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Physiological effects of posture on mask ventilation in awake stable chronic hypercapnic COPD patients

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Physiological effects of posture on mask ventilation in awake stable chronic hypercapnic COPD patients. R. Porta, M. Vitacca, E. Clini, N. Ambrosino. ©ERS Journals Ltd. 1999.

ABSTRACT: Stable chronic hypercapnic patients are often prescribed long-term mask noninvasive pressure support ventilation (NPSV). There is a lack of information on the effects of posture on NPSV. Therefore posture induced changes in physiological effects of NPSV in awake stable chronic hypercapnic patients were evaluated.

In 12 awake chronic obstructive pulmonary disease (COPD) patients breathing pattern, respiratory muscles, mechanics and dyspnoea (by visual analogue scale: VAS) were evaluated during spontaneous breathing (SB) in sitting posture and during NPSV in sitting, supine and lateral positions randomly assigned. Arterial blood gases were evaluated during SB and at the end of the last NPSV session (whatever the posture).

As expected NPSV resulted in a significant improvement in carbon dioxide tension in arterial blood (P_{a,CO_2}) (from 7.4±0.85 to 6.9±0.7 kPa). When compared with SB, sitting NPSV resulted in a significant increase in tidal volume and minute ventilation and in a significant decrease in breathing frequency. Inspiratory muscle effort as assessed by oesophageal pressure swings and pressure-time product per minute (from 14±4.8 to 6.2±3.5 cmH_2O, and from 240±81 to 96±60 $cmH_2O \cdot s \cdot min^{-1}$ respectively), intrinsic dynamic positive end expiratory pressure (from 2.7±2.3 to 1.4±1.3 cmH_2O) and expiratory airway resistance (from 18±7 to 5±3 $cmH_2O \cdot L \cdot s^{-1}$) decreased during sitting NPSV, whereas VAS did not change. Changing posture did not significantly affect any parameter independently of the patients weight, whether obese or not.

In awake stable hypercapnic chronic obstructive pulmonary disease patients changing posture does not significantly influence breathing pattern and respiratory muscles during noninvasive pressure support ventilation suggesting that mask ventilation may be performed in different positions without any relevant difference in its effectiveness.
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The respiratory effects of different postures during spontaneous breathing (SB) in healthy subjects and in patients with chronic obstructive pulmonary disease (COPD) have been widely studied [1, 2]. Differences in breathing pattern and mechanics have been reported when changing from upright or sitting to supine posture [3, 4]. In COPD patients supine position has been shown to be associated with more "effective" diaphragmatic function in comparison to sitting [5]. Changing posture was also reported to have important effects on breathing pattern, ventilation distribution and arterial blood gases in intubated and mechanically ventilated patients with severe acute hypoxaemic respiratory failure [6].

There is a lack of information on the physiological effects of posture during mechanical ventilation in stable COPD patients. This is relevant in the light of the fact that noninvasive positive pressure ventilation (NPPV) has been gaining increasing popularity to reverse acute or chronic respiratory failure (CRF) [7, 8]. Although the consensus is greater for CRF being consequent upon nonobstructive conditions than for COPD [8] a recent randomized clinical trial has shown that nocturnal noninvasive pressure support ventilation (NPSV) may be a useful addition to long-term oxygen therapy in hypercapnic COPD [9].

Although aimed to improve nocturnal hypoventilation, home NPSV is often prescribed after in-hospital practice sessions performed during wakefulness in a single position which may not necessarily be that used by patients during the night. Recently SCHONHOFER *et al.* [10] compared nocturnal and daytime NPPV in patients with CRF due to restrictive disorder. They did not show any significant differences on improvement in carbon dioxide tension in arterial blood (P_{a,CO_2}) between applications [10]. At present, no data are available on the physiological effects of posture during NPSV, namely on the reported unloading effects [11]. The aim of this study was therefore to evaluate breathing pattern, mechanics and respiratory muscle function in stable chronic hypercapnic patients performing NPSV in different positions.

