occlusion at end expiration. PEEP, whether external applied or auto-PEEP, should be subtracted from the pressure obtained by the manometer.

$$\text{C}_{\text{st}} = \frac{\text{Vt} - 3(\text{PIP})}{\text{P}_{\text{plat}} - \text{total PEEP}},$$

$$\text{C}_{\text{dyn}} = \frac{\text{Vt} - 3(\text{PIP})}{\text{PIP} - \text{total PEEP}},$$

where PIP is the peak inspiratory pressure, P_{plat} is the plateau pressure, total PEEP = (auto-PEEP+applied PEEP), C_{dyn} is the dynamic compliance

Statistical analysis

The collected data were coded, tabulated, and statistically analyzed using statistical package for social sciences program, software version 20 (SPSS, Inc., Chicago, IL).

Descriptive statistics were performed for numerical data by mean, SD, and minimum and maximum of the range, whereas they were carried out for categorical data by the number and percentage. Analyses were performed for quantitative variables using the independent sample *t*-test for parametric data between the two groups and the Mann-Whitney *U*-test for nonparametric data between the two groups

The χ^2 -test was used for qualitative data between groups when the cell contains more than 5 and the Fisher exact test when the cell contains less than 5.

Correlation between two variables determined using Pearson's correlation coefficient was as follows: weak ($r = 0-0.24$), fair ($r = 0.25-0.49$), moderate ($r = 0.5-0.74$), and strong ($r = 0.75-1$)

Simple logistic regression analysis was performed for the calculation of the odds ratio.

The level of significance was taken at *P* value 0.05 or less.

Results

Clinical and biochemical parameters of the 40 mechanically ventilated patients with COPD exacerbation and severe acute asthma on admission are illustrated in Table 1. The age of the patients was 63.15 ± 9.09 years. Men represented 40% of all patients

included in this study. In this work, 45% of the patients were smokers. Causes of acute respiratory failure were

Table 1 Demographic and clinical criteria on admission in all studied patients

Variables	Range (mean \pm SD)
Age (years)	45–78 (63.15 \pm 9.09)
Sex [N (%)]	
Male	16 (40)
Female	24 (60)
Smoking [N (%)]	
No	22 (55)
Yes	18 (45)
Cause of acute respiratory failure [N (%)]	
Acute exacerbation of COPD	31 (77.5)
Acute severe asthma	9 (22.5)
MV duration (days)	2–16 (5.67 \pm 4.25)
ICU duration (days)	3–21 (8.05 \pm 5.23)
ABGs (before MV)	
pH	7–7.42 (7.22 \pm 0.07)
PCO ₂ (mmHg)	52.6–149 (98.22 \pm 24.34)
PO ₂ (mmHg)	30.6–87.7 (55.45 \pm 14.58)
HI	61.2–175.4 (109.9 \pm 28.9)
Lung mechanics	
DSF	0.03–0.7 (0.43 \pm 0.15)
Cst (ml/cmH ₂ O)	6.1–42.9 (20.98 \pm 9.5)
Cdyn (ml/cmH ₂ O)	4.5–38.6 (13.85 \pm 7.29)
Raw (cmH ₂ O/l/s)	1.2–31.8 (15.68 \pm 7.82)
ETCO ₂ (mmHg)	13.7–65.3 (39.87 \pm 15.76)

Cdyn, dynamic compliance; COPD, chronic obstructive lung diseases; Cst, static compliance; DSF, dead-space fraction; ETCO₂, end-tidal carbon dioxide; HI, hypoxia index; MV, mechanical ventilation; PCO₂, partial pressure of carbon dioxide.

acute exacerbation of COPD in 77.5% of the patients and acute severe asthma in 22.5% of the patients (Table 1).

About 60% of all the patients ($n = 24$) were weaned successfully and survived. They were with a significantly lower age (60.71 \pm 8.95), had a heart rate less than 120, a lower MV duration (3.75 \pm 1.8 days), and a higher pH more than 7.32. They showed significant improvement in Cdyn (22.47 \pm 8.21) and had a lower Raw (10.99 \pm 18.7). Otherwise, there were no significant differences with regard to ETCO₂, DSF, and static compliance (Table 2).

A significant correlation was observed between the final ETCO₂ and Cdyn. However, no correlation was found between the final ETCO₂ and other physiological parameters (Table 3 and Fig. 1).

