

Polysomnography in stable COPD under non-invasive ventilation to reduce patient–ventilator asynchrony and morning breathlessness

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Abstract

Background Stable severe chronic obstructive pulmonary disease (COPD) patients with chronic hypercapnic respira-

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tory failure treated by nocturnal bi-level positive pressure non-invasive ventilation (NIV) may experience severe morning deventilation dyspnea. We hypothesised that in these patients, progressive hyperinflation, resulting from inappropriate ventilator settings, leads to patient–ventilator asynchrony (PVA) with a high rate of unrewarded inspiratory efforts and morning discomfort.

Methods Polysomnography (PSG), diaphragm electromyogram and transcutaneous capnography (PtcCO₂) under NIV during two consecutive nights using baseline ventilator settings on the first night, then, during the second night, adjustment of ventilator parameters under PSG with assessment of impact of settings changes on sleep, patient–ventilator synchronisation, morning arterial blood gases and morning dyspnea.

Results Eight patients (61±8 years, FEV₁ 30±8% predicted, residual volume 210±30% predicted) were included. In all patients, pressure support was decreased during setting adjustments, as well as tidal volume, while respiratory rate increased without any deleterious effect on nocturnal PtcCO₂ or morning PaCO₂. PVA index, initially high (40±30%) during the baseline night, decreased significantly after adjusting ventilator settings ($p=0.0009$), as well as subjective perception of PVA leaks, and morning dyspnea while quality of sleep improved.

Conclusion The subgroup of COPD patients treated by home NIV, who present marked deventilation dyspnea and unrewarded efforts may benefit from adjustment of ventilator settings under PSG or polygraphy.

Keywords Non-invasive ventilation · COPD · Patient–ventilator asynchrony

Abbreviations

COPD Chronic obstructive pulmonary disease
EEG Electroencephalogram

EMG	Electromyogram
EPAP	Expiratory positive airway pressure
I/E ratio	Inspiratory/expiratory ratio
IPAP	Inspiratory positive airway pressure
kHz	Kilohertz
kPa	Kilopascals
MAI	Microarousal index
NIV	Non-invasive ventilation
PaCO ₂	Arterial partial pressure of carbon dioxide
PEEP	Positive end-expiratory pressure
PEEP ₁	Intrinsic positive end-expiratory pressure
PSG	Polysomnography
PtcCO ₂	Transcutaneous measurement of arterial partial pressure of carbon dioxide
PVA	Patient–ventilator asynchrony
RR	Respiratory rate
SE	Sleep efficiency
SpO ₂	Oxygen saturation of Haemoglobin measured by pulse oximetry
TI _{MAX}	Maximal inspiratory time
TST	Total sleep time
VE	Minute ventilation
V _T	Tidal volume
WASO	Wake after sleep onset

Introduction

Despite the lack of evidence to support its use in chronic obstructive pulmonary disease (COPD), non-invasive ventilation (NIV) is widely prescribed in this indication in Europe [1]. Although impact on survival remains controversial [2–4], most authors will agree that there appears to be a group of COPD patients who clearly benefit from NIV. Indeed, several studies have described improvements in health-related quality of life [3, 5, 6], hospital admissions [7, 8], dyspnea [6] and quality of sleep [5].

Recent reviews have stressed the importance of identifying abnormal events occurring under NIV, and adjusting ventilator settings accordingly [9]. For instance, it is now established that patient–ventilator asynchrony (PVA) is frequent in patients undergoing chronic NIV who are considered effectively treated [10, 11]. PVA has a negative impact on quality of sleep and potentially on treatment efficacy [10]. Monitoring pulse oximetry (SpO₂) and transcutaneous capnography (PtcCO₂) alone do not explore this aspect of therapy adequately. Thus, in selected cases, polygraphy or polysomnography (PSG) might be ideal monitoring tools in addition to oxy-capnography for patients with home ventilation [12].

Among severe COPD under home NIV, patients are occasionally burdened by a severe dyspnea when interrupting their treatment after a night of NIV. We hypothesised that in these patients, inappropriate ventilator settings may lead to dynamic hyperinflation, increased intrinsic PEEP, resulting in unrewarded inspiratory efforts [13, 14] and morning discomfort when interrupting NIV [15]. We surmised that adjusting ventilator settings accordingly, during PSG under NIV, might improve quality of sleep, patient–ventilator synchronisation and morning dyspnea (‘deventilation dyspnea’).

