Effects of body position, hyperinflation, and blood gas tensions on maximal respiratory pressures in patients with chronic obstructive pulmonary disease

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Abstract

Background – Inspiratory muscle strength in patients with chronic obstructive pulmonary disease (COPD) can be affected by mechanical factors which influence the length of the diaphragm, and by non-mechanical factors. The aim of the present study was to evaluate firstly the effects of body position on respiratory pressures and, secondly, to determine the relative contribution of age, body mass index (BMI), lung volumes, and arterial blood gas tensions to respiratory muscle strength.

Methods – Thirty male patients with stable COPD (mean FEV_1 40·4% predicted) participated in the study. Maximal inspiratory and expiratory mouth pressures (PImax, PEmax) and maximal inspiratory transdiaphragmatic pressures (PDI) in the sitting and supine position, lung function, and arterial blood gas tensions were measured.

Results – Mean (SD) PImax in the sitting position was higher than in the supine position (7·1 (2·3) kPa *v* 6·4 (2·2) kPa respectively). In contrast, PDI in the sitting position was lower than in the supine position (10·0 (3·5) kPa *v* 10·8 (3·7) kPa respectively). PEmax was higher in the sitting position (9·3 (3·0) kPa) than in the supine position (8·7 (2·8) kPa). Significant correlations were found between inspiratory muscle strength on the one hand, and lung function parameters, BMI, and arterial blood gas tensions on the other.

Conclusions – Inspiratory muscle strength in patients with COPD is influenced by mechanical factors (body position, lung volumes) and non-mechanical factors (BMI, FEV_1, and blood gases). PImax and PEmax are lower in the supine position while, in contrast to healthy subjects, PDI is higher in the supine position than in the sitting position.

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Maximal inspiratory pressures (PImax) in patients with chronic obstructive pulmonary disease (COPD) are frequently reported to be lower than in normal adults.[1–6] In some studies, however, taking hyperinflation into account, PImax was similar or even increased in comparison with normal subjects.[7–10] Pressure generation by the diaphragm is influenced by the length of the diaphragm.[11 12] Theoretically it is possible to influence the length of the diaphragm in patients with COPD by changing position from sitting to supine. The flattened diaphragm will then be displaced upwards by the abdominal contents, and will achieve a more advantageous position on the length-tension curve. Higher transdiaphragmatic pressures (PDI) would therefore be expected in the supine position. This would be in contrast to healthy subjects in whom PDI is lower in the supine position.[13]

Besides mechanical factors, non-mechanical factors such as age,[13] height,[3] weight,[3 14] sustained overload,[15] hypoxaemia,[16] and hypercapnia,[17] may also influence respiratory pressures. In most studies the influences of these factors have been analysed separately. The purpose of the present study was, firstly, to evaluate the effects of body position on respiratory pressures and, secondly, to determine the relative contribution of mechanical and non-mechanical factors to respiratory muscle strength in stable patients with COPD.

Methods

STUDY DESIGN
Respiratory muscle strength measurements, lung function tests, and blood gas analyses were performed in 30 patients with COPD. Informed consent was obtained from all subjects. The study was approved by the hospital ethical committee.

PATIENTS
Thirty male patients with COPD, according to the criteria of the American Thoracic Society,[18] participated in the study. Age, body measures, and lung function data are shown in table 1. The patients were in a stable condition, defined as no change in FEV_1 during the preceding three months. Patients with other pulmonary diseases, a previous thoracotomy, diabetes mellitus, and neuromuscular disorders were excluded.

MEASUREMENTS
Pressure measurements
Maximal inspiratory and expiratory mouth pressures (PImax, PEmax) were measured with a device based on that used by Black and

Table 1 Patient characteristics, lung function data and blood gas values (n = 30)

	Mean (SD)
Age (years)	60·7 (6·5)
Height (cm)	174·8 (6·9)
Weight (kg)	74·0 (10·8)
BMI (kg/m²)	24·2 (2·8)
FEV_1 (%pred)	40·4 (19·0)
FEV_1 (l)	1·3 (0·7)
FIV_1 (l)	3·1 (0·8)
FRC (%pred)	132·4 (29·3)
RV (%pred)	146·7 (37·1)
TLC (%pred)	107·7 (16·1)
RV %TLC (%)	47·4 (9·1)
FRC %TLC (%)	63·3 (8·4)
Kco (%pred)	57·5 (29·1)
Sao_2 (%)	94·0 (2·3)
Pao_2 (kPa)	9·4 (1·1)
$Paco_2$ (kPa)	5·7 (0·7)

BMI = body mass index; FEV_1 = forced expiratory volume in one second; FIV_1 = forced inspiratory volume in one second; FRC = functional residual capacity; RV = residual volume; TLC = total lung capacity; Kco = diffusion capacity; Sao_2 = arterial oxygen saturation; Pao_2 = partial arterial oxygen pressure; $Paco_2$ = partial arterial carbon dioxide tension.