In Table 4, the logistic regression analysis showed a significant association between failure of weaning and each of age, MV duration, heart rate, pH, rapid shallow breathing index, and Cdyn.

The COPD patients who survived had a significantly higher Raw and lower compliance in comparison with the asthmatic group (Table 5).

Discussion

There are no clearly defined criteria for the start of invasive MV in COPD and asthma. In COPD, the

Table 2 Final outcome measures in both the successful and the failed weaning groups

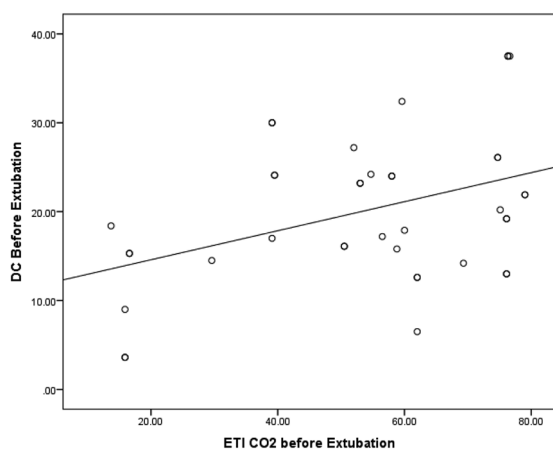
Variables	Successful weaning ($n = 24$)	Failed weaning ($n = 16$)	P value
Age (mean \pm SD) (years)	60.71 \pm 8.95	66.81 \pm 8.26	0.036*
Sex [N (%)]			
Males	9 (37.5)	7 (43.8)	0.693
Females	15 (62.5)	9 (56.2)	
Smoking	12 (50)	6 (37.5)	0.436
Cause of ARF [N (%)]			
COPD	17 (70.8)	14 (87.5)	0.216
Severe asthma	7 (29.2)	2 (12.5)	
MV duration (mean \pm SD) (days)	3.75 \pm 1.8	8.56 \pm 5.23	0.003*
Weaning success parameters			
Heart rate <120 [N (%)]	24 (100)	8 (50)	<0.001*
Respiratory rate <35 [N (%)]	23 (95.8)	13 (81.2)	0.132
pH >7.32 [N (%)]	23 (95.8)	9 (56.2)	0.002*
Hypoxic index >150 [N (%)]	24 (100)	14 (87.5)	0.076
RSBI (mean \pm SD)	58.21 \pm 28.8	120.43 \pm 44.01	<0.001*
Lung mechanics (mean \pm SD)			
DSF	0.31 \pm 0.14	0.33 \pm 0.21	0.74
ETCO ₂ (mmHg)	53.39 \pm 19.49	54.69 \pm 23.51	0.850
Cst (ml/cmH ₂ O)	31.03 \pm 11.78	25.63 \pm 13.41	0.151
Cdyn (ml/cmH ₂ O)	22.47 \pm 8.21	16.62 \pm 7.57	0.041*
Raw (cmH ₂ O/l/s)	10.99 \pm 18.7	11.17 \pm 6.01	0.036*

Cdyn, dynamic compliance; COPD, chronic obstructive lung diseases; Cst, static compliance; DSF, dead-space fraction; ETCO₂, end-tidal carbon dioxide; RSBI, rapid shallow breathing index; * $P < 0.05$ = significant.

most widely adopted approach is patient intubation if noninvasive MV fails after 1 h (clinical and/or worsening of blood gas criteria) [5]. In asthma, with early and aggressive management, the majority of the asthma attacks can be managed without the need for intubation and ventilation. In our study, a low percentage of the patients (22.5%) were asthmatic. Progressive exhaustion, respiratory arrest, a decreased level of consciousness, persistent respiratory acidosis (pH < 7.2), and unremitting hypoxemia (O_2 saturation < 90) are clear indications for intubation [19,20]. Respiratory acidosis is the best predictor of patient exhaustion [5].

