CO_2 Response and Duration of Weaning From Mechanical Ventilation

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BACKGROUND: The CO_2 response test measures the hypercapnic drive response (which is defined as the ratio of the change in airway-occlusion pressure 0.1 s after the start of inspiratory flow [$\Delta P_{0.1}$] to the change in P_{aCO_2} [ΔP_{aCO_2}]), and the hypercapnic ventilatory response (which is defined as the ratio of the change in minute volume to ΔP_{aCO_2}). OBJECTIVE: In mechanically ventilated patients ready for a spontaneous breathing trial, to investigate the relationship between CO_2 response and the duration of weaning. METHODS: We conducted the CO_2 response test and measured maximum inspiratory pressure (P_{Imax}) and maximum expiratory pressure (P_{Emax}) in 102 non-consecutive ventilated patients. We categorized the patients as either prolonged weaning (weaning duration > 7 d) or non-prolonged weaning (≤ 7 d). RESULTS: Twenty-seven patients had prolonged weaning. Between the prolonged and non-prolonged weaning groups we found differences in hypercapnic drive response (0.22 ± 0.16 cm H_2O/mm Hg vs 0.47 ± 0.22 cm H_2O/mm Hg, respectively, $P < .001$) and hypercapnic ventilatory response (0.25 ± 0.23 L/min/mm Hg vs 0.53 ± 0.33 L/min/mm Hg, respectively, $P < .001$). The optimal cutoff points to differentiate between prolonged and non-prolonged weaning were 0.19 cm H_2O/mm Hg for hypercapnic drive response, and 0.15 L/min/mm Hg for hypercapnic ventilatory response. Assessed with the Cox proportional hazards model, both hypercapnic drive response and hypercapnic ventilatory response were independent variables associated with the duration of weaning. The hazard ratio of weaning success was 16.7 times higher if hypercapnic drive response was > 0.19 cm H_2O/mm Hg, and 6.3 times higher if hypercapnic ventilatory response was > 0.15 L/min/mm Hg. Other variables ($P_{0.1}$, P_{Imax}, and P_{Emax}) were not associated with the duration of the weaning. CONCLUSIONS: Decreased CO_2 response, as measured by hypercapnic drive response and hypercapnic ventilatory response, are associated with prolonged weaning. *Key words: hypercapnia; mechanical ventilation; respiratory center; respiratory function test; ventilator weaning; critically ill.* [Respir Care 2011;56(8):1130–1136. © 2011 Daedalus Enterprises]

Introduction

Critically ill patients ready for weaning from mechanical ventilation but who fail a spontaneous breathing trial (SBT) have higher airway-occlusion pressure 0.1 s after the start of inspiratory flow ($P_{0.1}$)[1-5] and lower carbon dioxide (CO_2) response[6-9] than successfully weaned patients. However, those studies did not analyze the relationship between the CO_2 response and the duration of weaning. In those studies, CO_2 response was measured with the rebreathing method and evaluated based on the hypercapnic drive response (the change in $P_{0.1}$ induced by an increase in P_{aCO_2}), and the hypercapnic ventilatory response (the change in minute volume [$\dot{V}_E$] induced by an increase in P_{aCO_2}).

Chemosensitivity measured with the CO_2 response test provides an indication of the "integrity" of the respiratory system.[10] Any alteration of the respiratory system (metabolic control, neuromuscular, or ventilatory apparatus) can reduce the hypercapnic ventilatory response,[10] whereas reduced hypercapnic drive response indicates an alteration of the respiratory center, the neural transmission, or both,

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as suggested by Holle et al,[11] who found that progressive curarization reduced hypercapnic ventilatory response but did not reduce $P_{0.1}$ or hypercapnic drive response, even in individuals with severe respiratory muscle weakness.

A reduced CO_2 response may be a reason for prolonged weaning in some critically ill patients. In the new classification system from a recent international consensus conference, prolonged weaning is defined as the need for more than 3 SBTs or weaning that requires more than 7 days to successful extubation.[12] In critically ill mechanically ventilated patients ready for an SBT, we investigated the relationship between CO_2 response and the duration of weaning.

Methods

The study was conducted from May 2003 to October 2008, and was approved by the review boards of both hospitals. Informed consent was obtained from all the subjects or their closest relatives.