Hyatt.[1] A metal tube connected a plastic flanged mouthpiece to a wet spirometer (Pulmotest Godart, Bilthoven, The Netherlands). An occluding balloon within the tube could be inflated by the subjects themselves by squeezing a second external balloon. A small leak (internal diameter 1·1 mm, length 40 mm) in the mouthpiece prevented buccal muscles from producing significant pressures and closing the glottis. The pressure inside the mouthpiece was measured with a pressure transducer (Validyne DP103-32, Northridge, California, USA).

Transdiaphragmatic pressure (PDI) was measured with a flexible double lumen catheter (internal diameter of each lumen 1·1 mm)[19 20] inserted through the nose. The catheter was positioned with the distal opening of the gastric lumen 58 cm from the nares and the proximal opening of the oesophageal lumen 38 cm from the nares. The catheter was perfused with water at a constant flow of 99 ml/hour. The pressure generated by the water flow was 1·2 kPa. The proximal ends of the double lumen catheter were connected with two pressure transducers (Validyne DP15-32, range ± 40 kPa). The zero reference point was arbitrarily set as the pressure measured at functional residual capacity (FRC). PDI was calculated by subtracting oesophageal pressure (POES) from gastric pressure (PGA). PImax, PEmax, POES, and PGA were displayed on a chart recorder (Kipp en Zonen BD 101, Delft, The Netherlands). Pressure measurements were performed in the sitting and supine position, with the subjects wearing a noseclip. In either position the subjects maintained an identical posture and had appropriate support for their arms throughout the experiment.

Before each static inspiratory effort subjects inhaled to total lung capacity (TLC) and then exhaled to residual volume (RV), with visual feedback on the spirometer. They then closed the tube by inflating the balloon and performed a maximal inspiratory effort. Maximal inspiratory efforts were maintained for two seconds separated by at least 60 seconds between efforts.

PEmax was measured after inhaling to TLC, followed by inflating the balloon and perform-ing a maximal static expiratory effort. The inspiratory manoeuvre at RV and the expiratory manoeuvre at TLC were repeated until three reproducible measurements had been made with a maximal variability of 10%.[21] The highest value was used for analysis. For the sake of convenience, PImax and POES were expressed as positive values. At each measurement calibrations were made with a water manometer.

Predicted values for respiratory muscle strength were calculated according to Wilson et al[3] because these authors also used a flanged mouthpiece.

Lung function

Spirometric tests were performed with a wet spirometer and by a closed circuit helium dilution method (Pulmonet III, Sensormedics, Bilthoven, The Netherlands). Diffusion capacity (Kco) was measured by the single breath carbon monoxide method (Sensormedics 2450). Predicted spirometric values were derived from ERS standards.[22]

Blood gas values

Arterial blood samples were taken in the semirecumbent position at 09.00 hours after the patients had been lying down for at least 15 minutes. The samples were measured with a Corning Ph 127 blood gas analyser.

DATA ANALYSIS

Data are presented as mean (SD). To analyse the data statistically we performed Spearman correlation tests and Wilcoxon signed rank tests. Stepwise multiple regression analysis was used to assess the relative contributions of age, body mass index (BMI), FRC %TLC, FEV_1 (%pred), Sao_2, and $Paco_2$ to PImax, PDI, and PEmax. For all analyses the SAS package (SAS Institute Inc, Cary, North Carolina, USA) was used. p values < 0·05 were considered significant.

Results

PATIENTS

Thirty male patients with COPD were included in the study. There was a wide variation of airway obstruction (FEV_1 18–87% predicted) and hyperinflation was modest (mean FRC: 132 (29)% predicted). Four patients were hypoxaemic (Pao_2 < 8 kPa) and one patient was hypercapnic ($Paco_2$ > 6·5 kPa).