Materials and methods

Patients

Eligible patients fulfilled the following inclusion criteria: (1) Severe COPD according to GOLD guidelines [16], (2) home NIV initiated according to prevailing recommendations [17], (3) stable clinical condition defined as absence of exacerbation and of modifications of ventilator settings in the month preceding the study, (4) compliance to NIV of more than 4 h/day assessed by ventilator software, (5) presence of deventilation dyspnea defined as severe dyspnea occurring immediately after interruption of non-invasive ventilation and preventing the patient from getting out of bed and carrying out usual daily activities for more than 30 min after one night of NIV. Severe morning dyspnea was assessed by a chest therapist using a modified Borg scale. All patients with morning dyspnea score >4 were proposed to enter the study. Exclusion criteria were: (1) use of NIV in an acute exacerbation of COPD for hypercapnic ventilatory failure, (2) association of COPD with restrictive thoracic disorders (i.e. morbid obesity, cyphoscoliosis, neuromuscular disease, sequelae of tuberculosis), and (3) patients under 18 years old or not able to give informed consent.

Measurements

Polysomnography

Conventional polysomnography with patient under NIV included: standard electroencephalography (seven electrodes, F₄–F₃, O₁–O₂, C₃–C₄, C_z), left and right electrooculography, submental electromyography (EMG), measurement of airflow using a pneumotachograph, body position, thoracic and abdominal movements (Remlogic 1.1, Embla, Netherlands) and video recording. SpO₂ was measured continuously with a pulse oximeter and a finger probe. PtcCO₂ measurements were performed using a

transcutaneous capnograph with an ear probe heated at 43°C (Tosca 500, Radiometer, Switzerland). Sleep was scored according to standard criteria using 30-s epochs [18]. The investigator scoring the sleep study was blinded to the ventilator settings and subjective evaluation. The following sleep parameters were quantified: total sleep time (TST), sleep efficiency (SE—TST/total recording time \times 100), percentage of each sleep stage, wake after sleep onset (WASO) and sleep latency. As indices of sleep fragmentation, we considered the index of micro-arousals (MAI). MAI were defined as a return to alpha or fast frequency, well-differentiated from the background EEG activity lasting >1.5 s.

Diaphragm EMG

Surface diaphragm EMG was recorded using silver electrodes in all subjects only to monitor inspiratory effort [19] and facilitate detection of ineffective inspiratory efforts. The electrodes were placed in the right and left anterior axillary line in the seventh intercostal space with a horizontal distance of 2 cm. Diaphragmatic EMG was sampled at 0.2 kHz, bandpass-filtered between 10 and 70 Hz [20] and recorded with all other parameters on the Embla recording system.

Patient–ventilator asynchrony

Patterns of patient–ventilator asynchrony included ineffective triggering (unrewarded inspiratory efforts), double-triggering and auto-triggering, as defined by Thille et al. [13]. Unrewarded inspiratory efforts were identified as the occurrence of a definite diaphragmatic EMG signal, and/or presence of inspiratory thoracoabdominal movements on strain gauges without subsequent pressurisation by the ventilator. Auto-triggering was defined as a cycle delivered by the ventilator and not triggered by the patient. An unrewarded effort index was calculated as follows: [number of desynchronized cycles/total respiratory rate (i.e. cycles supported by the ventilator plus unrewarded efforts)] \times 100 (expressed as a percentage) [21]. Only epochs (3 min.) with an unrewarded effort index above 10% were scored as periods with asynchrony based on a study by Vittaca and al. [22]. PVA index was calculated as the ratio of total sleep time spent with unrewarded efforts on TST [10].

Ventilator built-in software

Compliance, estimated tidal volume (V_T), minute ventilation (VE), respiratory rate (RR), leaks and percent (%) of spontaneous inspirations were downloaded after both nights from built-in software of bi-level ventilators

(ResScan software, version 1.3, ResMed, Sydney, Australia). Estimation of leaks and V_T by ventilator software of VPAP III and VPAP IV ventilators (ResMed, Sydney, Australia) has been shown to be reliable in two recent independent studies [23, 24].