Methods

The Institutional Ethical Committee of Salvatore Maugeri Foundation, Gussago, Italy approved the investigative protocol of the study which was conducted according to the Declaration of Helsinki. Informed consent was obtained from the patients before the start of the procedure.

Patients

Twelve stable chronic hypercapnic COPD patients were studied. Diagnosis of COPD was made according to the American Thoracic Society (ATS) guidelines [12]. One patient suffered from associated fibrothorax, another had undergone right lobectomy 8 yrs before the study. In addition oxygen tension in arterial blood (P_{a,O_2}) and P_{a,CO_2} had to be <8.0 kPa (60 mmHg) and >6.0 kPa (45 mmHg) respectively, during SB of room air. As a matter of fact all of the patients in this study were well known in the authors' institution where they had been referred to the outpatients clinic for periodic medical visits and arterial blood gas controls to adjust their treatments. At the time that they were recruited to this study, they were all in stable condition, as assessed by stability in blood gas values and (pH >7.35) and were free from exacerbation in the preceding 4 weeks. Patients with other organ failure, cancer or an inability to cooperate were excluded from the study. All patients were on long-term oxygen therapy. Four patients were on a home NPSV program with a bi-level ventilator (BIPAP®; Respironics Inc., Murrysville, PA, USA), for 6–7 hrs during the night. Among others, 7 patients had experienced NPSV for acute exacerbations of their disease months before the study. All of the patients received regular treatment with inhaled bronchodilators but neither systemic nor inhaled steroids, apart from for exacerbations [13]. No change in the routine medical and oxygen therapy was made in the week before the study. The anthropometric, demographic and functional characteristics of all of the patients are illustrated in table 1.

Measurements

Body mass index (BMI) was calculated as body weight (kg·height·m^{-2}) and, according to the classification of the panel on energy, obesity, and body weight standard [14] and patients were defined as obese when their BMI was ≥29 kg·m^{-2}. Routine static and dynamic lung volumes were measured by means of a volume constant body plethysmograph (CAD-NET system 1085; Medical Graphic Corp., St. Paul, MN, USA) with the patient in the seated posture according to standard procedure, from days to weeks before the study (table 1) [15]. Arterial blood gases, were analysed by means of an ABL 330 radiometer (Radiometer, Copenhagen, Denmark), on blood samples drawn from the radial artery while breathing room air or with oxygen supplementation maintaining an arterial oxygen saturation (S_{a,O_2}) of >90%. Dyspnoea sensation was measured by means of a visual analogue scale (VAS) [16].

For the experimental procedure of this study, flow (V') and pressure at the airway opening (P_{ao}) were measured by means of a pneumotachograph pressure transducer (Bicore Irvine, CA, USA). The pneumotachograph was inserted between the nasal mask and the "plateau valve" of the ventilator circuit. Volume (V) was obtained by numerical integration of the flow signal. Changes in pleural pressure (P_{pl}) were estimated from changes in oesophageal pressure (P_{oes}) by means of the balloon-catheter technique with an oesophageal balloon catheter connected to a differential pressure transducer (±140 cmH_2O; Bicore, Irvine, CA, USA). Transpulmonary pressure (P_L) was obtained by subtraction of P_{oes} from P_{ao}.

Table 1. – The demographics, anthropometrics and respiratory function in patients according to body mass index (BMI)

