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Physiological comparison of three spontaneous breathing trials in difficult-to-wean patients

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Abstract *Purpose:* To compare cardiovascular and respiratory responses to different spontaneous breathing trials (SBT) in difficult-to-wean patients using T-piece and pressure support ventilation (PSV) with or without positive end-expiratory pressure (PEEP). *Methods:* Prospective physiological study. Fourteen patients who were monitored with a Swan-Ganz catheter and had failed a previous T-piece trial were studied. Three SBTs were performed in random order in all patients: PSV with PEEP (PSV-PEEP), PSV without PEEP (PSV-ZEEP), and T-piece. PSV level was 7 cmH_2O, and PEEP was 5 cmH_2O. Inspiratory muscle effort was calculated, and hemodynamic parameters were measured using standard methods. *Results [median (and interquartile range)]:* Most patients succeeded in the PSV-PEEP (11/14) and PSV-ZEEP (8/14) trials, but all failed the T-piece trial. Patient effort was significantly higher during T-piece than during PSV with or without PEEP [esophageal pressure-time product was 292 (238–512), 128 (58–299), and 148 (100–465) $cmH_2O \cdot s/min$, respectively, $p < 0.05$]. Left ventricular heart failure was observed in 11 of the 14 patients during the T-piece trial. Pulmonary artery occlusion pressure and respiratory rate were significantly higher during T-piece than with PSV-PEEP [21 (18–24) mmHg versus 17 (14–22) mmHg, $p < 0.05$ and 27 (21–35) breaths/min versus 19 (16–29) breaths/min, $p < 0.05$ respectively]. Tidal volume was significantly lower during the T-piece trial. *Conclusion:* In this selected population of difficult-to-wean patients, PSV and PSV plus PEEP markedly modified the breathing pattern, inspiratory muscle effort, and cardiovascular response as compared to the T-piece. Caregivers should be aware of these differences in SBT as they may play an important role in weaning decision-making.

Keywords Weaning from mechanical ventilation · Spontaneous breathing trial · Pressure support ventilation · Heart failure

Introduction

Early identification of patients able to progress to spontaneous breathing promotes shorter duration of mechanical ventilation and reduces complications [1]. Once a physician deems that a patient is ready to breathe without ventilatory assistance, a screening test is performed. If this is successful, a spontaneous breathing trial (SBT) of variable duration (usually from 30 to 120 min) is conducted. The SBT is needed to confirm the patient's ability to breathe without assistance [2]. Switching from mechanical to spontaneous ventilation can decrease left

ventricular performance by increasing preload and afterload [3], and unmask latent left ventricular heart failure (LVHF) [4]. In difficult-to-wean patients, T-piece spontaneous breathing often induces an increase in left ventricular filling pressure [5]. Early detection of cardiac origin as a cause of respiratory distress during a weaning trial is relevant since treatment with vasodilators and/or diuretics will usually improve clinical symptomatology and may hasten extubation [4].

The SBT is generally performed by disconnecting the patient from the ventilator and attaching a T-piece to the endotracheal tube. Some authors, however, prefer to use low levels of pressure support ventilation (PSV) with or without positive end-expiratory pressure (PEEP) [6, 7]. It has been reported that the rate of failure may be higher with a T-piece trial than with a PSV trial [8, 9], possibly because respiratory muscle energy expenditure is greater with the former [10–12]. A less demanding trial in terms of respiratory muscle effort, especially when PEEP is added to PSV, may also prevent the development of LVHF and thus blunt the clinical signs and symptoms of intolerance to unassisted T-piece spontaneous breathing.

In difficult-to-wean patients (i.e., those who had failed a first confirmatory T-piece trial), SBTs using a T-piece or low levels of PSV with and without PEEP have not been systematically compared in a physiological study exploring both cardiovascular and respiratory responses. We hypothesized that these three trials (T-piece, PSV with PEEP, and PSV without PEEP) elicit different responses of the cardiovascular and respiratory systems, especially in at risk populations of difficult-to-wean patients. Some of the results of this study have been previously reported in abstract form [13].

