Changes in occlusion pressure ($P_{0.1}$) and breathing pattern during pressure support ventilation

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Received 26 January 1998
Returned to author 31 March 1998
Revised manuscript received 3 July 1998
Accepted for publication 7 October 1998

Abstract

***Background*—The purpose of this study was to investigate changes in breathing pattern, neuromuscular drive ($P_{0.1}$), and activity of the sternocleidomastoid muscles (SCM) during a gradual reduction in pressure support ventilation (PSV) in patients being weaned off controlled mechanical ventilation.**

***Methods*—Eight non-COPD patients recovering from acute respiratory failure were included in this prospective interventional study. All patients were unable to tolerate discontinuation from mechanical ventilation. Each patient was evaluated during a period of spontaneous breathing and during PSV. Four successive levels of PSV were assessed in the following order: 20 cm H_2O (PS20), 15 cm H_2O (PS15), 10 cm H_2O (PS10), and 5 cm H_2O (PS5).**

***Results*—When pressure support was reduced from PS20 to PS10 the respiratory rate (f) and the rapid shallow breathing index (f/VT) significantly increased and tidal volume (VT) significantly decreased. These parameters did not vary when pressure support was reduced from PS10 to PS5. Conversely, $P_{0.1}$ varied negligibly between PS20 and PS15 but increased significantly at low PSV levels. $P_{0.1}$ values were always greater than 2.9 cm H_2O (4.1 (1.1) cm H_2O) when SCM activity was present. When contraction of the SCM muscles reappeared the $P_{0.1}$ was the only parameter that changed significantly.**

***Conclusions*—In postoperative septic patients the value of $P_{0.1}$ seems to be more useful than breathing pattern parameters for setting the optimal level of pressure assistance during PSV.**

(*Thorax* 1999;**54**:119–123)

Keywords: airway occlusion pressure; pressure support ventilation; breathing pattern

After a prolonged period of controlled mechanical ventilation, spontaneous breathing is often poorly tolerated. About 20% of the patients who have recovered from the acute phase of their disease require prolonged mechanical assistance.[1–3] One explanation for this is an inability of the ventilatory muscles to cope with the imposed workload.[4 5] During the weaning process it is important to return loads to the patient's ventilatory muscles in a controlled fashion.

A useful approach to the progressive reloading of ventilatory muscles is pressure support ventilation (PSV). The major advantage of PSV is the possibility of gradually reducing the workload imposed on the respiratory muscles.[5 6] An optimal level of PSV can be set to maintain substantial diaphragm activity during weaning from mechanical ventilation without diaphragmatic fatigue.[5] This can be accurately determined only by monitoring diaphragmatic fatigue and the work of breathing. Unfortunately, these parameters require invasive measurement techniques.

It has been proposed that the breathing pattern may be a predictor of workload for patients.[7] Breathing parameters, however, appear to be insufficient to assess accurately the work of breathing.[8–10] The only clinical parameter that is a useful predictor of diaphragmatic fatigue during PSV is the contraction of the sternocleidomastoid (SCM) muscles.[6]

Measurement of $P_{0.1}$, the negative airway pressure generated during the first 100 ms of an occluded inspiration, was introduced by Whitelaw *et al*.[11] Because it is measured at zero flow and thus is independent of respiratory system compliance and resistance, it is an estimate of the neuromuscular drive to breathe. $P_{0.1}$ values differentiate between those patients who can be successfully weaned from mechanical ventilation and those who cannot.[12–14] High $P_{0.1}$ values reflect an increased neuromuscular activation of the respiratory system and indicate a strong likelihood of inspiratory muscle fatigue. $P_{0.1}$ has also been shown to be closely correlated with the work of breathing in patients receiving PSV.[9 10] It was recently suggested that $P_{0.1}$ is a better index than breathing frequency for estimating the change in load for the respiratory muscles.[15]

Few studies have described both the breathing pattern parameters and changes in $P_{0.1}$ during variation of PSV levels.[9 10] Although they reported an increase in $P_{0.1}$ values with the decrease in PSV levels, Alberti and coworkers[9] showed a greater sensitivity of this index for high PSV levels whereas Berger and associates[10] found a greater sensitivity for low PSV levels.