	All patients (n=12)	BMI <29 (n=6)	BMI ≥29 (n=6)	p-value
Age yrs	66±5	67±3	64.5±6.5	NS
Sex M/F	10/2	6/0	4/2	
Height cm	166±7.5	166±8.3	166±7.4	NS
Weight kg	77±18	65±10	89±13.4	<0.005
BMI kg·m^{-2}	29±6	25±2.5	34±4.5	<0.001
FEV_1 L	0.90±0.31	0.76±0.31	1.02±0.26	NS
FEV_1 % pred	34±11.4	28±10.4	40±10	NS
FVC L	1.53±0.40	1.30±0.32	1.77±0.32	<0.02
FVC % pred	47±15	38±8.6	56±14	<0.02
FEV_1/FVC %	58±11	57±11	58±11	NS
RV/TLC %	64±9	70±9	59±5	<0.02
RV % pred	173±80	199±103	148±43	NS
PH	7.39±0.03	7.38±0.02	7.39±0.03	NS
P_{a,CO_2} kPa	7.4±0.85	7.6±1.06	7.3±0.6	NS
mmHg	55.6±6.4	57±8	55±5	
P_{a,O_2} kPa	7.3±0.5	7.2±0.4	7.5±0.26	NS
mmHg	55±4	54±3	56±2	

Data are presented as mean±SD. M: male; F: female; FEV_1: forced expiratory volume in one second; % pred: percentage of the predicted value; FVC: forced vital capacity; RV: residual volume; TLC: total lung capacity; P_{a,CO_2}: carbon dioxide tension in arterial blood; P_{a,O_2}: oxygen tension in arterial blood; p: level of statistical significance between obese (BMI ≥29) and nonobese patients; NS: nonsignificant.

Data analysis

The Bicore (Irvine, CA, USA) facilities were not used to process the signals. Rather, through the analogue output of the Bicore, the signals of V', P_{ao}, and P_{oes} were digitized by an analogue-to-digital converter with 12-bit resolution and fed into a personal computer at a sampling frequency of 100 Hz. The subsequent analysis was performed using a software package (Computo; Elekton Agliano, Terme, Italy). Tidal volume (V_T), respiratory frequency (f_R) and minute ventilation (V'_E), were computed from the volume signal. Total cycle duration (t_{tot}), inspiratory time (t_I), expiratory time (t_E), mean inspiratory flow (V_T/t_I) and duty cycle (t_I/t_{tot}), were calculated from the flow signal, as average values from 3 min continuous records of V' and V. Intrinsic dynamic positive end-expiratory pressure ($PEEPi_{,dyn}$) was measured as the negative deflection in P_{oes} swing from the onset of inspiratory effort to the onset of inspiratory flow. Changes in the magnitude of the effort of the inspiratory muscles were estimated from changes in P_{oes} swing. P_{oes} tidal swings were measured as well as the pressure-time product (PtP)for the inspiratory muscles calculated over a period of 1 min ($PtP_{,min}$) and also corrected per litre of ventilation ($PtP_{,min}/V'_E$). P_L was used to calculate pulmonary resistance at mid-inspiration (R_{awe}) according to the Mead and Whittemberger technique [17].

Setting of the ventilator. NPSV was delivered through a commercial nasal mask (Respironics, Murrysville, PA, USA) by means of a portable ventilator able to compensate for leaks (BiPAP®; Respironics Inc.). The ventilator was used in spontaneous mode (S) with peak inspiratory pressure at maximal values tolerated by the patients (mean 15±3SD cmH_2O) adding a positive end-expiratory pressure (PEEP) of 4 cmH_2O.

Experimental procedure. The study was performed under continuous monitoring of S_{a,O_2} (Kolormon; Kontron Instruments, Watford, UK). In all patients, awake in semirecumbent position, after the application of topical anaesthesia (xylocaine spray 10%), the balloon-tipped catheter was inserted through the nose into the middle third of the oesophagus and thereafter automatically inflated to 0.5 mL. The occlusion test [18] was performed to verify the correct positioning of the oesophageal balloon, and it was satisfactory in every instance. Thereafter the nose mask was applied and connected to the pneumotachograph. The commercial nasal mask was appropriately sized for each patient. Special care was devoted to ensure mouth closure throughout the procedure. A nurse not involved in the study was always present for patient care. The ventilator circuit was equipped with the Sanders NRV-2 valve (Respironics Inc.) to prevent CO_2 rebreathing [19].