Patients and methods

This study was approved by the Ethics Committee of Sant Pau Hospital. All patients and/or their surrogates were informed, and written consent was given prior to inclusion in the study.

The physiological study was performed over a 10-month period in a 16-bed medical-surgical intensive care unit of a university hospital. All consecutive patients requiring mechanical ventilation were prospectively screened and followed until they fulfilled usual weaning criteria.

Our at-risk population consisted of those patients who failed a confirmatory SBT with a T-piece within 60 min of its initiation and already had a Swan-Ganz catheter in place for clinical reasons. After failing the first SBT with a T-piece, patients were reconnected to the ventilator in volume assist-control mode (ACV), and a double balloon esophageal-gastric catheter (see physiological measurements, below) was passed through a nostril after topical anesthesia. After clinical stabilization under ACV (baseline), three SBTs were performed in random order in all participants: T-piece, PSV-PEEP (PSV level of 7 cmH_2O with a PEEP of 5 cmH_2O), and PSV-ZEEP (PSV level of 7 cmH_2O and zero PEEP). A PSV level of 7 cmH_2O was set since it has been used in other studies and has been found to compensate for the impedance of the ventilator and its circuits [8]. Heated humidifiers were used during PSV to avoid an increase in work of breathing induced by the deadspace and resistance of the heat and moisture exchangers [14]. The SBTs were stopped if any signs of poor tolerance developed and were considered a success if no signs of poor tolerance occurred within 60 min of initiation. Between each test, the patient was ventilated in ACV for 20–30 min to achieve a cardio-respiratory status equal to baseline.

At the time of the study, a Doppler-echocardiography machine was not available in our department. Consequently, our patients were frequently monitored using a Swan-Ganz catheter.

Spontaneous breathing trial failure

The initial SBT with a T-piece was performed in all patients who fulfilled the usual criteria [15]: $SpO_2 \geq 90\%$ with a $FiO_2 \leq 0.5$ and a PEEP ≤ 5 cmH_2O, hemodynamic stability defined by absence of vasopressors, no continuous sedation, Glasgow Coma Scale ≥ 10 (maximum score is 15 points), and a respiratory rate to tidal volume quotient <100 breaths/min/l.

The SBT with a T-piece was considered a failure if the patient developed one of the following signs or symptoms of poor tolerance within 60 min of its initiation: respiratory rate greater than 35 breaths/min, increased accessory muscle activity, unbearable dyspnea, persistent decrease in SpO_2 below 90%, heart rate above 140 beats/min or increased by 20%, arrhythmias, drop in systolic blood pressure below 80 mmHg or a rise above 160 mmHg, agitation, diaphoresis, or depressed mental status. The same criteria were used to define SBT failure when PSV-PEEP and PSV-ZEEP were tested.

Physiological measurements

Pulmonary artery occlusion pressure (PAOP) was recorded at the end of expiration. Measurements were performed at the end of each SBT, and LVHF was defined by a PAOP $\geq$18 mmHg [16]. PAOP was measured after systematic verification of zero reference at the level of right atrium.

Cardiac output was measured by the thermodilution method using 10 ml of saline at room temperature, and the average of three measurements was taken. Pulmonary artery blood was withdrawn in commercially available

pre-heparinized plastic syringes and was immediately analyzed with an ABL 520 gas analyzer (Radiometer, Copenhaguen). Airflow and the airway, esophageal, and gastric pressures were measured with standard methodology and proper testing [17]. Signals were digitized and stored for subsequent calculations. Details regarding these measurements and signal acquisition are provided in the Electronic Supplementary Material (ESM).