In the present study, arterial blood gas analysis on admission revealed a marked respiratory acidosis and hypoventilation (pH 7.22 ± 0.07 , $PaCO_2$ 98.22 ± 24.34), in addition to poor oxygenation as (PaO_2 55.45 ± 14.58) the PaO_2/FiO_2 ratio less than 150 (109.9 ± 28.9) (Table 1). For COPD exacerbation, a Glasgow coma score of less than 11, respiratory rate more than 30, and pH less than 7.25 at admission were associated with a 70% risk of intubation [21]. However, the pH value should not be used as the only deciding

Fig. 1



Correlation between the final end-tidal carbon dioxide ($ETCO_2$) and dynamic compliance (Cdyn).

Table 3 Correlation between the final $ETCO_2$ and the DSF and the PH, the $PaCO_2$, the Raw, the Cst, and the Cdyn

Variables	Final $ETCO_2$		Final DSF	
	R	P value	R	P value
pH	0.262	0.102	-0.281	0.079
CO_2	-0.049	0.763	0.091	0.576
Raw (cmH ₂ O/l/s)	0.018	0.910	-0.224	0.165
Cst (ml/cmH ₂ O)	0.333	0.035*	-0.289	0.071
Cdyn (ml/cmH ₂ O)	0.408	0.009*	-0.139	0.393
MV time	0.016	0.921	0.085	0.604

Cdyn, dynamic compliance; Cst, static compliance; DSF, dead-space fraction; $ETCO_2$, end-tidal carbon dioxide; MV, mechanical ventilation; $PaCO_2$, arterial partial pressure of carbon dioxide; * $P < 0.05$ = significant.

Table 4 The logistic regression analysis, taking failed extubation as the dependent variable and examining the different variables that showed at least a tendency towards statistical significance in the univariate analysis

Variables	Odds ratio	95% CI	P value
Age (mean \pm SD) (years)	1.09	1.002–1.179	0.045*
Sex			
Male	1.296	0.36–4.69	0.693
Female	0.77	0.21–2.79	0.693
Smoking	0.6	0.16–2.18	0.438
Cause of ARF			
Acute exacerbation of COPD	2.88	0.51–16.15	0.229
Acute severe asthma	0.35	0.06–1.94	0.229
MV duration (days)	1.51	1.1–2.08	0.011*
Weaning success parameters			
Heart rate <120	0.02	0.001–0.39	0.001*
Respiratory rate <35	0.19	0.02–2	0.166
pH >7.32	0.06	0.006–0.52	0.011*
Hypoxic index >150	0.12	0.005–2.64	0.178
Lung mechanics (final) (mean \pm SD)			
DSF	2.08	0.05–85.78	0.7
DSF >0.6	2.54	0.37–17.25	0.341
Cst (ml/cmH ₂ O)	0.96	0.91–1.02	0.188
Cdyn (ml/cmH ₂ O)	0.91	0.82–0.99	0.04*
Raw (cmH ₂ O/l/s)	1.001	0.96–1.045	0.97
$ETCO_2$ (mmHg)	1.003	0.97–1.03	0.845
RSBI	1.04	1.02–1.06	0.001*

Cdyn, dynamic compliance; COPD, chronic obstructive lung diseases; Cst, static compliance; DSF, dead-space fraction; $ETCO_2$, end-tidal carbon dioxide; MV, mechanical ventilation; RSBI, rapid shallow breathing index; * $P < 0.05$ = significant.

Table 5 Changes in lung mechanics before extubation in the survivors of both the COPD and the acute severe asthma groups of patients

Variables	Acute severe asthma (N = 7)	COPD (N = 17)	P value
DSF	0.4 \pm 0.08	0.43 \pm 0.14	0.506
Raw (cmH ₂ O/l/s)	4.2 \pm 0.91	13.78 \pm 21.77	0.004*
Cst (ml/cmH ₂ O)	38.78 \pm 14.43	27.82 \pm 9.18	0.035*
Cdyn (ml/cmH ₂ O)	28.02 \pm 9.84	20.18 \pm 6.45	0.030*
$ETCO_2$	63.9 \pm 20.3	49.06 \pm 17.95	0.090

Cdyn, dynamic compliance; COPD, chronic obstructive lung diseases; Cst, static compliance; DSF, dead-space fraction; $ETCO_2$, end-tidal carbon dioxide; * $P < 0.05$ = significant.

factor, but rather should be used in conjunction with other factors such as the patient's mental status, comorbid conditions, patient's code status, etc. [7]. The major physiologic defects in COPD are an increased dead space, severe ventilation-perfusion misdistributions, marked airflow limitation, air trapping, and hyperinflation. Such defects frequently result in poor oxygenation and hypercapnia [22].