Patients

In 2 medical-surgical intensive care units (ICUs) we studied 102 non-consecutive patients recovering from the acute phase of critical illness after $\geq$ 5 days of mechanical ventilation. All subjects underwent a daily screening by the physician in charge. The routine clinical criteria for considering an SBT were that the patient was hemodynamically stable, without sedation, awake, able to obey oral commands, core temperature $<$ 38.3°C, improvement or resolution of the condition that required mechanical ventilation, $P_{aO_2}/F_{IO_2} > 150$ mm Hg, and PEEP $\leq$ 8 cm H_2O.[12] Patients were excluded if they previously presented any neurological disorder, had undergone tracheostomy before the first SBT, or had an acute respiratory failure due to an exacerbation of previously diagnosed COPD.

Protocol

When the patient was ready for an SBT, respiratory neuromuscular function was evaluated via measurement of maximum inspiratory pressure (P_{Imax}), maximum expiratory pressure (P_{Emax}), $P_{0.1}$, and CO_2 response test. All these measurements were carried out in the semirecumbent position, immediately following endotracheal suctioning. We continuously recorded electrocardiogram, heart rate, pulse oximetry, and invasive systemic blood pressure.

The physician in charge, who was unaware of the respiratory neuromuscular function results, conducted an SBT (with T-piece connected to the endotracheal tube) 15–30 min after the respiratory neuromuscular measurements. The T-piece SBT was for 2 hours, with the same F_{IO_2} used during mechanical ventilation. Patients who tolerated the 2-hour SBT without signs of distress were extubated. Signs of distress were defined as respiratory rate $>$ 35 breaths/min, arterial oxygen saturation $<$ 90%, heart rate $>$ 140 beats/min, systolic blood pressure $>$ 180 mm Hg or $<$ 90 mm Hg, agitation, diaphoresis, or anxiety. If signs of distress developed, we restarted mechanical ventilation in an assist control or pressure support mode for 24 hours, then conducted another SBT. The decision to extubate or to restart mechanical ventilation was made by the physician in charge, according to the above-mentioned criteria.

Maximum Inspiratory and Expiratory Pressure

P_{Imax} and P_{Emax} were measured, after 1–2 min of spontaneous breathing, with an external pressure transducer, via a unidirectional valve (Hans Rudolph, Shawnee, Kansas) connected to the endotracheal tube. P_{Imax} was obtained at residual volume by occluding the inspiratory port of the unidirectional valve. P_{Emax} was measured at total lung volume by occluding the expiratory port.[13] After 20–25 seconds of occluded inspiration or expiration, we recorded the most negative and positive pressures. Two maneuvers were performed and the highest value was used for analysis.

Airway-Occlusion Pressure 0.1 Second After the Start of Inspiratory Flow

$P_{0.1}$ was measured by means of the built-in system in the ventilator (Evita 2 Dura or Evita 4, Dräger, Lübeck, Germany).[14,15] $P_{0.1}$ was calculated as the mean of 5 measurements at each point of the study.[5]

CO_2 Response Test

We chose as the CO_2 response test the method of reinhalation of expired air,[8,16,17] which involves inserting a length of corrugated tubing (inner diameter 22 mm) (Corr-A-Flex II, Hudson RCI, Temecula, California) between the Y-piece and the endotracheal tube, which increases the dead space with a volume similar to the tidal volume obtained with a pressure support of 7 cm H_2O. The mean dead space we created was 414 $\pm$ 71 mL.

We obtained baseline values for CO_2 response test during 5 min of pressure support ventilation with a pressure of 7 cm H_2O, 0 PEEP, and F_{IO_2} of 1.0 to prevent hypoxemia and hypoxic stimuli. Then we recorded respiratory rate, $P_{0.1}$, and $\dot{V}_E$ from the ventilator, and took an arterial blood sample. We initiated the CO_2 response test by increasing the dead space while maintaining the same ventilatory support. When the exhaled CO_2 (measured via capnography) had increased by approximately 10 mm Hg, we again measured respiratory rate, $P_{0.1}$, and $\dot{V}_E$, and took another arterial blood sample. After the CO_2 response test the added dead space was removed and the patient was returned to

his or her original assisted ventilation mode. All blood samples were analyzed with a blood gas analyzer (IL-1650, Instrument Laboratory, Izasa, Spain).