The objective of this study was to assess the changes in breathing pattern parameters and neuromuscular drive during gradual reduction of the PSV level in patients being weaned from mechanical ventilation and to determine whether a $P_{0.1}$ threshold exists when SCM contractions appear.

Methods

Eight patients recovering from acute respiratory failure of various causes were studied prospectively following approval by the institutional

Table 1 Clinical and respiratory characteristics of the patients during spontaneous breathing

Patient	Sex	Age (years)	Pa_{O_2} (mm Hg)	Pa_{CO_2} (mm Hg)	pH	PImax (cm H_2O)	$P_{0.1}$/Pi max	CMV duration (days)	Diagnosis
1	M	67	58	38	7.49	60	0.1	3	Postoperative sepsis (colonic surgery)
2	F	73	133	33	7.33	44	0.13	20	Postoperative sepsis (coronary bypass)
3	F	89	96	35	7.49	25	0.25	18	ARDS (pneumonia)
4	M	69	72	37	7.49	69	0.09	21	Postoperative sepsis (coronary bypass)
5	M	49	112	36	7.38	32	0.186	11	Postoperative sepsis (gastrectomy)
6	F	78	74	60	7.39	50	0.055	6	Postoperative sepsis (abdominal aortic surgery)
7	F	65	101	34	7.43	45	0.108	16	Postoperative sepsis (abdominal aortic surgery)
8	M	70	67	51	7.42	52	0.29	18	Postoperative sepsis (oesophagectomy)

Pa_{O_2}, Pa_{CO_2} = arterial oxygen and carbon dioxide tensions; PImax = maximal inspiratory pressure; $P_{0.1}$ = occlusion pressure; CMV = continuous mechanical ventilation; ARDS = adult respiratory distress syndrome.

ethics committee. All patients had undergone continuous mechanical ventilation for more than 48 hours. At the beginning of the study no patient had an internal positive end expiratory pressure (iPEEP) as measured during continuous mechanical ventilation by an end expiratory occlusion manoeuvre obtained by depression of the expiratory pause button on the Servo Ventilator 900C (Siemens, Berlin, Germany). The clinical and respiratory characteristics of the patients are shown in table 1. All patients were considered to be ready for weaning off ventilation by the following criteria: partial or total recovery from their underlying condition, body temperature <38.5°C, no evidence of infection, satisfactory renal function, neuropsychological state compatible with autonomous respiration, correct metabolic equilibrium, haemoglobin level >8 g/dl, absence of clinical signs of left ventricular dysfunction, and no cardiac rhythm or conduction disturbances. All sedative drugs, hypnotics, and narcotics were withheld for 24 hours prior to the weaning trial.

All weaning trials were performed in the morning. Patients were maintained in a semi-recumbent position. They underwent a 20 minute test period of spontaneous T-piece breathing with an F_{IO_2} of 40%. All spirometric, gas exchange, and pressure measurements ($P_{0.1}$, PImax) were then performed. If fewer than nine of the following 11 weaning criteria were satisfied, controlled ventilation was reinstituted and the patient was included in the study: effective cough, coordinated thoraco-abdominal movement, absence of SCM muscle activity, respiratory frequency (f) <35 breaths/min, tidal volume (VT) >5 ml/kg, minute ventilation (VE) <10 l/min, vital capacity (VC) >10 ml/kg, arterial oxygen tension (Pa_{O_2}) >60 mm Hg (>8 kPa) or Sa_{O_2} >90%, Pa_{CO_2} <50 mm Hg (6.66 kPa), Pa_{O_2}/F_{IO_2} ratio >200, and no cyanosis. After initial spontaneous T-piece breathing four successive levels of pressure support were used in the following order: (1) 20 cm H_2O (PS20), (2) 15 cm H_2O (PS15), (3) 10 cm H_2O (PS10), and (4) 5 cm H_2O (PS5) delivered via a Siemens Servo-Ventilator 900C. Each level was used for a trial period of 30 minutes. The trigger sensitivity was set to its minimal level (0 cm H_2O). No external PEEP was used. The F_{IO_2} was constant for each patient during the study. All recordings and measurements were obtained during the last 10 minutes of each trial in the following order: breathing pattern, palpation of SCM muscles, $P_{0.1}$ measurements.