Questionnaires

After both nights, to evaluate patient comfort and patients' perception of patient–ventilator synchronisation, airflow, leaks and noise of ventilator, an eight-item visual analogue scale (VAS) was administered the morning following each sleep study [25]. Higher values indicate better comfort. Patients were also asked to quantify their dyspnea on a visual analogue scale while withdrawing from pressure support in the morning (10—worst dyspnea ever experienced in the morning, 0—no dyspnea at all).

Study protocol

Patients were admitted to the sleep laboratory for two consecutive nights. On the first night, ventilator settings were those normally used by the patient, thus eliminating the need of an accommodation night. This first night aimed to detect and quantify PVA in highly symptomatic COPD patients on their usual NIV settings. In our institution, ventilation settings are adjusted by chest therapists, under the supervision of the attending pulmonary physician, aiming at patient comfort and optimal control of daytime and nocturnal SpO_2 and $PtcCO_2$. On the second night, the therapeutic target was to reduce sleep-disordered breathing and patient–ventilator asynchrony. The investigators adjusted ventilator settings using polysomnography and monitoring of $SpO_2/PtcCO_2$.

Our approach was as follows: in case of unrewarded inspiratory efforts, we first reduced pressure support, then, if necessary, we increased expiratory positive airway pressure (EPAP) [15] and set expiratory trigger at a higher percentage of peak inspiratory flow to avoid delayed cycling [26, 27] under close monitoring of $PtcCO_2$. When time to peak pressure (pressurisation time) was set at 100 msec, we increased it by 50–100 msec. Back-up respiratory rate was increased if unrewarded inspiratory efforts persisted. Maximal inspiratory time (TI_{MAX}) was calculated in order to maintain a physiological I/E ratio for COPD patients between 1:2.5 and 1:3.

If auto-triggering was identified, mask fit was adapted to prevent air leaks and inspiratory trigger sensitivity was reduced [28]. Inspiratory time (TI_{MIN}) was increased if double triggering was frequent [13]. Special attention was

given to mask leaks in order to limit their impact on patient–ventilator asynchrony.

Statistical analysis

Statistical testing was performed using Prism4 software (GraphPad, San Diego, CA, USA). When data were normally distributed (Shapiro–Wilk test), results were expressed as mean±SD. All variables between the two nights of the study protocol were compared using a paired *t* test. Differences were considered significant when probability of a type I error was <0.05.

The study protocol was approved by the Ethics Committee for Medical Research of Geneva University Hospital and written informed consent was obtained from all patients. The trial was registered at www.clinicaltrials.gov (Trial No. NCT01180439)

Results

The study flow chart is depicted in Fig. 1. Among severe COPD patients treated by our outpatients clinic, 9/32 (28%) exhibited ‘deventilation dyspnea’ and fulfilled inclusion criteria. We included eight consecutive COPD patients (aged 61±8.3 years, three males, five females, on NIV for 14.3±12.5 months), admitted for a scheduled routine control of their nocturnal ventilation (*n*=4 on VPAP ST III, *n*=4 on VPAP IV bi-level ventilators; ResMed, Sydney, Australia) between October 2009 and

March 2010. One patient declined. Patients’ main characteristics are shown in Table 1. All patients used their ventilator more than 4 h per night (Mean±SD9:06±3:20 hours) and suffered from severe morning dyspnea after interrupting their NIV (referred to as ‘deventilation dyspnea’).

Ventilator settings at baseline and adjustments under PSG

Baseline ventilator settings and settings after adjustment under PSG are shown in Table 2. Seven patients used full-face masks and one patient used a nasal interface. The major change performed in order to decrease unrewarded inspiratory efforts detected on PSG, was a decrease in pressure support in all cases ([IPAP–EPAP] baseline, 13.6±1.8 cm H₂O; adjusted, 10.3±1.7 cm H₂O; *p*=.0005). In seven cases, IPAP was reduced by 1–5 cm H₂O. In four cases, EPAP was also increased by 1–4 cm H₂O. Back-up rate was increased in four cases. Time to peak was slightly increased in all cases by 50–100 msec. TI_{MAX} was adjusted to obtain a I/E ratio between 1:2.5 and 1:3.