We calculated the changes in $\dot{V}_E$, $P_{0.1}$, and P_{aCO_2} as the difference between the baseline value and the value at the end of the CO_2 response test. We calculated the hypercapnic drive response as the change in $P_{0.1}$ divided by the change in P_{aCO_2} ($\Delta P_{0.1}/\Delta P_{aCO_2}$), and the hypercapnic ventilatory response as $\Delta \dot{V}_E/\Delta P_{aCO_2}$.

Other Variables

We also took into account clinical variables that might be associated with the duration of weaning, including age, Simplified Acute Physiology Score (SAPS) II, and duration of mechanical ventilation before the first SBT.

Definitions

The duration of mechanical ventilation before the first SBT was defined as the number of days between the beginning of mechanical ventilation and the patient's first SBT. Duration of weaning was defined as the time elapsed between the first SBT and the day of successful weaning. Weaning was considered successful if SBT was successful, tracheal extubation was achieved, and re-intubation was not required within 48 hours after extubation; or, in patients with later tracheostomy, withdrawal of mechanical ventilation was achieved and mechanical ventilation was not required within 48 hours.[12] We considered it weaning failure if a patient had any signs of distress during the SBT or re-intubation was required within 48 hours after extubation; or, in patients with later tracheostomy, mechanical ventilation was required within 48 hours after withdrawal of mechanical ventilation.[12] We categorized weaning as prolonged if weaning time was more than 7 days.[12] Patients were followed-up until discharged from hospital.

Statistical Analysis

Categorical data are expressed as number and percentages. Continuous variables are expressed as mean ± SD or as median and IQR. Differences between the non-prolonged and prolonged weaning groups were compared with the paired *t* test and the chi-square test. We used receiver operating characteristic curve analysis to assess the ability of hypercapnic drive response and hypercapnic ventilatory response to discriminate between patients with prolonged weaning and non-prolonged weaning.[12] The threshold value for the optimal cutoff point was selected according to the minimum sum of false-positive and false-negative test results.[18] A result was deemed true-positive when the test predicted prolonged weaning and prolonged weaning actually occurred; false-positive when the test predicted prolonged weaning but non-prolonged weaning occurred; true-negative result occurred when the test predicted non-prolonged weaning and the non-prolonged weaning actually occurred; and false-negative when the test predicted non-prolonged weaning but prolonged weaning occurred. We also analyzed the sensitivity and specificity of the hypercapnic drive response and hypercapnic ventilatory response for predicting prolonged weaning. We used the Kaplan-Meier method and the log-rank test to estimate the duration of weaning for threshold values of hypercapnic drive response and hypercapnic ventilatory response values. We used a multivariate Cox proportional hazards model to identify independent variables that influenced the duration of weaning. Patients who died during weaning were omitted from the Kaplan-Meier and multivariate analyses. The analyses were performed with statistics software (SPSS 15.0, SPSS, Chicago, Illinois).

Results

We studied 102 patients (59 men). The cohort's mean ± SD age was 62 ± 14 years. Twenty-seven patients (26%) had prolonged weaning and 75 patients (74%) had non-prolonged weaning. Between the prolonged and non-prolonged weaning groups there were no differences in age, sex, body mass index, or severity of illness (Table 1). Patients with prolonged weaning had a higher incidence of COPD, pneumonia, and tracheostomy, longer duration of ventilation before SBT, longer ICU stay and hospital stay, and higher ICU mortality than patients with non-prolonged weaning (see Table 1). The main ICU admission diagnoses of the 5 patients with COPD were pneumonia (3 patients), pancreatic abscess (1 patient), and postoperative state of thoracic aortic aneurysm (1 patient).

Patients with prolonged weaning had lower baseline pH, P_{Emax}, P_{Imax}, hypercapnic drive response, and hypercapnic ventilatory response, and higher baseline respiratory rate and $P_{0.1}$ than patients with non-prolonged weaning (Table 2). Baseline $\dot{V}_E$, P_{aCO_2}, P_{aO_2}/F_{IO_2}, and plasma bicarbonate were not different between the groups.