RESPIRATORY MUSCLE FORCE AND EFFECT OF BODY POSITION

Sitting PImax and PEmax were lower than predicted, PImax being 89·1 (28·1)% predicted (7·1 (2·3) kPa) and PEmax 74·1 (23·4)% predicted (9·3 (3·0) kPa). The low value for PEmax could be caused by the closing of the glottis when performing a PEmax manoeuvre, so PEmax was also measured at the oesophageal level. At this level the value was significantly
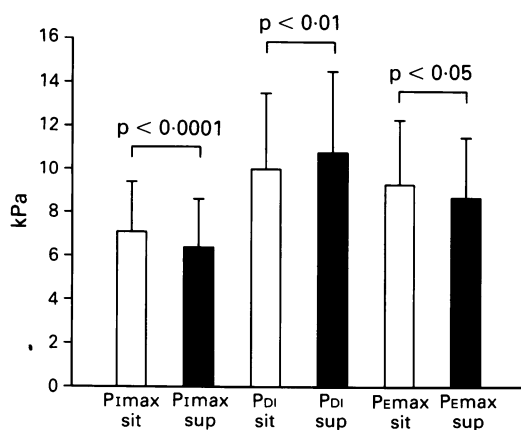
Figure 1 Maximal inspiratory mouth pressure (PImax), maximal inspiratory transdiaphragmatic pressure (PDI) and maximal expiratory mouth pressure (PEmax) in sitting (sit) and supine (sup) positions.
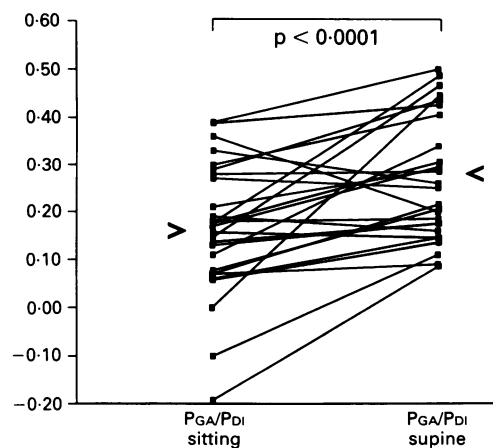

Figure 2 PGA/PDI in sitting and supine positions (> and < indicate mean values).

higher than at the mouth (10·8 (2·9) kPa v 9·3 (3·0) kPa (p < 0·01)). The lung volume (expressed as percentage of the predicted TLC) at which maximal inspiratory pressures in the sitting position were measured was 60·8 (14·1)%. The supine maximal inspiratory pressures were measured at a significantly higher lung volume (64·4 (15·3)% of TLC predicted (p < 0·001)). This happened despite patients being asked to expire to RV in both positions. In the supine position the same end expiratory level was not reached by 24 of the 30 patients in comparison with the sitting position.

PDI could not be measured in three patients because of their inability to swallow the double lumen catheter. The mean value in the remaining 27 patients was 10·0 (3·5) kPa.

When changing position from sitting to supine PImax decreased by 0·7 (0·6) kPa (p < 0·0001) and PEmax decreased by 0·6 (1·2) kPa (p < 0·05). In contrast, PDI increased by 0·9 (1·5) kPa (p < 0·01) (fig 1). To determine whether the diaphragm is a better pressure generator in the supine position the two components of PDI (PGA and POES) were studied. They showed different responses to the change in body position. PGA increased from 1·8 (1·4) kPa in the sitting position to 3·0 (2·1) kPa in the supine position (p < 0·0001). POES decreased from 8·1 (2·8) kPa in the sitting position to 7·8 (2·6) kPa in the supine position, but this change did not reach significance. The changes in PGA and POES in relation to PDI are shown in figs 2 and 3. The mean differences between the sitting and supine ratios PGA/PDI and POES/PDI were 0·11 (0·13) (p < 0·0001) and −0·11 (0·13) (p < 0·0001), respectively. In the sitting position the ratio PImax/POES was 0·07 (0·11) (p < 0·01) higher than in the supine position (fig 4).