Physiological assessment of patient effort

Breathing pattern and minute ventilation were determined from the airflow signal. Respiratory muscle energy expenditure was quantified using the esophageal pressure-time product (PTP), taking intrinsic positive end-expiratory pressure (PEEPi) into account [10, 18]. The inspiratory work of breathing (WOB) per breath was computed according to common methods [18–20]. Details regarding these calculations are provided in the ESM.

Statistical analysis

Continuous variables are expressed as median and interquartile range (IQR). We compared the values obtained at the end of the three SBTs (T-piece, PSV-PEEP, and PSV-ZEEP), before reconnecting the patient to ACV. To test differences between SBTs, we used the nonparametric Friedman test because of the small sample size. If a significant difference appeared, a Wilcoxon test was used to perform pairwise comparisons. Statistical analysis was done using the SPSS statistical software (SPSS 13, Chicago, IL).

Results

The physiological study comparing the three SBT was performed in 14 patients. Twenty-nine patients could not be included. The study flowchart is shown in Fig. 1. Patients' clinical characteristics are given in Table 1.

Ventilatory parameters, gas exchange, and inspiratory muscle effort

Although there were no significant differences between ACV and PSV-PEEP in terms of respiratory rate and tidal volume, there was a marked difference between PSV trials and T-piece trial (Table 2). PEEPi increased significantly during PSV-ZEEP and T-piece (Table 2). The FiO_2 did not differ: it was 0.38 (0.3–0.44) during all the study. Arterial blood gases showed a significant increase

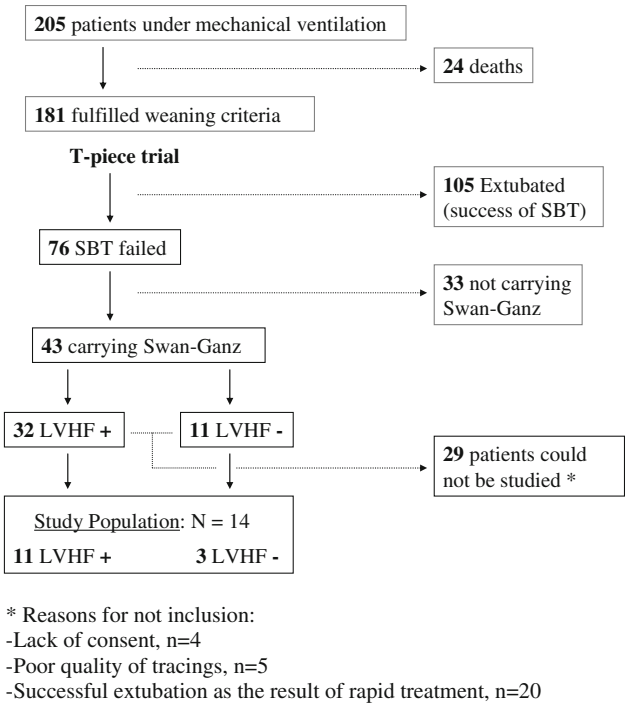

Fig. 1 Flow chart: Left ventricular heart failure (*LVHF*) defined by a pulmonary artery occlusion pressure ≥18 mmHg occurred in 32 patients, accounting for 74% of the patients with a Swan-Ganz catheter (43 patients) and 42% of the total group of patients who failed a T-piece trial (76 patients)

in $PaCO_2$ and a significant decrease in pH during T-piece, whereas PaO_2 did not significantly change (Table 2).

Patients' inspiratory effort increased markedly from both ACV and PSV-PEEP to PSV-ZEEP and T-piece, either expressed as PTP or WOB. Data are shown in Table 2, and representative tracings can be seen in Fig. 2.

Major expiratory muscle activity with positive pressure swings higher than 5 cmH_2O was observed in only three patients during PSV-ZEEP and T-tube trial and in no patients during PSV-PEEP.