Sleep structure and respiratory events under NIV

Unrewarded inspiratory efforts were the most prevalent patient–ventilator asynchrony. PVA index was reduced after ventilator adjustment from 40.5±31.0 to 6.7±7.3%, *p*=0.009 (Fig. 2). Neither double triggering nor auto-triggering were identified during both nights. Fig-

Fig. 1 Study flow chart

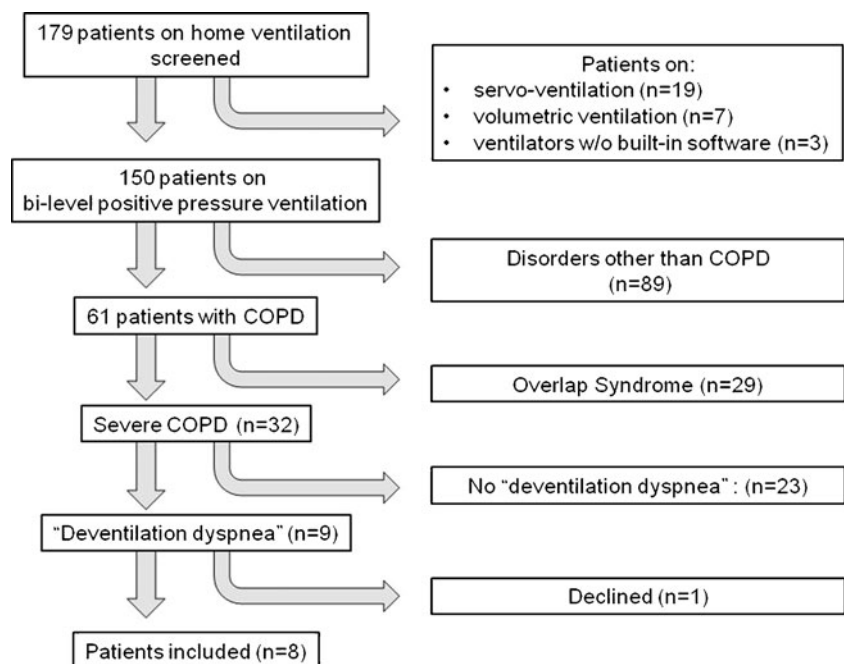


Table 1 Patients main characteristics

Patients	Age	Gender	Ventilated for (months)	FEV ₁ (% predicted)	TLC (% predicted)	RV (% predicted)
1	56	M	39	25	120	240
2	62	M	4	21	124	217
3	58	F	3	30	130	231
4	65	F	3	23	129	222
5	52	M	10	28	118	213
6	79	F	14	39	154	228
7	62	F	17	30	111	185
8	55	F	24	47	108	147
Mean	61		14.3	30.4	124.2	210.4
SD	8		12.5	8.7	14.3	30.4

ure 3 shows a representative correction of unrewarded inspiratory efforts by NIV adjustment under sleep polygraphy.

Neither total sleep time (TST 445±126 vs. 448±96 min), wake after sleep onset (WASO 41±21 vs. 41±24 min), nor micro arousal index (MAI 18±9 vs. 20±12 h) were altered by changes in ventilator settings.

Subjective assessment

The major impact of ventilator adjustment was a marked decrease in morning dyspnea measured by an adapted Borg Scale (values from 0 to 10, increasing values mean increasing dyspnea), for all subjects as shown in Fig. 3 (5.8±2.5 vs. 2.3±1.6, $p=0.006$).