MEASUREMENTS

Breathing pattern parameters were recorded for one minute using an electronic spirograph (Ultima SV, Datex, Helsinki, Finland) and the following measurements were made: VT, f, VE, inspiratory time (Ti), total time of the respiratory cycle (Ttot), ratio of inspiratory to total time of the respiratory cycle (Ti/Ttot), and the rapid shallow breathing index (f/VT). Airway pressure was recorded at the proximal end of the endotracheal tube with a differential pressure transducer ±50 cm H_2O (Validyne MP45) and a model CD15 carrier demodulator.

Table 2 Variation in breathing pattern and occlusion pressure ($P_{0.1}$) with different levels of pressure support (PS) ventilation

	f (breaths/min)	VE (l/min)	VT (l)	VT/Ti (l/s)	Ti/Ttot	f/VT (breaths/min/l)	$P_{0.1}$ (cm H_2O)
PS20	16 (4.3)	11.2 (2.1)	0.76 (0.15)	0.62 (0.21)	0.35 (0.06)	21.9 (6.7)	0.9 (0.3)
	12 to 20	8.2 to 13	0.63 to 0.88	0.45 to 0.80	0.30 to 0.40	16 to 27	0.6 to 1.1
PS15	24.2 (3.7)	11.9 (1.4)	0.51 (0.12)	0.55 (0.18)	0.36 (0.05)	51.2 (20.3)	1.4 (0.7)
	21 to 27	8.5 to 15.8	0.41 to 0.61	0.40 to 0.70	0.32 to 0.40	34 to 68	0.8 to 2
PS10	28.5 (5.2)	12.1 (2.4)	0.43 (0.08)	0.51 (0.12)	0.39 (0.04)	69.7 (23.3)	2.2 (1.2)
	24 to 33	10.1 to 14.1	0.36 to 0.50	0.41 to 0.61	0.36 to 0.42	50 to 89	1.2 to 3.2
PS5	28.1 (6.4)	12.1 (4.4)	0.41 (0.10)	0.47 (0.13)	0.40 (0.04)	74.4 (32.1)	4.1 (1.7)
	23 to 33	10.8 to 13.2	0.33 to 0.50	0.36 to 0.58	0.35 to 0.45	48 to 101	2.7 to 5.6
SB	30.9 (6.4)	10.6 (2.9)	0.34 (0.08)	0.42 (0.11)	0.42 (0.06)	95.1 (31.8)	6.7 (3.6)
	26 to 36	8.2 to 13	0.27 to 0.41	0.32 to 0.51	0.37 to 0.46	68 to 122	3.7 to 9.7
ANOVA repeated factor	p=0.0001	NS	p=0.0001	NS	NS	p=0.0001	p=0.0001
PLSD of Fisher							
PS20 vs PS15	p<0.05		p<0.05			p<0.05	NS
PS15 vs PS10	p<0.05		p<0.05			p<0.05	p<0.05
PS10 vs PS5	NS		NS			NS	p<0.05
PS5 vs SB	NS		p<0.05			p<0.05	NS

Values are mean (SD) with 95% confidence intervals.
f = respiratory rate; VE = minute volume; VT = tidal volume; VT/Ti = mean inspiratory flow; Ti/Ttot = ratio of inspiratory to total time of the respiratory cycle; f/VT = rapid shallow breathing index; $P_{0.1}$ = occlusion pressure; SB = spontaneous breathing.