FACTORS INFLUENCING RESPIRATORY MUSCLE FORCE

Spearman correlation coefficients between maximal respiratory pressures on the one hand and age, BMI, lung function parameters, and blood gas values on the other are shown in

table 2. The highest correlation coefficients were found between maximal inspiratory pressures and FRC %TLC (%) and arterial oxygen saturation (tension). PEmax was not correlated with any of these parameters, and was only significantly correlated with PImax and PDI; both correlation coefficients were 0·60 (p < 0·001).
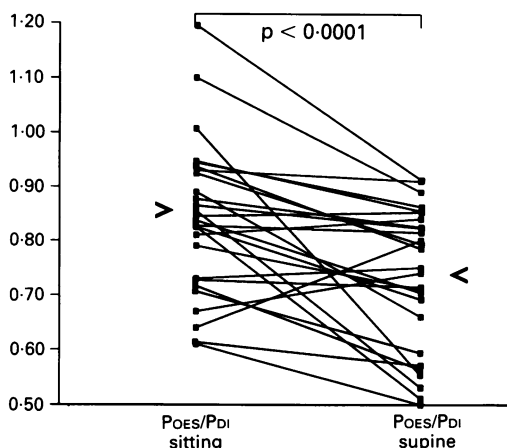

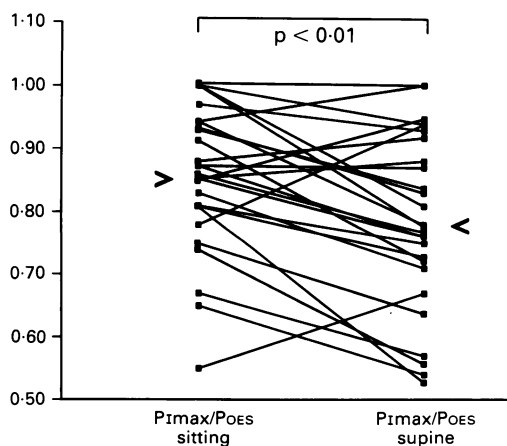*Figure 3 POES/PDI in sitting and supine positions (> and < indicate mean values).*

Figure 4 PImax/POES in sitting and supine positions (> and < indicate mean values).

Table 2 Spearman correlation coefficients between maximal inspiratory (PImax) and expiratory (PEmax) pressures and maximal inspiratory transdiaphragmatic pressure (PDI) and age, BMI, lung function parameters, and blood gas values

	PImax (kPa)	PImax (%pred)	PDI (kPa)	PEmax (kPa)	PEmax (%pred)
Age (years)	−0·32	0·05	−0·36	−0·35	−0·21
BMI (kg/m²)	0·30	0·39*	0·34	0·06	0·06
FRC %TLC (%)	−0·59***	−0·61***	−0·54**	−0·09	−0·05
RV %TLC (%)	−0·48**	−0·44**	−0·45*	−0·05	0·00
FEV₁ (%pred)	0·49**	0·60***	0·43*	0·08	0·08
FIV₁ (l)	0·48**	0·51**	0·48*	0·25	0·23
Sao₂ (%)	0·62***	0·55**	0·64***	0·27	0·19
Pao₂ (kPa)	0·58***	0·51**	0·67***	0·30	0·24
Paco₂ (kPa)	−0·41*	−0·27	−0·30	−0·25	−0·15

For definition of abbreviations see footnote to table 1.
* p < 0·05; ** p < 0·01; *** p < 0·001.

Table 3 Stepwise multiple regression analyses, r², and p values to predict maximal inspiratory (Pimax), expiratory (PEmax), and transdiaphragmatic (PDI) pressures

	Step	Variable	r²	p
PImax (kPa)	1	FRC %TLC	0·34	0·001
	2	Sao₂	0·43	0·004
PImax (%pred)	1	FRC %TLC	0·30	0·002
PDI (kPa)	1	Sao₂	0·33	0·002
PEmax	1	Age	0·14	0·004
PEmax (%pred)	–	–	–	–

For definition of abbreviations see footnote to table 1.

Stepwise multiple regression analysis showed that PImax (kPa) could be predicted from a combination of Sao₂ and FRC %TLC:

PImax (kPa) = − 17·8 − 0·12 Sao₂ + 0·34 FRC %TLC (r² = 0·44).

PDI (kPa) could be predicted only by Sao₂:

PDI = − 70·2 + 0·85 Sao₂ (r² = 0·33).

PEmax (kPa) was predicted by age:

PEmax = 20·1 − 0·18 age (r² = 0·14).