Hemodynamic parameters and SBT tolerance

Four patients had a PAOP ≥18 mmHg while ventilated in ACV (nos. 1, 3, 8, and 12). PAOP was 18 mmHg or higher in 7 patients during PSV-PEEP, in 9 patients during PSV-ZEEP, and in 11 patients during the T-piece trial. Nearly 80% (8/11) of patients who had a PAOP ≥18 mmHg during the T-piece trial failure had prior cardiovascular disease: five had systemic arterial hypertension, and three had myocardial ischemia (Table 1). In addition, more than 50% (6/11) also had a chronic obstructive pulmonary disease (Table 1). Although PAOP showed a trend to increase from ACV to PSV-PEEP [15 (12–18) mmHg and 17 (14–22), $p = 0.05$], it increased

Table 1 Patient characteristics

N	Age (year)	Sex	SAPS II	Comorbidities	Reason for MV	PAOP ≥18 mmHg during T-piece	Duration of MV (days)	Hospital outcome
1	76	F	45	MI	Cardiac arrest[a]	Yes	40	D
2	66	F	19	No	Pneumonia	Yes	21	S
3	55	M	26	COPD	Mitral valve replacement[a]	Yes	11	S
4	56	M	29	COPD	Exacerbation of COPD	Yes	6	S
5	69	M	25	HT	Acute pancreatitis	Yes	21	D
6	69	M	45	COPD, HT	Exacerbation of COPD	Yes	6	S
7	79	M	53	MI	CABG	Yes	30	D
8	78	M	27	COPD, MI	Aortic valve replacement[a]	Yes	4	S
9	79	M	27	No	Abdominal surgery	No	16	S
10	58	M	22	COPD, HT	Pneumothorax	Yes	15	D
11	39	M	25	No	Septic Shock	No	10	S
12	75	M	68	COPD, HT	Abdominal aortic aneurysm[a]	Yes	21	S
13	69	M	30	HT	Abdominal surgery	Yes	30	D
14	67	M	37	COPD	Pneumonia	No	13	S
Median (IQR)	69 (58–77)		28 (25–45)				16 (9–23)	

SAPS simplified acute physiological score, *MV* mechanical ventilation, *PAOP* pulmonary artery occlusion pressure, *COPD* chronic obstructive pulmonary disease, *MI* myocardial ischemia, *HT* arterial hypertension, *CABG* coronary artery by-pass grafting, *S* survival, *D* death, *IQR* interquartile range

[a] PAOP >18 mmHg during volume assist-control ventilation

markedly during PSV-ZEEP [20 (15–25) mmHg] and during the T-piece trial [21 (18–24) mmHg]. Cardiac output and the mixed venous oxygen saturation remained unchanged (Table 3). Systolic blood pressure and heart rate increased significantly during T-piece (Table 3).

All patients failed the T-piece trial within the first 60 min of disconnection, but 11 (79%) successfully completed the PSV-PEEP trial, and 8 (57%) successfully completed the PSV-ZEEP trial. Individual data are shown in Table 4.

A cumulative (from the moment of admission) positive fluid balance was observed in four patients on the study day, but in no patients on the extubation day. Extubation was performed, on average, 5 days after the study day. The cumulative fluid balance was significantly lower when extubation was carried out: −935 (−3,343 to 342) ml the study day versus −5,065 (−11,710 to −3,530) ml the extubation day, $p < 0.01$ (Table 4).

Discussion

This study suggests that in our selected difficult-to-wean patients, clinical and physiological responses differ depending on the type of SBT used to ascertain whether or not a patient is ready for extubation. The breathing pattern and cardiovascular responses differed considerably among the three SBTs. Eleven of 14 (79%) patients who failed a T-piece trial successfully completed a PSV-PEEP trial, and 8/14 (57%) succeeded the PSV-ZEEP trial. PAOP that was normal during ACV increased to over 18 mmHg in 7/10 patients during T-piece, in 5/10 during PSV-ZEEP, and in only 3/10 during PSV-PEEP. This suggests that both mechanical ventilation and PEEP protected left ventricular function. In these selected individuals, the use of even low levels of ventilatory support may confuse clinicians about the clinical tolerance to unassisted spontaneous breathing.