For hypercapnic drive response the area under the receiver operating characteristic curve to discriminate between prolonged and non-prolonged weaning was 0.84 ± 0.05 (95% CI 0.75–0.90), and for hypercapnic ventilatory response it was 0.77 ± 0.05 (95% CI 0.68–0.85). For hypercapnic drive response the optimal cutoff point (ie, with the fewest false classifications) was 0.19 cm H_2O/mm Hg, and for hypercapnic ventilatory response it was 0.15 L/min/mm Hg (Fig. 1). Sixteen of the 19 patients who had hypercapnic drive response of ≤ 0.19 cm H_2O/mm Hg, and 13 of 16 patients who had hypercapnic ventilatory response of ≤ 0.15 L/min/mm Hg, had prolonged weaning. All 12 patients who had both hypercapnic drive response and hypercapnic ventilatory response

Table 1. Subjects (n = 102)

	Prolonged Weaning (no. = 27)	Non-prolonged Weaning (no. = 75)	P
Age (y)	63 ± 14	62 ± 14	.78
Female, no. (%)	12 (44)	31 (41)	.78
Body mass index (kg/m^2)	29 ± 6	28 ± 6	.20
SAPS II score at ICU admission	42 ± 12	42 ± 13	.86
COPD, no. (%)	4 (15)	1 (1)	.02
Main Diagnosis, no. (%)			.11
Pneumonia	15 (56)	27 (36)	
Non-pulmonary sepsis	5 (18)	29 (39)	
Postoperative state	6 (22)	11 (15)	
Cardiac failure	1 (4)	2 (3)	
Thoracic trauma	0	2 (3)	
Other*	0	4 (5)	
Catecholamines used, no. (%)	25 (93)	60 (80)	.23
Ventilation duration before SBT, median (IQR), d	15 (9–16)	10 (6–16)	.58
Weaning duration, median (IQR), d	16 (11–38)	2 (0–8)	< .001
Tracheotomy after SBT, no. (%)	17 (63)	3 (4)	< .001
Re-intubation within 48 h, no. (%)	10 (37)	5 (7)	< .001
ICU stay, median (IQR), d	45 (28–70)	21 (13–36)	< .001
Hospital stay, median (IQR), d	69 (35–106)	41 (29–70)	.002
ICU mortality, no. (%)	8 (30)	4 (5)	.002
Hospital mortality, no. (%)	11 (41)	18 (24)	.10

± values are mean ± SD.
* Acute asthma, pulmonary hemorrhage, atelectasis, obesity-hypoventilation syndrome.
SAPS = Simplified Acute Physiology Score
ICU = intensive care unit
SBT = spontaneous breathing trial

Table 2. Baseline Ventilation and Blood Gas Values on the Day of the First Spontaneous Breathing Trial

	Prolonged Weaning (no. = 27) (mean ± SD)	Non-prolonged Weaning (no. = 75) (mean ± SD)	P
Respiratory rate (breaths/min)	31 ± 7	26 ± 7	.02
Minute volume (L/min)	10.5 ± 2.5	11.0 ± 3.2	.46
pH	7.38 ± 0.06	7.43 ± 0.05	< .001
P_{aCO_2} (mm Hg)	43 ± 9	40 ± 8	.06
P_{aO_2}/F_{IO_2} (mm Hg)	405 ± 103	436 ± 102	.18
CO_3H^- (mmol/L)	25.8 ± 3.4	26.7 ± 2.6	.16
P_{Emax} (cm H_2O)	22 ± 14	28 ± 14	.08
P_{Imax} (cm H_2O)	39 ± 15	49 ± 20	.02
$P_{0.1}$ (cm H_2O)	3.9 ± 2.0	3.0 ± 1.9	.04
$\Delta P_{0.1}/\Delta P_{aCO_2}$ (cm H_2O/mm Hg)	0.22 ± 0.16	0.47 ± 0.22	< .001
$\Delta \dot{V}_E/\Delta P_{aCO_2}$ (L/min/mm Hg)	0.25 ± 0.23	0.53 ± 0.33	< .001

P_{Emax} = maximum expiratory pressure
P_{Imax} = maximum inspiratory pressure
$P_{0.1}$ = airway-occlusion pressure 0.1 s after the start of inspiratory flow
$\Delta P_{0.1}/\Delta P_{aCO_2}$ = hypercapnic drive response
$\Delta \dot{V}_E/\Delta P_{aCO_2}$ = hypercapnic ventilatory response

values below the thresholds had prolonged weaning (Fig. 2). The accuracies of the threshold values of hypercapnic drive response and hypercapnic ventilatory response to predict prolonged weaning were similar (Table 3).