Table 3 shows the complete data with values of p and r².

Discussion

The purpose of this study was to evaluate the effects of body position on respiratory pressures and to determine the relative contribution of mechanical and non-mechanical factors to respiratory muscle strength in patients with COPD. Firstly, we found that body position influences respiratory pressures. PImax and PEmax were higher in the sitting position than in the supine position, while PDI, in contrast to healthy subjects, was higher in the supine position than in the sitting position (fig 1). Secondly, mechanical and non-mechanical factors indeed contributed to respiratory muscle strength. This was indicated by significant correlations between inspiratory muscle strength and static lung volumes (mechanical factors), and between inspiratory muscle strength and BMI, FEV₁, Sao₂, and Paco₂ (non-mechanical factors).

The diaphragm appeared to be a better pressure generator in the supine position in these patients with COPD. In this position higher PDI values were caused by an increase in the ratio PGA/PDI combined with a decrease in the ratio POES/PDI (figs 2 and 3). The difference between the ratio PImax/POES in the sitting and supine positions was − 0·07 (fig 4),

since PImax decreased more than POES in the supine position. The reason why POES did not decrease significantly in contrast to PImax might be the elastic recoil pressure of the lungs which influences PImax but not POES. The supine inspiratory pressures were measured at 64·4 (15·3)% of TLC predicted, while the sitting inspiratory pressures were measured at 60·8 (14·1)% of TLC predicted, although the patients were asked to expire as far as possible before they performed a maximal inspiratory manoeuvre in both positions. At higher lung volumes the elastic recoil pressure of the lungs increases, and thus the difference between inspiratory mouth pressures and inspiratory oesophageal pressures will increase.[6] This is confirmed by the findings in the present study: the mean difference between PImax and POES in the sitting position was 1·0 kPa, while the mean difference in the supine position was 1·4 kPa.

In healthy subjects both PImax and PDI were lower in the supine position.[13] The effect of postural changes on maximal respiratory pressures in patients with COPD has not previously been described. Swings in PDI during tidal breathing in the supine position were significantly higher than in the sitting position.[23] This is in line with the findings in the present study. An explanation might be that in the supine position the abdominal contents displace the flattened diaphragm upwards leading to a more favourable position on the length-tension curve. However, the increased force generated by the diaphragm apparently cannot compensate for the decreased phasic and tonic activity of the scalene, sternocleidomastoid, and parasternal-intercostal muscles and the decreased compliance of the rib cage in the supine position.[24 25] This may explain why in our patients PImax in the supine position was significantly lower than PImax in the sitting position.

PImax values were lower than predicted (89·1 (28·1)%), but these pressures were measured at 60·8 (14·1)% predicted TLC. Rochester[26] suggested that the observed values of PImax in patients with COPD should be compared with the values that normal subjects would achieve at similar lung volumes. For normal subjects we expected 80–90% of the predicted PImax at 60% TLC.[6 27] This means that PImax, after correction for lung volume, was not lower than in normal subjects as was suggested in previous studies.[7-10] Also, in a postmortem study diaphragm dimensions of patients with and without COPD did not differ significantly.[28] PImax values in our patients (7·1 (2·3) kPa) were within the same range as PImax values in patients with COPD described in previous studies (7·1 (2·4) kPa,[10] 5·6 (2·5) kPa,[4] 6·0 (1·9) kPa,[5] and 8·0 (2·7) kPa[29]). These different results may be explained by differences in patient characteristics. The relation between PImax and FEV₁ (% predicted) in these studies seems linear (fig 5).

For PDI no predicted values are available. Mean PDI in normal subjects varied from 9·1 kPa to 15·0 kPa and in patients with COPD from 3·7 kPa to 9·8 kPa.[21] We measured a mean
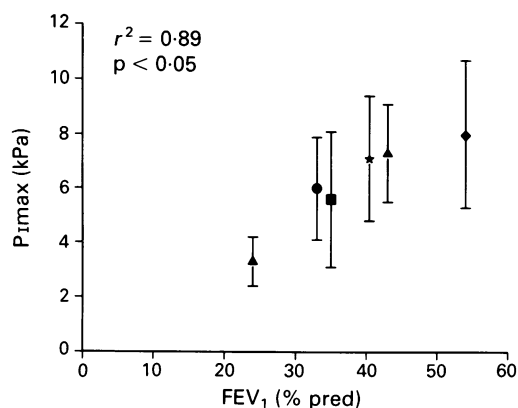
Figure 5   Correlation between PImax and FEV₁ in different studies. ■, ref 4; ▲, ref 7; ●, ref 5; ◆, ref 29; ★, present study.