Table 3 Activity of sternocleidomastoid (SCM) muscles with different levels of pressure support ventilation (PSV)

Level of PSV	Patient no. 1	2	3	4	5	6	7	8
PS20	−	−	−	−	−	−	−	−
PS15	−	−	−	−	−	−	−	−
PS10	−	−	−	−	−	−	−	+
PS5	+	+	+	+	+	−	+	+
SB	+	+	+	+	+	−	+	+

− = absence of SCM activity; + = presence of SCM activity; SB = spontaneous breathing.

$P_{0.1}$ measurement during spontaneous breathing (T-piece)

The $P_{0.1}$ was measured using a one way silent manually activated valve that was occluded at the end of expiration by means of a syringe. The patient saw neither the valve nor the operator, and thus could not anticipate the occlusion that lasted, on average, less than 300 ms.

$P_{0.1}$ measurement during PSV

The $P_{0.1}$ was measured using an end expiratory occlusion manoeuvre obtained by depressing the expiratory pause button on the Servo Ventilator 900C.[16] The inspiratory scissors valve remained closed at the end of expiration and the flap valve closed on the expiratory side, resulting in inspiratory effort against a closed system. Once the initial inspiratory effort was completed, the inspiratory button was released and normal respirations resumed.

Maximal inspiratory pressure (PImax)

The technique of Marini *et al*[17] was employed, using an unoccluded exhalation circuit, to measure PImax with a differential pressure transducer ±150 cm H_2O (Newark Electronics, USA). A minimum of three measurements were performed. The $P_{0.1}$/PImax ratio was then calculated. All signals were amplified, recorded and printed on a Gould Windograf recorder. $P_{0.1}$ and PImax measurements were made at paper speeds of 50 mm/s and 10 mm/s, respectively. A minimum of 3–5 reproducible measurements were performed for $P_{0.1}$, each separated by the time needed for a return to resting ventilation levels. Instruments were calibrated before each procedure with two water manometers.

Two physicians independently assessed activity of the SCM muscles by palpating the muscles in the neck. SCM contraction was considered to be present if there was agreement between the two physicians regarding the presence of SCM activity. The optimal level of PSV was defined by the lowest PSV level without SCM contraction.

The following parameters were continuously monitored: cardiac frequency, oscillometrically measured systolic blood pressure, mean and diastolic arterial blood pressure, end tidal CO_2, and pulse oximetry (Ultima SV, Datex, Helsinki, Finland). Arterial blood gases were sampled during mechanical ventilation and immediately following the spontaneous T-piece breathing trial via an arterial catheter.

The physician terminated the trial if a patient had any of the following signs of poor tolerance: f >35 breaths/min, Sao_2 <90%, heart rate >140 beats/min, systolic blood pressure >180 mm Hg or <90 mm Hg, agitation, or anxiety.