Table 2 Ventilator settings before and after adjustment under PSG

Patients	IPAP (cmH ₂ O)	EPAP (cmH ₂ O)	Pressure support (cmH ₂ O)	Back-up rate (/min)	Time to peak (ms)	TI _{MAX} (s)
Initial ventilator settings						
1	22	5	17	13	100	1.2
2	18	6	12	14	100	1.2
3	18	6	12	14	100	1.2
4	19	5	14	14	100	1.2
5	16	4	12	12	100	1.5
6	19	4	15	14	100	1.4
7	18	5	13	14	100	1.1
8	22	8	14	14	100	1.3
Mean	19	5.4	13.6	13.6	100	1.26
SD	2.1	1.3	1.8	0.7	0	0.13
Adjusted ventilator settings						
1	17	5	12	13	200	1.2
2	17	6	12	14	200	1.1
3	14	5.4	8.6	14	150	1.3
4	15	6	9	19	150	1.1
5	14	6	8	16	150	1.1
6	16	4	12	14	150	1.4
7	18.6	9	9.6	19	200	1.1
8	20	9	11	17	200	1.2
Mean	16.5*	6.3***	10.3**	15.8***	175**	1.19***
SD	2.1	1.8	1.7	2.4	26.7	0.11

FEV₁ Forced expiratory volume in 1 s, TLC total lung capacity, RV residual volume, IPAP inspiratory positive airway pressure, EPAP expiratory positive airway pressure, RR respiratory rate, TI_{MAX} maximal inspiratory time

* $p=0.04$; ** $p<0.0005$; *** $p = NS$, P values for paired t tests comparing baseline vs. adjusted ventilator settings

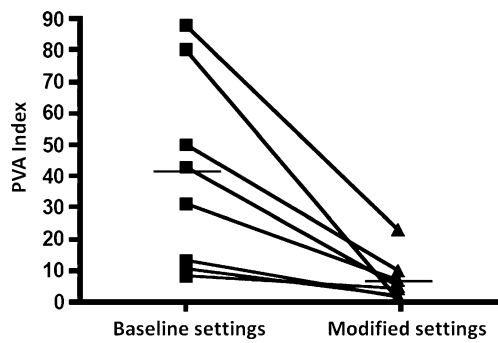
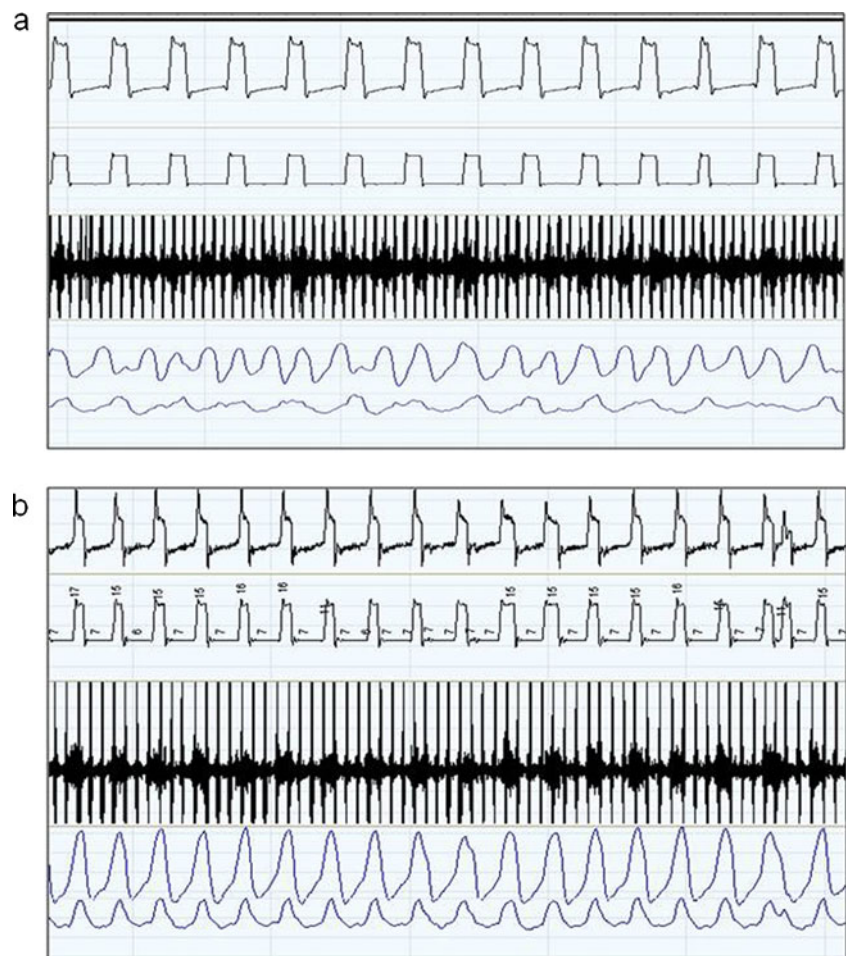