The Kaplan-Meier analysis and log-rank test (Fig. 3) indicated that the duration of weaning was significantly longer in patients with hypercapnic drive response $\leq$ 0.19 cm H_2O/mm Hg ($P < .001$) and hypercapnic ven-

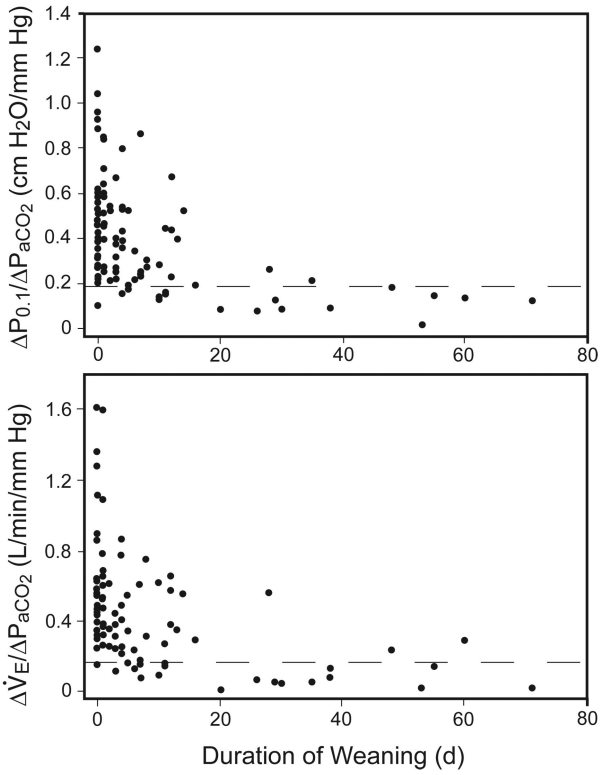

Fig. 1. Hypercapnic drive response (ratio of change in airway-occlusion pressure 0.1 s after the start of inspiratory flow [$\Delta P_{0.1}$] to change in P_{aCO_2} [ΔP_{aCO_2}]) and hypercapnic ventilatory response (ratio of change in minute volume [$\dot{V}_E$] to ΔP_{aCO_2}) versus duration of weaning. The dashed lines represent the optimal cutoff points for hypercapnic drive response (0.19 cm H_2O/mm Hg) and hypercapnic ventilatory response (0.15 L/min/mm Hg).

tilatory response ≤ 0.15 L/min/mm Hg ($P < .001$) than in the other patients.

The Cox proportional hazards model found that hypercapnic drive response and hypercapnic ventilatory response were independent variables associated with the duration of weaning. The hazard ratio for freedom from mechanical ventilation was 16.7 times higher if hypercapnic drive response was > 0.19 cm H_2O/mm Hg, and 6.3 times higher if hypercapnic ventilatory response was > 0.15 L/min/mm Hg (Table 4). There was no interaction between the variables. Other variables entered in the multiple logistic-regression model, including duration of mechanical ventilation before the first SBT, baseline respiratory rate, baseline P_{aCO_2}, baseline pH, baseline $P_{0.1}$, P_{Imax}, and P_{Emax}, were not significantly related to the duration of weaning.

Discussion

Our results indicate that a reduced hypercapnic drive response and a reduced hypercapnic ventilatory response are associated with prolonged weaning. Contrarily, baseline respiratory rate, P_{Imax}, P_{Emax}, baseline $P_{0.1}$, and the duration of mechanical ventilation before the first SBT were not significantly associated with the duration of weaning in the multivariate analysis. The interest of these findings is that, in some patients, dysfunction of the neurorespiratory system may be a pathophysiologic mechanism of prolonged weaning, suggesting a failure of the respiratory system to overcome superimposed respiratory loads.