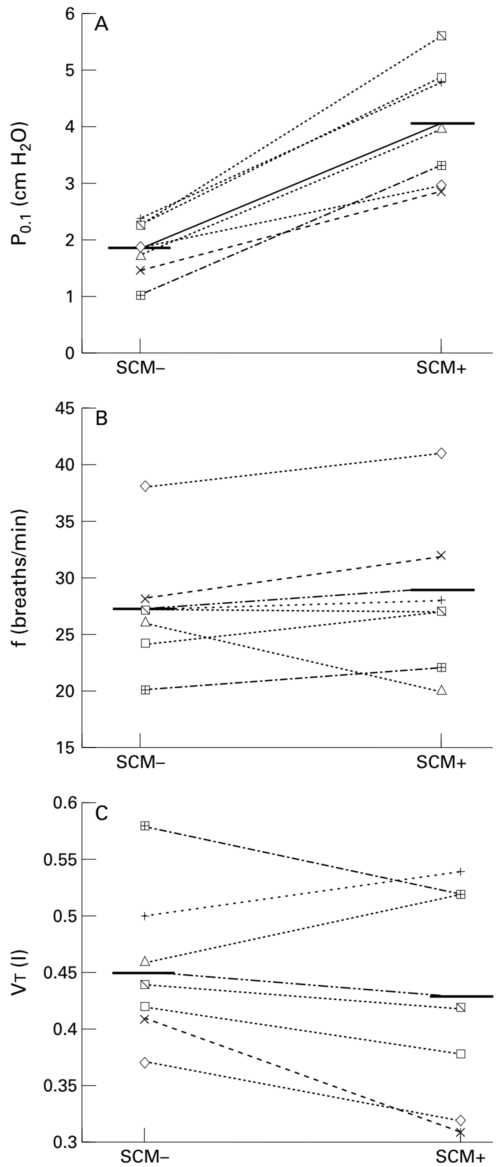

Figure 1 Variations in (A) occlusion pressure ($P_{0.1}$), (B) respiratory frequency (f), and (C) tidal volume (VT) with contraction of the sternocleidomastoid (SCM) muscle. SCM− = the lowest PSV level for each patient without SCM activity; SCM+ = the first PSV level at which SCM activity occurred. $P_{0.1}$ was the only parameter significantly modified when SCM muscle activity was present. Horizontal lines indicate mean values.

STATISTICAL ANALYSIS

Results are expressed as mean (SD) values with 95% confidence intervals. The ANOVA test for repeated measurements was simultaneously applied to all five treatments and the comparison test used was the PLSD of Fisher. The Wilcoxon test for small samples was used to compare quantitative variables when activity of the SCM muscles was present. A p value of <0.05 was considered significant.

Results

The breathing pattern and occlusion pressure data are summarised in table 2. When pressure

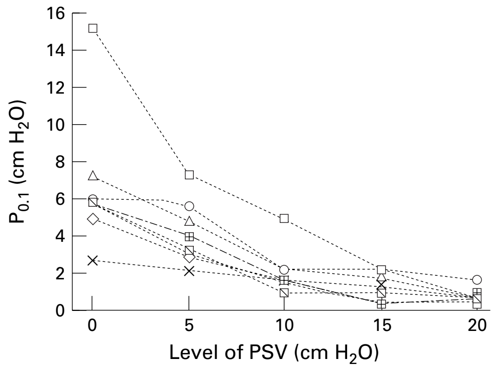

Figure 2 Individual values of $P_{0.1}$ with different levels of pressure support ventilation (PSV).

support was reduced, f and f/VT significantly increased from PS20 to PS15 and from PS15 to PS10 and VT decreased significantly between these stages. These parameters did not vary significantly between PS10 and PS5. Conversely, there was only negligible variation in $P_{0.1}$ between PS20 and PS15 but it increased significantly from PS15 to PS10 and from PS10 to PS5. VE, VT/Ti, and Ti/Ttot did not vary significantly when the PSV level was reduced.

Contraction of the SCM muscles occurred in seven patients during spontaneous breathing and at PS5, but only in patient 8 at PS10 (table 3). Only $P_{0.1}$ was modified when SCM muscle activity was present (fig 1; $p<0.05$). When there was no SCM muscle activity $P_{0.1}$ values were always lower than 2.9 cm H_2O (1.8 (0.5) cm H_2O). No activity of the SCM muscles was seen in patient 6 during spontaneous breathing and PSV, and his $P_{0.1}$ was lower than 2.8 cm H_2O at each stage. Individual values of $P_{0.1}$ are shown in fig 2.

All patients remained stable with no signs of poor tolerance during the trial.

Discussion

In postoperative septic patients we have shown that the value of $P_{0.1}$, as measured using the technique of Brenner *et al*,[16] appears more useful than breathing pattern parameters for setting the optimal level of pressure assistance during PSV. When SCM muscle contraction occurred it was the only parameter significantly modified .