Fig. 2 Patient–ventilator asynchrony index (PVA index: total time spent with unrewarded efforts/total sleep time) for each patient before and after NIV adjustment with polysomnography

The eight-point VAS also showed significant improvements for perception of patient–ventilator synchronisation ($p=0.04$), perception of leaks ($p=0.04$) and overall quality of sleep ($p=0.01$) (Fig. 4). None of the VAS scales decreased (i.e. worsened).

Fig. 3 a Example of unrewarded inspiratory efforts with initial ventilator settings. From top to bottom—flow, pressure, diaphragm EMG, thoracic and abdominal belts. This 60-s epoch shows major patient–ventilator asynchrony. Unrewarded efforts can easily be identified by comparing the EMG trace or that of the abdominal and thoracic belts with the flow or pressure traces. The patients' inspiratory efforts do not trigger the ventilator which is on its back-up rate.

b Correction of patient–ventilator asynchrony by adjustment of ventilator settings with polysomnography (60-s epoch). Flow, pressure, diaphragm EMG and abdominal and thoracic belts are perfectly synchronised. All inspiratory efforts trigger the ventilator resulting in an increased respiratory rate



Data provided by ventilator built-in software

Changes in estimated tidal volume (V_T), total ventilation (VE), spontaneous respiratory rate (RR) and percentage of inspiratory cycles triggered by the patient are reported in Table 3. Ventilation pattern changed after adjusting ventilator parameters, with a significant decrease in V_T , and increase in spontaneous RR . The increase in percentage of inspiratory cycles triggered by the patient most probably reflects a better patient–ventilator synchronisation and improved detection of inspiratory efforts by the ventilator [12]. Average leaks were low during both recordings for all patients with a nonsignificant trend for lower leaks (95% percentile) after adjusting ventilator settings (28 ± 25 vs. 17 ± 19 L/min, $p=0.057$).

Nocturnal SpO_2 and nocturnal/daytime CO_2 values

Mean nocturnal SpO_2 was $92\pm 3\%$ on the first night and $91\pm 2\%$ after adjustment of settings under PSG (p value NS). Oxygen desaturation index was low and did not differ

Fig. 4 Correction of deventilation dyspnea by adjusting ventilator settings with polysomnography is illustrated for each individual patient on a modified Borg scale (a, upper left). On a 0–10 visual analogue scale, subjective assessment of ventilator synchronisation (b), leaks (c) and overall quality of sleep (d) all improved