T-piece versus pressure support trials: clinical implications

We found that the patient effort required during a T-piece trial was higher than during a PSV trial. Furthermore, we found that the effort was higher during the PSV-ZEEP trial than during the PSV-PEEP trial. As long as support was added, the failure rate during SBT decreased. Esteban et al. [8] found that more patients failed the T-piece trial (22%) than the PSV trial (14%). Ezingeard et al. [9] performed a PSV-ZEEP trial in 31 patients immediately after a T-piece trial failure and found that the PSV trial was successful in most patients (68%), a similar figure to our 57%. In these two studies [8, 9], patients who succeeded a PSV trial were extubated without a significant increase in re-intubation rate as compared to T-piece. Thus, it could be argued that we delayed extubation of those individuals who succeeded a PSV trial. However, we studied a selected population of difficult-to-wean patients: the median duration of mechanical ventilation was 16 days, they had failed a previous SBT with a T-piece, they also had multiple comorbidities, and they were relatively older than the populations in the other studies. In the study by Esteban et al. [8], two-thirds of the patients were successfully extubated after 6 days of mechanical ventilation and a first weaning trial.

Table 2 Comparison of respiratory parameters during T-piece and pressure support trials

	ACV	PSV-PEEP	PS-ZEEP	T-Piece
Respiratory frequency (breaths/min)	18 (16–24)	19 (16–29)	22 (16–34)*	27 (21–35)*
Tidal volume (ml)	443 (382–513)	354 (299–483)	328 (273–478)	325 (254–443)**
F/VT (breaths/min/l)	44 (35–60)	58 (35–88)	89 (39–97)*	92 (48–134)**
Minute ventilation (l/min)	9.2 (7.7–10.1)	8.8 (6.2–10.5)	9.5 (6.2–11.4)	8.2 (6.7–10.7)
pH (units)	7.39 (7.35–7.46)	7.37 (7.33–7.44)	7.35 (7.32–7.45)	7.34 (7.29–7.42)**
PCO2 (mmHg)	46 (40–53)	48 (44–54)	52 (44–58)	55 (43–60)*
PO2 (mmHg)	99 (83–111)	94 (85–105)	85 (79–105)	80 (69–95)
Intrinsic PEEP (cmH2O)	0.8 (0.2–6.3)	1.2 (0.6–5.2)	2.1 (1.1–8.6)*	3.8 (1.3–8.1)*
PTPes (cmH2O.s/min)	66 (24–173)	128 (58–299)	148 (100–465)*	292 (238–512)**
Work of breathing (Joule/min)	3.1 (1.1–8.8)	6.3 (1.0–15.1)	7.7 (5.3–22.2)*	12.6 (8.2–24.1)**

Values given were recorded immediately prior to patient reconnection to ACV

Data are expressed as median and (interquartile range)

ACV assist-control ventilation, *PSV* pressure support ventilation, *PEEP* positive end-expiratory pressure, *ZEEP* zero end-expiratory pressure, *F/VT* respiratory frequency (*F*) divided by the tidal volume (*VT*), *PTPes* esophageal pressure time product

* $p < 0.05$ between T-piece or PS-ZEEP versus PS-PEEP

** $p < 0.05$ between T-piece versus PSV-ZEEP and PS-PEEP

In the study by Ezingeard et al. [9], no patient had cardiac failure, and patients were successfully extubated after 8–9 days of mechanical ventilation. In addition, their study was conducted in patients who had been intubated with relatively narrow endotracheal tubes (7.5-mm internal diameter), and the vast majority of patients who were considered as T-piece trial failure were deemed so only on the basis of an increased respiratory rate. It is well known that even low levels of PSV profoundly modify the breathing pattern [21–23] and that narrow endotracheal tubes may promote an increase in respiratory rate [24].