Statistical analysis

Results are expressed as mean±1SD. All variables used were analysed by means of Skewness, Kurtosis and Wilk′s W Statistic for testing normality. Differences among postures and between sitting SB and NPSV were evaluated by analysis of variance (ANOVA) for repeated measures. Post-hoc or t-test with Bonferroni adjustment were applied as required by ANOVA interaction. A p-value <0.05 was considered to be statistically significant. Regression analysis was used to assess relationships between VAS and PEEPI,dyn and *Pt*P,min.

Results

The demographics, anthropometrics and lung function of all of the patients in the study are shown in table 1. Six out of 12 patients were obese with a BMI of 34±4.5 $kg \cdot m^{-2}$ (in comparison with BMI 25±2.5 $kg \cdot m^{-2}$ of non-obese patients).

Table 2. – Breathing pattern and respiratory mechanics during spontaneous breathing and noninvasive pressure support ventilation (NPSV) in sitting posture in all patients (n=12)

	Sitting SB	p-value	Sitting NPSV
*f*R bpm	19±5.5	<0.05	17±5.6
*V*T mL	464±102	<0.02	653±155
*V*T/*t*I $L \cdot s^{-1}$	0.37±0.11	<0.02	0.47±0.12
*t*I/*t*tot	0.39±0.04	NS	0.38±0.05
*V′*E $L \cdot min^{-1}$	9±2.4	<0.05	11±3
PEEPI,dyn cmH_2O	2.7±2.36	<0.01	1.4±1.3
*Pt*P,min $cmH_2O \cdot s^{-1} \cdot min^{-1}$	240±81	<0.01	96±60
*Pt*P,min/*V′*E $cmH_2O \cdot s^{-1} \cdot min^{-1}$	30±12	<0.005	8.8±5
*P*oes,swing cmH_2O	14±4.8	<0.01	6.2±3.5
*R*awe $cmH_2O \cdot L^{-1} \cdot s^{-1}$	18±7	<0.01	5±3

Data are presented as mean±SD. *f*R: respiratory frequency; bpm: breaths per minute; *V*T: tidal volume; *t*I: duration of inspection; *t*tot: duration of total breathing cycle; *V′*E: minute ventilation; PEEPI,dyn: intrinsic dynamic positive end-expiratory pressure; *Pt*P,min: pressure time produce of the inspiratory muscles per minute; *Pt*P,min/*V′*E: pressure time produce per minute corrected for ventilation; *P*oes,swing: oesophageal pressure swing; *R*awe: expiratory airway resistance; NS: nonsignificant.

All of the patients completed the experimental procedure. As expected NPSV resulted in a significant decrease in P_{a,CO_2} (from 7.4±0.85 to 6.9±0.7 kPa; p<0.005) whereas the target S_{a,O_2} >90% was maintained in all patients ($P_{a,O_2}/F_{I,O_2}$ from 274±61 to 261±38). Table 2 and figure 1 show the effects of NPSV on breathing pattern and respiratory mechanics. As expected, when compared with SB, sitting NPSV resulted in a significant increase in *V*T and *V′*E, and a significant decrease in *f*R. Inspiratory muscle effort as assessed by *P*oes swings, *Pt*P,min, *Pt*P,min/*V′*E significantly decreased during NPSV in addition to, PEEPI,dyn and *R*awe. Table 3 shows the effect of different postures on breathing pattern and indices of inspiratory