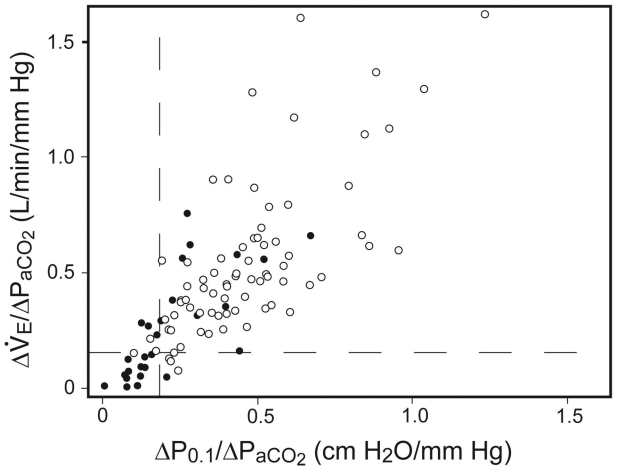

Fig. 2. Hypercapnic drive response and hypercapnic ventilatory response (defined as in Fig. 1) in 102 ventilated patients. The black dots represent patients who had prolonged weaning. The circles represent patients who had non-prolonged weaning. The dotted lines represent the optimal cutoff points for hypercapnic drive response (0.19 cm H_2O/mm Hg) and hypercapnic ventilatory response (0.15 L/min/mm Hg).

The reduced hypercapnic drive response in critical ill patients ready for an SBT could be explained by idiopathic genetic factors,[19-21] acquired factors during critically illness, or both. The idiopathic genetic explanation is based on the fact that 10–15% of healthy subjects have a reduced response to hypercapnia.[19,20] These healthy subjects have normal P_{aCO_2} despite having low chemosensitivity, while lung function is normal.[20,21] However, it is unknown whether these idiopathic genetic factors could affect ventilation after an acute process that requires mechanical ventilation.

Acquired factors that could reduce the hypercapnic drive response in the critically ill include a dysfunction of either the respiratory center or the neural transmission (eg, phrenic neuropathy), or both.[10,11] An alteration of the respiratory center may be present despite a high baseline $P_{0.1}$, as these may not be high enough according to the level of chemical stimulus. This could be the result of the respiratory depressant effect of benzodiazepines and opioids administered during mechanical ventilation,[22-24] the metabolic alkalosis induced by treatment with diuretics,[25,26] or other unknown factors, especially in patients with idiopathic reduced CO_2 response. Supporting the potential dysfunction of the respiratory neural transmission in critically ill patients, Maher et al[27] found that half of 40 patients with prolonged weaning had bilateral or unilateral phrenic neu-

Table 3. Sensitivity, Specificity, and Positive and Negative Predictive Value of Hypercapnic Drive Response and Hypercapnic Ventilatory Response for Predicting Prolonged Weaning

	Sensitivity Median (IQR)	Specificity Median (IQR)	Positive Predictive Value	Negative Predictive Value
$\Delta P_{0.1}/\Delta P_{aCO_2} \leq 0.19$ cm H_2O/mm Hg	0.59 (0.49–0.69)	0.96 (0.90–0.99)	0.84	0.87
$\Delta \dot{V}_E/\Delta P_{aCO_2} \leq 0.15$ L/min/mm Hg	0.48 (0.38–0.58)	0.96 (0.90–0.99)	0.81	0.84

$\Delta P_{0.1}/\Delta P_{aCO_2}$ = hypercapnic drive response
$\Delta \dot{V}_E/\Delta P_{aCO_2}$ = hypercapnic ventilatory response