Effects of PEEP and role of LVHF

We found that more patients succeeded the PS trial when a PEEP of 5 cmH_2O was added. There are several possible explanations. First, the application of PEEP was associated with a marked decrease in respiratory muscle energy expenditure. This is in agreement with previous reports that found that the magnitude of patient effort decreased by 30–40% [10]. Second, PEEP attenuated PEEPi, and according to findings by other authors [19, 25], this could also have reduced the WOB required to trigger the ventilator. Thirdly, PAOP values were decreased when PEEP was added. Indeed, PEEP is known to improve left ventricular performance [26], especially in patients with LVHF [27].

The large swings in pleural pressure that occurred when PEEP was not used (i.e., T-piece and PSV-ZEEP) increased afterload, thereby decreasing left ventricular performance [3]. The increase in PEEPi that was observed when external PEEP was withdrawn could also increase right ventricular afterload [5]. These phenomena may generate a vicious circle by further increasing both inspiratory effort and myocardial oxygen demand; this could potentially cause myocardial ischemia, mitral regurgitation, and a magnification in pulmonary edema, specially in patients with abnormal systolic ventricular function [28]. Interestingly, Jubran et al. [29] found a marked increase in the WOB due to an increase in inspiratory airway resistance and the development of PEEPi in patients who failed a T-piece trial. These abnormalities are compatible with an early phase of a cardiogenic pulmonary edema [29].

In a subsequent study, Jubran et al. [30] found a marked increase in PAOP (from 11 to 27 mmHg) and a drop in mixed venous oxygen saturation in patients who failed a T-piece trial. These patients were unable to increase their cardiac output to counteract the increase in oxygen consumption during spontaneous ventilation. In contrast, we did not find a drop in mixed venous oxygen saturation during failed SBTs. The discrepancies between the two studies could have several explanations. Our patients had a well-preserved cardiac output, suggesting that systolic function was relatively normal. Although we did not perform an echocardiography exam, we speculate that the main cause of LVHF might be diastolic dysfunction. Eight of our patients had cardiovascular comorbidities (hypertension or myocardial infarction) and had a median age of 69 years. It has been described that populations like this are prone to develop diastolic dysfunction [31–33].

The baseline PAOP was relatively high in our study, probably due to hypervolemia. Overload associated with a normal or high cardiac output may also avoid insufficient oxygen transport, and our patients were rapidly reconnected to the ventilator when the SBT failure occurred, thus minimizing the likelihood of a drop in mixed venous oxygen saturation. Finally, we cannot rule out that myocardial ischemia and/or dynamic mitral regurgitation could have contributed to the development of an increased PAOP [5, 28, 32, 34].