In our study, the successfully weaned patients represent 60% ($n = 24$) of all patients, and 40% ($n = 16$) of all

patients failed weaning and died. Similar findings were reported in the study and 31% ($n = 4$) failed weaning trials and finally died.

Our findings revealed that the mortality rate was lower among asthmatic patients (22.5%), whereas it reached 45.1% among the COPD patients. Williams *et al.* [23] has reviewed 28 publications on ventilation in asthma and found a range of mortalities from 0 to 38% (mean 13%). Mortality and morbidity figures seem to be decreasing in recent years with the avocation of controlled hypoventilation [19,20].

Patients with COPD who require MV generally have a greater dependence on ventilators than those with asthma. In addition to an excessive workload and the weakened pressure-generating capacity of the inspiratory muscles [24,25], tracheal obstruction may be an important factor prolonging ventilator dependence [26]. Data from weaning units describe another picture: 42% of the patients with COPD in a weaning unit became partially or completely ventilator dependent and 23% of them died there [25].

In the present study, it was found that logistic regression analysis showed a significant association between weaning failure and an older age. In the study of Ali *et al.* [27], the principal findings are that

- (a) Both COPD-related hypercapnic respiratory failure and an inability to clear secretions were the most common causes of extubation failure in the elderly,
- (b) The presence of underlying pulmonary disease, the need for MV for more than 4 days, and severe hypoalbuminemia are associated with an increased risk of extubation failure in the elderly, and
- (c) Extubation failure carries a higher risk of morbidity and mortality.

Some patients can be weaned successfully from MV within hours, and for others, it may take longer, possibly days or weeks. The MV duration (lower mean \pm SD in days) observed in the success group was 3.75 ± 1.8 compared with 8.56 ± 5.23 in the failure group of the current study. Logistic regression analysis also showed a significant association between weaning failure and prolonged MV duration (days) (odds ratio 1.51, $P = 0.011$) (Table 4). A prolonged MV, especially in COPD exacerbation patients, yields a poor chance for weaning [28,29].

The present study revealed that a heart rate less than 120, a rapid shallow breathing index (<105), and pH more than 7.32 were the most important predictors of successful weaning (Tables 2 and 4)

Physiologic parameters such as minute ventilation (<15 l), respiratory rate (<30), Vt (>325 ml), and maximum inspiratory pressure (<-15) have some utility in predicting the patient's ability to sustain spontaneous ventilation [30–32].

The PaO₂/inspired fraction of oxygen (PaO₂/FiO₂) ratio is still the most frequently used variable for evaluating the severity of lung failure and is included in the current definition of acute lung injury/acute respiratory distress syndrome [33]. From this PaO₂/FiO₂ ratio, the higher the FiO₂, the poorer the prognosis [34]. Despite its limitations, this ratio remains the most commonly used means of assessing the severity of lung disease. Despite this, our findings showed that the hypoxia index is not a dependable parameter for weaning as some of the failed weaning patients had PaO₂/FiO₂ more than 150. The oxygen index [(mean airway pressure \times FiO₂ \times 100)/PaO₂] accounts better for the influence of ventilator pressures on the oxygenation value [6,35].

Assessment of respiratory mechanics

Because of the lack of physiological monitoring of the respiratory muscle function, the ventilator is set essentially by common practice and the effects of MV on the inspiratory effort and work of breathing are not directly measured [36].

In our study, the survivors had a higher dynamic and static compliance (22.47 ± 8.21 and 31.03 ± 11.78 , respectively); also, they had a lower Raw (10.99 ± 18.7) than patients who failed weaning (Cdyn, 16.62 ± 7.57 ; Raw, 11.17 ± 6.01). Logistic regression analysis also showed that Cdyn is the most important one of the predictable parameters of weaning; this in addition to the significant correlation between Cdyn and ETCO₂ (Table 4 and Fig. 1).

Consistent with our results, many studies [30–32] reported that Cdyn more than 22 and static compliance more than 33 were associated with successful weaning. Depending on the tracheal tube size, the resistance may be as great as $10 \text{ cmH}_2\text{O} \pm 1 \pm 1$ s, whereas compliance may be as small as $0.06 \pm 1.20 \text{ cmH}_2\text{O}$ [10].