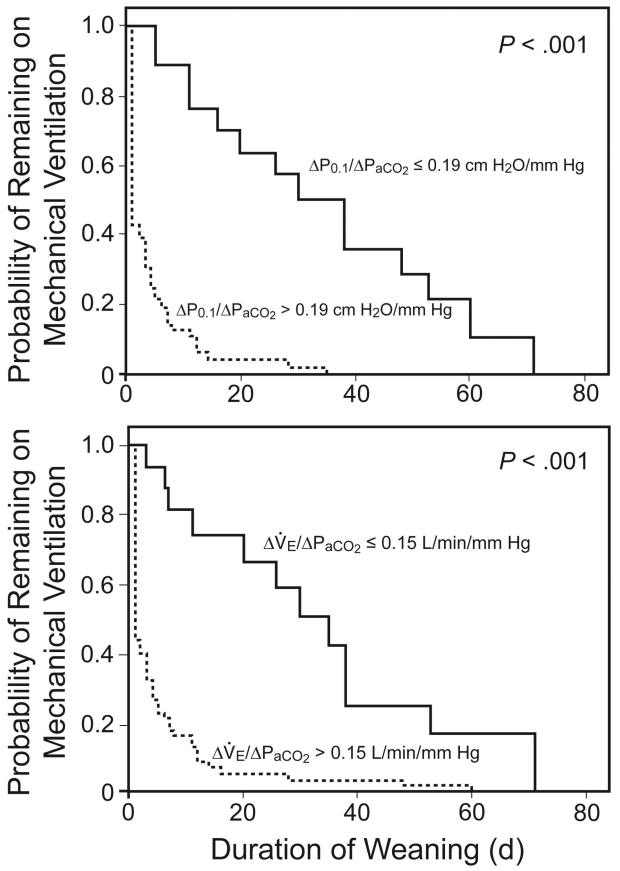

Fig. 3. Kaplan-Meier curves of the probability of remaining on mechanical ventilation after the first spontaneous breathing trial with hypercapnic drive response (defined as in Fig. 1: $\Delta P_{0.1}/\Delta P_{aCO_2}$) and hypercapnic ventilatory response (defined as in Fig. 1: $\Delta \dot{V}_E/\Delta P_{aCO_2}$). The solid line depicts patients whose CO_2 response was below the cutoff point and the dotted line depicts patients whose CO_2 response was above the cutoff point. The *P* values are via long-rank test between groups.

ropathy, evaluated via electrophysiological studies of phrenic nerve conduction and needle electromyography of the chest wall and diaphragm.

Baseline $P_{0.1}$, P_{Imax}, and P_{Emax} were significantly associated with the duration of weaning in the univariate analysis. Similarly, De Jonghe et al[28] found that P_{Imax}, P_{Emax}, and limb muscle strength were independent predictors of duration of weaning in separate multivariate models, each including either P_{Imax}, P_{Emax}, or limb muscle strength, and other variables such as COPD, SAPS II score, female sex, and cardiac failure. The association between limb muscle strength and duration of weaning was previously established.[29,30] In our multivariate analysis, hypercapnic drive response and hypercapnic ventilatory response were stronger predictors than the other variables associated with prolonged weaning, including baseline $P_{0.1}$, P_{Imax}, and P_{Emax}, probably because among all these parameters, only the CO_2 response is affected by the whole respiratory system.[10]

Table 4. Variables Independently Associated With Successful Extubation After the First Spontaneous Breathing Trial*

	Hazard Ratio (95% CI)	*P*
$\Delta P_{0.1}/\Delta P_{aCO_2} > 0.19$ cm H_2O/mm Hg	16.7 (13.7–75)	< .001
$\Delta \dot{V}_E/\Delta P_{aCO_2} > 0.15$ L/min/mm Hg	6.3 (1.2–33)	.03

* Via Cox regression model.
$\Delta P_{0.1}/\Delta P_{aCO_2}$ = hypercapnic drive response
$\Delta \dot{V}_E/\Delta P_{aCO_2}$ = hypercapnic ventilatory response

Despite the moderate accuracy of the CO_2 response test, we think that, when a first SBT has failed, the bedside measurement of CO_2 response can help to identify patients who will need prolonged weaning, mainly when the reason for the failure is not known. Thus, the CO_2 response test can be clinically useful to early identify patients who may benefit most from a particular therapy, such as rehabilitation or tracheostomy. This hypothesis is in consonance with a recent study that found that prolonged weaning, which accounts for 14% of intubated patients, was associated with a tracheostomy rate of 68%,[31] similar to the percentage of tracheostomy in our study.

Limitations

First, we studied non-consecutive patients, which could have caused a patient-selection bias toward patients with septic shock and longer mechanical ventilation time. Thus, our results cannot be extrapolated to general critically ill patients. Second, we measured $P_{0.1}$ with a built-in function

in the ventilator, instead of with the conventional method,[1,4] and the Evita ventilator tends to overestimate high $P_{0.1}$ values and to underestimate low $P_{0.1}$ values.[15] As hypercapnic drive response evaluates the $P_{0.1}$ change induced by a CO_2 increase, not the absolute $P_{0.1}$ value, the systematic error may be reduced. Third, the inter-individual coefficient of variation of $P_{0.1}$ on repeated measurements during CO_2 rebreathing trials is described to be near to 60%.[32] Fourth, we did not perform phrenic or diaphragmatic electromyography, which would be needed to distinguish between a central component and a neural transmission component. However, electromyography is invasive and less available in the ICU environment.

Conclusion

In mechanically ventilated patients ready for an SBT, reduced hypercapnic response is associated with prolonged weaning.

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