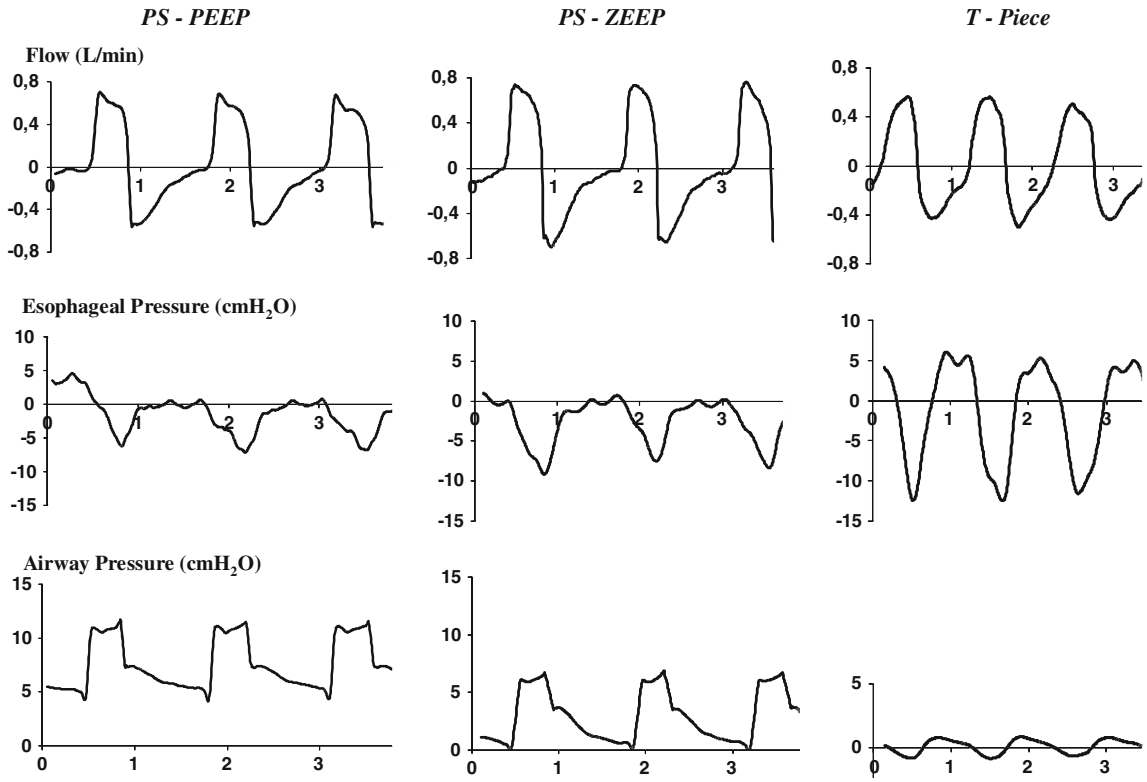

Fig. 2 Flow, esophageal and airway pressure signals (from *top* to *bottom*) showing a patient's ventilatory pattern during the three spontaneous breathing trials. Time is expressed in seconds on the *X axis*

Table 3 Comparison of hemodynamic parameters during T-piece and pressure support trials

	ACV	PS-PEEP	PS-ZEEP	T-Piece
Heart rate (beats/min)	88 (80–98)	89 (81–100)	94 (87–103)	98 (88–106)*
Systolic blood pressure (mmHg)	116 (108–131)	124 (118–139)	138 (118–158)	158 (126–162)**
PAOP (mmHg)	15 (12–18)	17 (14–22)	20 (15–25)*	21 (18–24)*
Cardiac output (l/min)	7.1 (6.1–7.6)	7.2 (6.1–7.8)	7.2 (6.1–7.8)	6.9 (6.3–7.8)
SVO_2 $(\%)_2$	69 (64–73)	69 (65–71)	69 (65–74)	69 (64–74)

Values given were recorded immediately prior to patient reconnection to ACV

Data are expressed as median and (interquartile range)

ACV assist-control ventilation, *PSV* pressure support ventilation, *PEEP* positive end-expiratory pressure, *ZEEP* zero end-expiratory pressure, *PAOP* pulmonary artery occlusion pressure, SVO_2 mixed venous oxygen saturation

* $p < 0.05$ between T-piece or PS-ZEEP versus PS-PEEP

** $p < 0.05$ between T-piece versus PS-ZEEP and PS-ZEEP

Limitations

As in other studies in this field [5, 35], a major limitation of the present work is selection bias, since all the included patients had failed a previous SBT and were monitored with a Swan-Ganz catheter.

Respiratory failure was considered to be associated with LVHF if PAOP reached at least 18 mmHg. Measures of PAOP above 18 mmHg suggest a hydrostatic cause of pulmonary edema, and although this threshold is used to differentiate pulmonary edema [16] from acute respiratory distress syndrome [36], several issues should be raised. The PAOP may be higher than 18 mmHg without hydrostatic edema due to elevations in pleural pressure generated by external PEEP or by PEEPi [37]. Patients with a chronically elevated PAOP (as seen in patients with valvular heart diseases) do not show the classical clinical signs and symptoms of acute pulmonary edema. Moreover, pleural pressure increases when expiratory muscles contract, and it is difficult to determine PAOP in this scenario [38]. Finally, we did not determine the real transmural PAOP [39, 40].