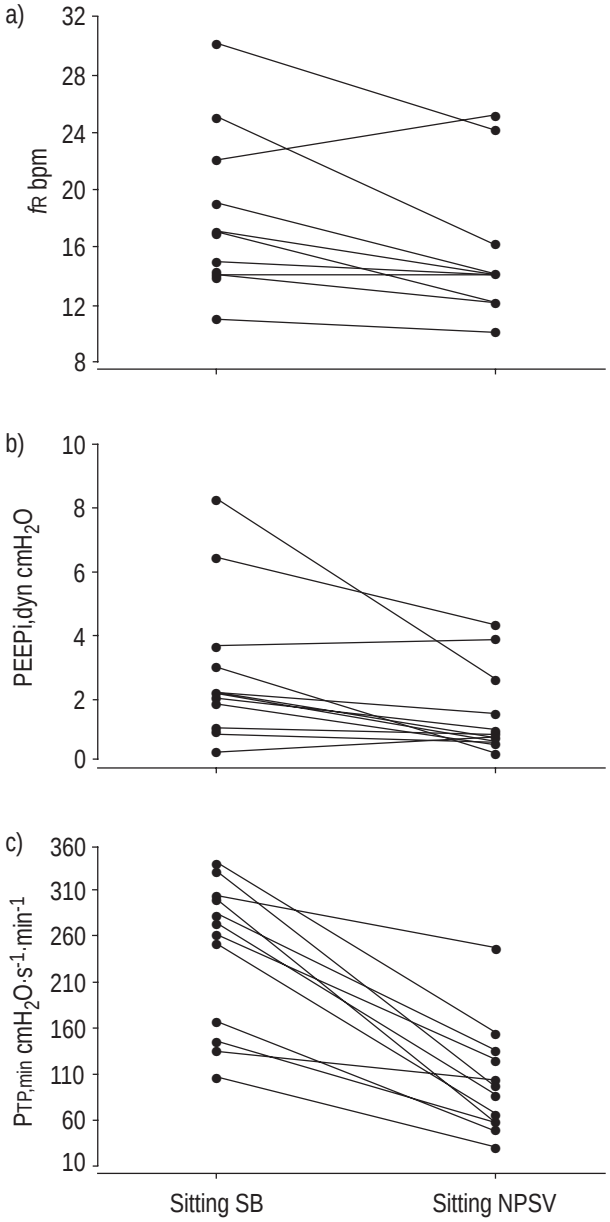

Fig. 1. – Noninvasive pressure support ventilation (NPSV) induced individual patient (n=12) changes in a) respiratory frequency (*f*R); b) intrinsic dynamic positive end expiratory pressure (PEEPI,dyn); and c) pressure time product of the inspiratory muscles per minute (*Pt*P,min), in sitting spontaneous breathing (SB) and sitting NPSV. bpm: breaths per minute.

Table 3. – Breathing pattern and respiratory mechanics in different postures during noninvasive pressure support ventilation (NPSV) in all patients (n=12)

	Sitting	Supine	Lateral
f_R bpm	17±5.6	15±4.5	18±4
V_T mL	653±155	788±264	670±158
V_T/t_I L·s^{-1}	0.47±0.12	0.47±0.1	0.51±0.12
t_I/t_{tot}	0.38±0.05	0.38±0.07	0.40±0.05
V'_E L·min^{-1}	11±3	11.4±2.5	12±2
PEEPI,dyn cmH_2O	1.4±1.3	1.8±1.5	1.8±1.7
PtP,min cmH_2O·s^{-1}·min^{-1}	96±60	107±42	104±64
PtP,min/V'_E cmH_2O·s^{-1}·min^{-1}	8.8±5	9.4±5.6	9±6
$P_{oes,swing}$ cmH_2O	6.2±3.5	7.3±2.6	6.5±3.5
R_{awe} cmH_2O·L^{-1}·s^{-1}	5±3	6±2	5±3

Data are presented as mean±SD. f_R: respiratory frequency; bpm: breaths per minute; V_T: tidal volume; t_I: duration of inspection; t_{tot}: duration of total breathing cycle; V'_E: minute ventilation; PEEPI,dyn: intrinsic dynamic positive end-expiratory pressure; PtP,min: pressure time produce of the inspiratory muscles per minute; PtP,min/V'_E: pressure time produce per minute corrected for ventilation; $P_{oes,swing}$: oesophageal pressure swing; R_{awe}: expiratory airway resistance.

muscle effort under NPSV. No significant difference between sitting posture and each of the other positions was found in any parameter.