Our findings confirmed that COPD patients who survived had a significantly higher resistance and both lower static and Cdyn than asthmatic patients who survived (Table 5). Patients with COPD have an increased fixed expiratory airflow resistance. Alveolar attachments that normally keep the smaller airways open through radial traction are lost. This leads to airway narrowing and collapse. The greater airway collapse may also be due fundamentally to the destruction of the lung parenchyma (particularly in emphysema) as well as due to a loss of elastic rebound of the lungs [7].

In this study, the aim was to calculate the DSF and to demonstrate whether this parameter can be useful in determining the prognosis. Logistic regression analysis showed no significant association between the DSF and extubation failure, with odds ratio equal to 2.08 (95% confidence interval: 0.05–85.78, $P = 0.7$). We also found no significant correlation between DSF and pH, PaCO₂, and lung mechanics parameters. These findings do not contribute to the evaluation of the patient's condition, nor do they enable us to predict the length of artificial ventilation necessary for patients with acute respiratory failure due to chronic air flow obstruction.

Few studies have demonstrated the usefulness of simple biochemical and clinical markers that need only some calculations of the PETCO₂ concentration and DSF using a simple equation such as the Bohr used in our study [15,16].

In line with our study, Farah and Makhoul [22] concluded that the evaluation of DSF does not provide a factor in estimating the length of treatment for patients with acute respiratory failure due to COPD exacerbation. No other studies succeeded in utilizing the measurement of DSF in COPD exacerbation patients as a predictor for weaning from MV information [22]. Bouso *et al.* [37] reported that in a pediatric population receiving MV due to a variety of etiologies, the VD/Vt ratio was unable to predict the populations at risk of extubation failure or of reintubation.

In contrast to our findings, many studies demonstrated the utility of DSF measurement at the time of diagnosis in patients with acute respiratory distress syndrome and in patients with pulmonary embolism [15,38]. González-Castro *et al.* [39] found that logistic regression analysis showed a significant association between the VD/Vt and extubation failure, with odds ratio equal to 1.52 (95% confidence interval: 1.11–2.09, $P = 0.008$). More research is necessary to explore the reasons for differing patient management and outcomes.

In contrast, the appropriate use of ETCO₂ monitors as a noninvasive direct method of assessing PaCO₂ in ventilated patients remains unclear [22]. Through our results, clearly, logistic regression analysis showed no significant association between weaning failure and ETCO₂ (Table 4), and no correlation between arterial PaCO₂ and ETCO₂ (Table 3). However, Adel *et al.* [40] found a significant correlation between ETCO₂ and arterial PCO₂ throughout the period of MV in CB and emphysematous patients; hence, monitoring ETCO₂ provided a good noninvasive assessment of hypercapnic episodes during weaning

from MV. Belpomme *et al.* [41] noticed that there was a wide variation in the gradient between PaCO₂ and ETCO₂ depending on the patient's condition, and this relationship does not remain constant over time. Many studies [28,41,42] concluded that ETCO₂ is a less accurate measure of PaCO₂ with Vt breathing and in patients with pulmonary disease.

Conclusion

Mechanical ventilatory support plays a crucial role in the management of severe airflow obstruction, especially when patients confront life-threatening respiratory failure.

PaCO₂-ETCO₂/PaCO₂ may be a useful serial measurement in the critically ill patient because all the necessary data are easily obtained and calculation is significantly simpler than for VD alv/Vt alv (Bohr–Fowler).

Weaning failure is commonly multifactorial in origin, and thus an index that assesses a single physiologic function may not be optimal. Indeed, this was shown by the data in the present and previous studies, which showed that indexes assessing the major determinants of the weaning outcome had limited diagnostic accuracy when used individually.

Also, it was concluded that DSF was unable to predict the populations at risk of extubation failure in patients with obstructive airflow diseases who were mechanically ventilated.

We limited our study population to medical patients, as it is generally more difficult to predict the weaning outcome in these patients than in surgical patients. Second, the technique of making the physiologic measurements needs to be stated clearly. More research is necessary to explore the reasons for differing patient management and outcomes.

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Conflicts of interest

There are no conflicts of interest.

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