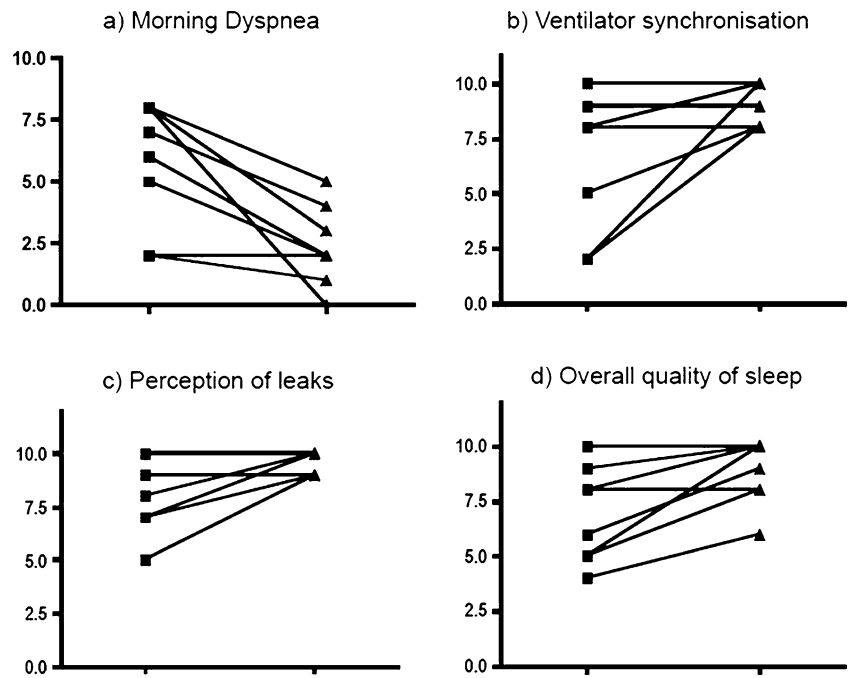


Table 3 Data downloaded from ventilator software, transcutaneous capnography and arterial blood gases: baseline values and values after adjusting ventilator parameters

Patients	V_T (ml)	RR (/min)	VE (l/min)	Percent (%) of spontaneous inspirations	PtcCO ₂ (kPa)	PaCO ₂ (kPa)
Initial ventilator settings						
1	850	16	13.8	52	7.7	6.5
2	660	14	7.6	36	6.7	5.7
3	410	17	6.3	9	6.9	7.6
4	650	15	8.2	7	6.6	8.4
5	650	12	10	31	9.6	10
6	650	14	8	25	6.3	6.9
7	350	21	10.8	90	8.9	8.4
8	600	17	8.1	90	7.4	7.6
Mean	603	15.8	9.1	43	7.5	7.6
SD	157	2.7	2.4	33	1.2	1.3
Adjusted ventilator settings						
1	450	26	13.2	96	8.6	6
2	380	18	7.3	86	6.8	5.8
3	340	20	7	40	6.8	6.9
4	480	21	6.2	63	6.8	7.9
5	350	17	9.4	46	8.8	10.5
6	500	18	7.8	85	6.4	6.5
7	350	20	11	63	7.8	7.7
8	600	17	8.1	40	6.4	7.1
Mean	431*	19.6**	8.8***	65***	7.3***	7.3***
SD	93	3	2.3	22	1	1.5

V_T Tidal volume, RR respiratory rate, VE minute ventilation, PtcCO₂ transcutaneous CO₂, PaCO₂ morning arterial CO₂ under NIV

* $p=0.01$; ** $p=0.02$; *** $p = NS$, P values for paired t tests comparing baseline vs. adjusted ventilator settings

between both nights (3.8 ± 6.0 vs. 3.5 ± 2.9 , p value NS). Despite a significant reduction in pressure support after adjusting ventilator settings, mean PtcCO₂ decreased slightly although not significantly (7.5 ± 1.2 vs. 7.3 ± 1.0 kPa). Average morning PaCO₂ under NIV also decreased after adjustment of ventilator settings (7.6 ± 1.3 vs. 7.3 ± 1.5 kPa, $p=0.057$).

Discussion

Among severe COPD patients treated at home by NIV, nearly one third complained of ‘deventilation dyspnea’. Our study shows in this population that a very high rate of patient–ventilator asynchrony may occur (mainly unrewarded inspiratory efforts) and remain undetected without PSG. Adjusting ventilator settings after monitoring of nocturnal NIV by PSG can: (1) improve markedly patient–ventilator synchronisation, particularly triggering of ventilator; (2) improve patient comfort; (3) lead to a marked decrease in morning ‘deventilation dyspnea’; (4) change ventilation pattern to a more physiological mode for severe COPD, i.e. increase spontaneous respiratory rate with a slight decrease in tidal volume, without any deterioration of PaCO₂ or PtcCO₂.

The observation that a high rate of unrewarded efforts occurred and was undetected in these patients confirms previous observations showing a high rate of patient–ventilator asynchrony in stable patients under NIV, most often not reported by the patient [10, 11, 14]. Although systematic polysomnography is clearly not warranted in stable patients under NIV, in these eight cases, PSG was indicated by the importance of morning deventilation dyspnea and the modest impact of NIV on PtcCO₂ [12]. Adjusting ventilator settings had a nonsignificant impact on arterial blood gases, but a marked effect on patient comfort, patient–ventilator synchrony and morning dyspnea. Whether these changes are relevant as major endpoints of NIV is debatable, but it seems reasonable to assume that these improvements may have an impact on quality of life and acceptance of treatment. Indeed, morning deventilation dyspnea is a source of important discomfort and distress in these patients.