Figure 2 shows individual changes of f_R, PEEPI,dyn and PtP,min in all patients. Table 4 and table 5 show the effects of NPSV on inspiratory muscles and mechanics according to BMI. Obesity had no effect upon any of the results.

NPSV did not influence dyspnoea as assessed by VAS (10±9%, 7±6%, 9±8% and 10±9% at baseline and during sitting, supine and lateral NPSV, respectively). There was no correlation between dyspnoea and PtP,min or PEEPI,dyn during NPSV.

Discussion

This study shows that changing posture does not significantly influence the effect of NPSV on breathing pattern and respiratory muscles function of awake stable hypercapnic patients with COPD whether overweight or not.

Long-term NPPV has been proposed with the theoretical goals of resting inspiratory muscles, improving respiratory muscle function, resetting respiratory drive, improving the recruitment of nonventilated pulmonary zones, improving quality of sleep and control of nocturnal hypoventilation [21]. For long-term use the ventilator is usually set after a training period performed during wakefulness in a single position which ma y not necessarily be that used by the patients during sleep. Daytime mechanical ventilation in awake patients was reported to be as equally effective in reversing chronic hypercapnia as nocturnal mechanical ventilation [10]. On the contrary, the most effective posture to be adopted during long-term NPPV has still to be defined. Only 38% of 26 papers published since 1991–1998 report the position adopted by the patients during NPPV in stable CRF patients; among these 79% report sitting and 21% report supine position. To the best of the authors′ knowledge this is the first study of the effects of

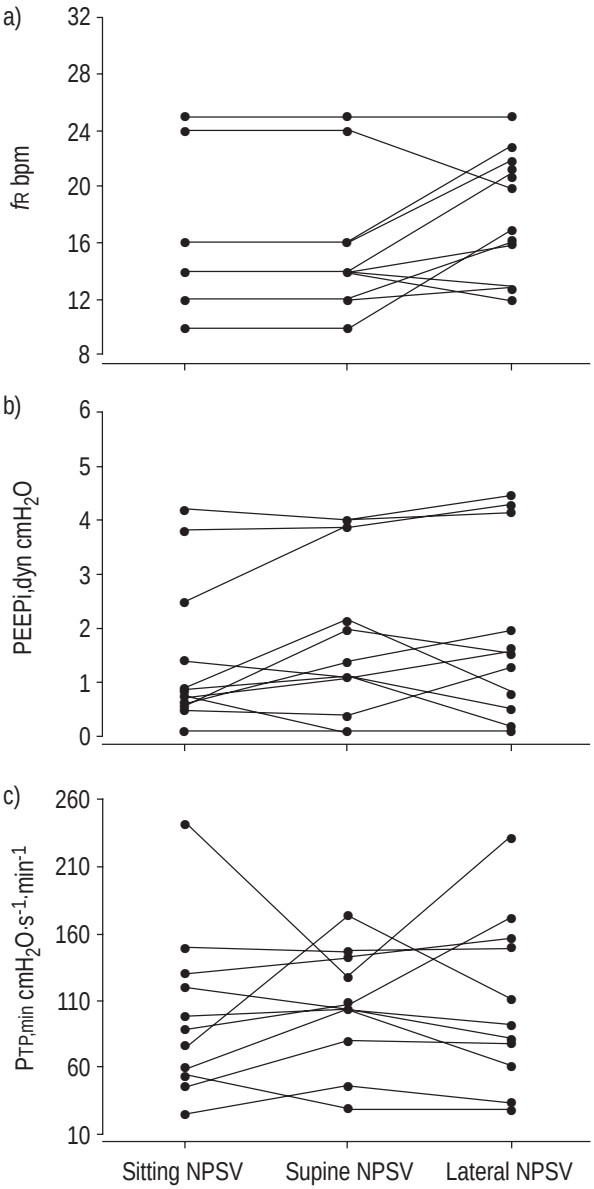

Fig. 2. – Individual changes (n=12) in different postures in: a) respiratory frequency (f_R); b) intrinsic dynamic positive end-expiratory pressure PEEPI,dyn and PtP,min. Abbreviations as in figure 1.

different postures on respiratory muscles and breathing pattern in awake stable COPD patients during NPSV.