PDI of 10·0 (3·5) kPa. The observed difference between healthy subjects and patients with COPD may be explained by a different position on the length-tension curve due to changes in lung volumes.[12 30-32] This may explain the correlation between maximal inspiratory pressures and FRC and RV as percentages of TLC in the present study.

In previous studies the variability of PEmax in patients with COPD was larger than that of PImax (20·6 (4·1) kPa,[10] 13·7 (4·8) kPa,[4] 13·8 (6·5) kPa,[5] and 10·0 kPa[6]), whereas in our study PEmax was 9·3 (3·0) kPa. PEmax in the present study was rather low in comparison with the other studies. An explanation might be the closing of the glottis by some patients when performing a PEmax manoeuvre because PEmax measured in the oesophagus was significantly higher than PEmax measured in the mouth (10·8 (2·9) kPa *v* 9·3 (3·0) kPa, respectively (p<0·01)). Decramer *et al*[6] suggested that minor variations in the technique contributed to high variations in PEmax values. Another explanation for the low PEmax values in the present study could be generalised muscle weakness. This was probably not the case, since PImax (% predicted) was less decreased than PEmax (% predicted). PImax is influenced by hyperinflation and generalised muscle weakness, while PEmax is influenced by generalised muscle weakness alone.[4] Moreover, malnutrition may be responsible for lower PEmax values,[33] but this was unlikely since BMI was within the normal range. In view of these considerations it is most likely that the low PEmax values in the present study were due to the technique used.

Besides mechanical factors (body position, static lung volumes), non-mechanical factors may influence respiratory muscle strength. Significant correlation coefficients were found between inspiratory pressures and BMI, FEV₁, SaO_2, and $PaCO_2$ (table 2).

The effect of BMI on inspiratory pressures is known. Undernourished patients with COPD have lower inspiratory muscle strength than well nourished patients.[33]

The relation between FEV₁ and PImax might suggest that the impaired muscle function results in lower FEV₁ values. It is also possible, however, that a decrease in FEV₁

contributes to the impairment of inspiratory muscle function because of a sustained overload.[15] Indeed, the diameters of the type I and type II fibres of the diaphragm in normal subjects were significantly higher than in patients with COPD.[34] Moreover, there was a linear relation between FEV₁ and diaphragmatic fibre diameters in these patients.

Little is known about the consequences of chronic hypoxaemia on respiratory muscles, as is present in patients with chronic respiratory failure, or about the influence of respiratory muscle dysfunction on PaO_2. It has been suggested that impairment of the respiratory muscles causes microatelectasis resulting in a decrease of PaO_2.[35 36] The consequences of acute hypoxaemia on respiratory muscles in normal persons have been investigated. One study showed that the respiratory muscles failed more quickly during low oxygen breathing,[16] but this was not confirmed in a later study.[37]

A negative correlation between PImax and $PaCO_2$ has been found in previous studies.[4 17 38] Rochester *et al*[4] showed a correlation of −0·66 in 18 patients with COPD with PImax <5·5 kPa. Braun *et al*[38] studied the correlation between PImax and $PaCO_2$ in patients with proximal myopathies and found a correlation coefficient of −0·59 when respiratory muscle strength was less than 50% predicted. Diaphragmatic contractility was reduced when end tidal FCO_2 was raised above 7·5%.[17]

Multiple regression analysis was used to determine the relative contribution of influencing factors such as age, BMI, blood gas tensions, and lung function to respiratory muscle strength (table 3). Only low predictive values were obtained. This suggests that other individual features such as elastic recoil pressure of the respiratory system and physical fitness also influence maximal respiratory pressures.

In conclusion, this study shows that respiratory pressures in patients with COPD are influenced by body position. PImax and PEmax are lower in the supine position while, in contrast to healthy subjects, PDI is higher in the supine position than in the sitting position. In addition, significant correlations have been found between maximal respiratory pressures and mechanical and non-mechanical factors, although the value of these factors in predicting maximal respiratory pressures is low.

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