It may be objected that correction of alveolar hypoventilation in our patients was insufficient. Indeed, both morning arterial blood gases and nocturnal capnography show a residual hypercapnia. Recent publications by Windisch et al. suggest that normalising PaCO₂ under NIV may be an important goal, through the use of high levels of pressure support and high back-up respiratory rates: this approach was shown to be feasible, to improve daytime PaCO₂, daytime dyspnea, HRQL and FEV₁ [6, 29, 30]. The use of high-intensity ventilation and its application in clinical

practice still remains a subject of debate. Indeed, several publications on long-term NIV in COPD have used much lower insufflation pressures [2–5]. In our centre, our aim is to obtain the best compromise between reduction of PaCO₂/PtcCO₂, induced leaks and patient tolerance. Thus, for all patients, initial settings of pressure support were as high as tolerated. Residual hypercapnia in very severe COPD treated by NIV is frequently reported. All patients included in our study had severe COPD according to GOLD criteria, had had several exacerbations with acute hypercapnic respiratory failure, and had a baseline PaCO₂ >7.3 kPa.

The mechanisms suggested for the relationship between nocturnal PSG findings and ‘deventilation dyspnea’ are hypothetical and probably differ according to the underlying pathology. In selected subjects with severe COPD and static hyperinflation, excessive pressure support might lead to dynamic hyperinflation and increased PEEP_i [31] which are major determinants of unrewarded efforts [21]. Dynamic hyperinflation has been shown to correlate strongly with exercise-induced dyspnea in COPD [32] and may explain the importance of ‘deventilation dyspnea’. Therefore, patient–ventilator asynchrony may, in these subjects, be a marker of inappropriate ventilator settings as demonstrated in the acute setting [26]. Arguments in favour of such a mechanism are—in the patients studied—severity of bronchial obstruction and the presence of static air trapping which may curtail the patients’ ability to take in large tidal volumes on NIV without developing dynamic hyperinflation (Table 1). However, the setting of this study did not allow performing serial measurements of inspiratory capacity after cessation of NIV; furthermore, PEEP_i could not be estimated non-invasively.

NIV can thus be considered as a two-edged sword in severe COPD. On one hand, Diaz et al. have shown that efficient NIV may decrease residual volume, functional residual capacity and total lung capacity when compared to sham NIV [33]. On the other hand, inappropriately high pressure support may have in certain patients a negative impact on lung mechanics and increase unrewarded efforts and patient–ventilator asynchrony [26]. Finding the best compromise between too high pressure support (with patient–ventilator asynchrony), efficacy of ventilation and patient comfort under monitoring by polygraphy or PSG might be a relevant clinical target.

Limitations of this study are its non-blinded and non-randomised design. The sequence of night sleep studies were dictated by necessity of a pragmatic step-by-step assessment: the first night to identify patient–ventilator asynchrony during sleep, the second to correct them under appropriate monitoring. As patients were long-time NIV users, the first night effect on sleep quality can be considered as negligible. Moreover, ineffective efforts are objective measurements not influenced by randomization. Thus, we believe that the initial PSG reliably reflected patient–ventilator interactions at home. The advantage of our study design was to validate an effective and

feasible procedure for NIV adjustment in clinical practice for COPD complaining from ‘deventilation dyspnea’. The sample size is another limitation of our study. This may have precluded the demonstration of significant improvements in objective sleep quality which is suggested by nearly significant increases in sleep efficiency ($69\pm 18\%$ vs. $76\pm 13\%$, n.s.) and slow wave and REM sleep (% N3+REM $29\pm 12\%$ vs. $38\pm 14\%$, n.s.).

In summary, this study shows that in a selected group of stable severe COPD with hyperinflation treated by NIV, patient–ventilator asynchrony and ineffective inspiratory efforts are frequent, most probably increasing work of breathing and decreasing the efficacy of ventilation; this observation may explain the occurrence of morning deventilation dyspnea. After a diagnostic PSG or polygraphy, adjusting ventilator settings may lead to a significant decrease of ineffective inspiratory efforts and a significant improvement of patient comfort during and after NIV.

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Conflict of interest None.

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