The respiratory effects of different postures in normal subjects and patients breathing spontaneously are still being discussed [1–6]. Significant decreases in V_T, V'_E and V_T/t_I [22–24], a decrease in lung compliance and an increase in respiratory resistance in supine *versus* sitting position have been reported by some authors [25] but not by others [26]. In obese subjects a reduction in functional residual capacity (FRC) and dynamic lung compliance and an increase in respiratory resistance are reported when the supine posture is adopted [27, 28]. Fewer data are available in COPD: in these patients relief of dyspnoea was reported when shifting from upright seated to supine position and attributed to better diaphragmatic force-generating ability, this position allowing lengthening of diaphragm fibres [5]. The current study confirms

Table 4. – Inspiratory muscles effort and respiratory mechanics during spontaneous breathing (SB) and noninvasive pressure support ventilation (NPSV) in sitting posture according to body mass index (BMI)

	Sitting SB		Sitting NPSV	
	BMI <29	BMI ≥29	BMI <29	BMI ≥29
PEEPI,dyn cmH_2O	2.1±1.1	3.3±3.2	1.2±1.3*	1.55±1.5§
$P_{oes,swing}$ cmH_2O	15±4.5	12±4.8	7±3.8*	5.5±3.2§
PtP,min $cmH_2O \cdot s^{-1} \cdot min^{-1}$	255±63	226±101	117±70*	74±43§
PtP,min/V'E $cmH_2O \cdot s^{-1} \cdot min^{-1}$	32±12	28±12	11±6*	7±4§
Rawe $cmH_2O \cdot L^{-1} \cdot s^{-1}$	19±8	16±7	6±4*	4±2§

Data are presented as mean±SD (n=12). ANOVA test: *: $p<0.05$ *versus* BMI <29 in sitting SB; §: $p<0.05$ *versus* BMI ≥29 in sitting SB; no significant differences between BMI <29 and BMI ≥29 in the same condition. PEEPI,dyn: intrinsic dynamic positive end-expiratory pressure; $P_{oes,swing}$: oesophageal pressure swing; PtP,min: pressure time produce of the inspiratory muscles per minute; PtP,min/V'E: pressure time produce per minute corrected for ventilation; Rawe: expiratory airway resistance.

the well reported unloading effect of NPSV. In sitting position P_{oes} swing, PtP,min and PtP,min/V'E decreased by 46%, 39% and 34% respectively during NPSV. The main finding of the study is that neither supine nor lateral postures induced significant variations in the unloading effect of NPSV observed in the sitting posture. In the above quoted study by DRUZ and SHARP [5] in spontaneously breathing COPD patients the upright sitting position induced a 28% reduction in the neuromechanical efficiency of the diaphragm (as assessed by the diaphragmatic electromyography transdiaphragmatic pressure ratio) in comparison to supine value. Therefore, although the current study measured respiratory muscle function during SB only in sitting position, the study shows that NPSV is able to abolish posture induced differences in respiratory muscles function. Furthermore, contrary to what has been reported for SB, in this study changing posture did not influence breathing pattern during NPSV [22, 23]. The lack of differences in the unloading effect among different postures was also confirmed in four anecdotal patients in whom transdiaphragmatic pressure was also measured. As a whole the results show no differences in breathing pattern and inspiratory muscle effort in the three different postures adopted during NPSV.

In the study an external PEEP of 4 cmH_2O was applied during NPSV with the rationale that it may counterbalance the PEEPI,dyn, *i.e.* the inspiratory threshold load which has been observed in stable COPD patients [29]. Indeed NAVA *et al.* [11] found that the application of 5 cmH_2O PEEP during both 10 and 20 cmH_2O pressure support ventilation increased the unloading effect of NPSV on the inspiratory muscles in stable COPD patients. In accordance with the lack of change in breathing pattern and inspiratory muscle function, no differences in dyspnoea were found when changing posture during NPSV. Although the patients in the current study were awake, at least theoretically the application of 4 cmH_2O of PEEP during NPSV might have counterbalanced the impairment of upper airways tone considered as a responsible factor (among others) for the increase in respiratory resistances.