The Swan-Ganz catheter is a reference method to identify the onset of LVHF during weaning from mechanical ventilation [5, 34], but this is an invasive method and does not allow to determine the mechanism of LVHF. In contrast, echocardiography could detect

Table 4 Individual patient tolerance to pressure support trials, cumulative fluid balance, and extubation outcome

Patients	Success PSV-PEEP, *n* (%)	Success PSV-ZEEP, *n* (%)	Fluid balance at study day (ml)	Fluid balance at extubation day (ml)	Delay between study and extubation (days)
1[a]	No[b]	No[b]	−900	−5,440	3
2	No[b]	No[b]	−1,100	−16,800	12
3[a]	Yes[b]	Yes[b]	50	−5,700	3
4	Yes	Yes	−800	−3,500	2
5	Yes	No[b]	−5,300	Not extubated	–
6	No[b]	No[b]	−970	−330	4
7	Yes	Yes[b]	−4,250	−4,260	1
8[a]	Yes[b]	Yes[b]	−2,560	−3,620	1
9	Yes	Yes	−3,100	−3,380	1
10	Yes	Yes	−700	Not extubated	–
11	Yes	Yes	1,220	−4,690	1
12[a]	Yes[b]	No[b]	4,200	−11,900	15
13	Yes[b]	No[b]	4,600	−12,560	3
14	Yes	Yes	−9,000	−11,140	13
Median (IQR)	11 (79%)	8 (57%)	−935 (−3,343 to 342)	−5,065 (−11,710 to −3,530)	3 (1–10)

PSV pressure support ventilation, *PEEP* positive end-expiratory pressure, *ZEEP* zero end-expiratory pressure, *IQR* interquartile range

[a] Pulmonary artery occlusion pressure >18 mmHg during volume assist-control ventilation

[b] Pulmonary artery occlusion pressure >18 mmHg during the trial

diastolic heart failure when an episode of LVHF occurs in patients with a normal systolic function [5, 31, 32].

Although we found a negative cumulative fluid balance immediately before the study, we cannot exclude the possibility that the increase in PAOP was related to hypervolemia. It has been shown that a positive fluid balance was associated with failure to wean and with a higher risk of extubation failure [41, 42]. Our data actually suggest that fluid overload probably played a role since the cumulative fluid balance was significantly more negative on the extubation day than on the study day (Table 4). We did not systematically look for other potential causes of SBT failure, such as muscle weakness or critical illness-associated polyneuropathy. Our study is also limited by the fact that our clinical suspicion of diastolic heart failure (because of plausible clinical findings and symptoms, and a rapid response to vasodilators and diuretics) could not be assessed with echocardiographic examination.

Finally, we did not extubate the patients who succeeded a PSV trial. We did not do this because it has been shown that a spontaneous breathing trial using T-piece mimics the WOB performed after extubation well [43], and an extubation failure is associated with high mortality [44]. Nevertheless, we acknowledge that selected patients might benefit from noninvasive ventilation after a risky extubation [45, 46].

Conclusion

In this selected population of difficult-to-wean subjects, the patients' clinical and physiological behavior differed markedly when a confirmatory SBT was carried out with different techniques. The addition of ventilator support, in terms of PSV or PSV plus PEEP, profoundly modified the breathing pattern, inspiratory muscle effort, and the cardiovascular response when compared to a T-piece trial. Whether we should extubate difficult-to-wean patients who do not tolerate a confirmatory T-piece trial but do tolerate low levels of PSV without PEEP remains unsolved.

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