Several authors have found that VT varies directly, and f and f/VT inversely, with the level of PSV.[18–20] Thus, when muscles are almost unloaded (at high levels of PSV) the patient breathes with high VT and low frequency. VT and Ti depend only on the level and cessation criteria of PSV and the mechanical properties of the system.[21 22] In the present study VT was more than 10 ml/kg at PSV20, indicating that the respiratory muscles were almost totally unloaded. This is in agreement with other studies.[7 10] In contrast, an excessive workload (for low levels of PSV) leads to rapid shallow breathing. However, as already described,[10 23] VT, Ti, and f did not change significantly at low levels of PSV10 to PSV5.

During weaning off continuous mechanical ventilation it is difficult to determine the optimal level of PSV as defined by the maintenance of diaphragm activity and the avoidance of diaphragmatic fatigue. Specific indexes of inspiratory muscle fatigue are only obtained using invasive techniques. However, in the study by Brochard *et al*[5] contraction of SCM muscles was evaluated at the bedside and appeared at the same time as diaphragmatic fatigue, as evaluated by electromyography. In the present study none of the breathing pattern parameters was significantly modified when contraction of SCM muscles reappeared. Other authors have found that, for an "acceptable breathing pattern" with f between 15 and 25 breaths/min, the respiratory muscle workload can be excessive leading to fatigue and predisposing to muscle atrophy.[8] Conversely, persistent tachypnoea (range 22–38 breaths/min) occurred in the absence of patient work of breathing.[10] Similarly, Alberti *et al*[9] found no correlation between breathing pattern parameters and work of breathing. This suggests that breathing pattern parameters are not an accurate assessment of the optimal threshold of PSV.

Occlusion pressure has been shown to be a predictor of the success of weaning in patients with or without obstructive lung disease.[12–14 24] High levels of $P_{0.1}$ are associated with increased respiratory effort and indicate an inability to breath independently with success. In contrast, lower values of $P_{0.1}$ are associated with effective weaning. Moreover, Murciano *et al*[13] have shown that during weaning trials a good relationship exists between $P_{0.1}$ and the high to low ratio of the diaphragmatic electromyogram. In our study $P_{0.1}$ varied inversely with the level of PSV. More interesting was the fact that $P_{0.1}$ did not change significantly with high levels of PSV. Similarly, Berger *et al* found that $P_{0.1}$ did not vary with further increases in PSV to levels above the crossover to total unloading.[9] At these high levels we assume that patients were almost completely unloaded and the only work of breathing was to trigger the ventilator. Conversely, $P_{0.1}$ changed significantly with lower PSV levels when contraction of the SCM muscles occurred. At this time inspiratory effort increases in order to keep alveolar ventilation in an acceptable range. Thus, an acute increase in the $P_{0.1}$ value may signal an insufficient PSV level. In a recent study Lotti *et al*[25] found a significant concomitant reciprocally opposed change in $P_{0.1}$ and PSV level.

Seven patients exhibited SCM muscle activity during which the mean $P_{0.1}$ value was 4.1 (1.1) cm H_2O. When no activity of these muscles was detected the $P_{0.1}$ values were always lower than 2.9 cm H_2O. During PSV Alberti *et al*[9] reported that $P_{0.1}$ was closely correlated with the work of breathing and found, by extrapolation from linear regression analysis, a $P_{0.1}$ value of 3.2 cm H_2O corresponding to a work of breathing "threshold" level of 0.75 J/l of ventilation. It has been suggested that weaning may be indicated at this value of work of breathing.[26] Our results corroborate this threshold in non-COPD patients. Berger *et al*[10] reported higher $P_{0.1}$ values when patients were almost totally unloaded, but COPD and non-COPD patients were mixed in this study.

In conclusion, the results of this preliminary study suggest that, in postoperative septic patients, a $P_{0.1}$ cut off of 2.9 cm H_2O separates patients with positive SCM activity from those with negative SCM activity. This hypothesis now needs to be tested on a much larger group of patients in order to know whether a specific cut off value of $P_{0.1}$ can be used in practice to adjust PSV step by step downwards and if a programme incorporating this strategy is more successful in weaning patients than some other programmes.

The authors are grateful to Dr Laurent Brochard for very useful comments. The authors thank Yves Goudard for his technical assistance.

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