In a recent study PANKOW *et al.* [30] observed that application of NPSV can unload the inspiratory muscle in obese subjects. In that study, the authors found a significant 40% decrease in inspiratory muscle activity during NPSV in comparison to unsupported breathing. In the obese patients (BMI ≥29 $kg \cdot m^{-2}$) of the current study these observations were confirmed [30], but the authors were not able to find any difference in breathing pattern and respiratory mechanics when compared to the nonobese patients. However, obese patients in the current study showed a mean BMI lower than the patients of the PANKOW *et al.* study [30]. The lack of differences between these two groups may be explained by the inclusion of patients with moderate obesity and slightly better spirometry values than nonobese patients.

Limitations of the study

The small number of evaluated patients may reduce the power of statistical analysis to detect small differences between positions.

This study did not compare baseline and NPSV arterial blood gases in different positions since it was beyond the aims of the study. DIAZ *et al.* [31] have shown that in COPD patients submitted to NPPV due to acute respiratory failure (ARF) the improvement in arterial blood gases was essentially due to attainment of an efficient breathing pattern rather than to an improvement in the ventilation/perfusion ratio. Therefore because in the current study the breathing pattern under NPSV did not change with different postures and the comparison of arterial blood gases

Table 5. – Respiratory muscles effort and airway resistance during noninvasive pressure support ventilation (NPSV) in different postures according to body mass index (BMI)

	Sitting		Supine		Lateral	
	BMI <29	BMI ≥29	BMI <29	BMI ≥29	BMI <29	BMI ≥29
PEEPI,dyn cmH_2O	1.2±1.3	1.55±1.5	1.6±1.3	1.9±1.7	1.5±1.6	2±1.9
$P_{oes,swing}$ cmH_2O	7±3.8	5.5±3.2	7±3	7.7±2.2	7.4±3.7	5.5±3.4
PtP,min $cmH_2O \cdot s^{-1} \cdot min^{-1}$	117±70	74±43	117±18	98±56	111±73	91±68
PtP,min/V'E $cmH_2O \cdot s^{-1} \cdot min^{-1}$	11±6	7±4	9±5	9±6	10±7	7±5
Rawe $cmH_2O \cdot L^{-1} \cdot s^{-1}$	6±4	4±2	7±3	7.7±2.2	7.4±3.7	5.5±3.4

Data are presented as mean±SD (n=12). ANOVA test: no significant differences between BMI <29 and BMI ≥29 in different postures. PEEPI,dyn: intrinsic dynamic positive end-expiratory pressure; $P_{oes,swing}$: oesophageal pressure swing; PtP,min: pressure time produce of the inspiratory muscles per minute; PtP,min/V'E: pressure time produce per minute corrected for ventilation; Rawe: expiratory airway resistance.

assessed under NPSV in the three postures did not show any significant differences it can be speculated that postures per se did not influence arterial blood gases under NPSV.

These results must be considered with caution. Indeed results obtained during a diurnal application of NPSV in awake subjects may not apply during sleep [32]. Discrepancies between wakefulness and sleep may be induced by large variations in upper airway patency and by a significantly different pattern of respiratory muscle recruitment, particularly during rapid eye movement (REM) sleep. Furthermore during REM sleep FRC may be amplified because of changes in muscle tone. Supine posture may increase loading on the upper airway and this may be relevant during sleep. The hypothesis of a similar pattern of respiratory mechanics supine or seated during sleep should be verified by further studies.

In conclusion changing posture does not significantly influence the effect of noninvasive pressure support ventilation on breathing pattern and respiratory muscle activity in awake stable hypercapnic patients independently of body weight.

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