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Nocturnal saturation and respiratory muscle function in patients with chronic obstructive pulmonary disease

Y F Heijdra, P N R Dekhuijzen, C L A van Herwaarden, H Th M Folgering

Abstract

Background — Nocturnal desaturations, mainly caused by hypoventilation, occur frequently in patients with chronic obstructive pulmonary disease (COPD). Daytime arterial oxygen and carbon dioxide tensions (PaO₂ and PaCO₂) appear to predict which patients will desaturate at night. It is unknown if respiratory muscle strength, which may be decreased in these patients, plays an additional part.

Methods — Polysomnography, maximal respiratory pressures, lung function, and arterial blood gas tensions were measured in 34 patients with COPD (mean (SD) forced expiratory volume in one second (FEV₁) 41·7 (19·9)% pred). Nocturnal saturation was investigated.

Results — Significant correlations were found between the mean nocturnal arterial oxygen saturation and maximal inspiratory mouth pressure (r = 0·65), maximal inspiratory transdiaphragmatic pressure (r = 0·55), FEV₁ (r = 0·61), transfer coefficient (KCO) (r = 0·38), arterial oxygen saturation (SaO₂) (r = 0·75), and PaCO₂ (r = −0·44). Multiple regression analysis showed that 75% of the variance in nocturnal SaO₂ was explained by a combination of SaO₂ (70%) and FEV₁ (5%).

Conclusion — Inspiratory muscle strength and nocturnal saturation data are correlated, but daytime SaO₂ and FEV₁ remain the most important predictors of nocturnal saturation.

(Thorax 1995;50:610–612)

Keywords: nocturnal saturation, respiratory muscle strength, chronic obstructive pulmonary disease.
Nocturnal saturation and respiratory muscle function in COPD

Pimax (kPa)

Daytime parameters

FEV₁=forced expiratory volume in one second; Kco—carbon monoxide transfer coefficient.

Pimax=static maximal inspiratory mouth pressure; Kco (% pred)

FEV₁ (% pred)

Sao₂ (arterial oxygen saturation; Paco₂ = arterial carbon dioxide tension; REM=rapid eye movement sleep.

expressed as absolute values. Predicted values for respiratory muscle strength were derived from Wilson et al.13

DATA ANALYSIS

Data are presented as means (SD). Spearman correlation tests were performed, p values of <0.05 being considered significant. Stepwise multiple regression analysis was used to assess which parameters were independent predictors of the nocturnal and daytime saturation. The significance level for retention in the model was 0.05.

Results

The mean age of the patients was 61.4 (6.4) years. They had a wide variation in airways obstruction (FEV₁ 0.6–3.21, mean 41.7 (19.9)% pred). They were hyperinflated (functional residual capacity (FRC) (127.8 (31.2)% pred) and had a low gas transfer coefficient capacity (Kco) of 57.5 (28.5)% pred. Four patients were hypoxaemic (Pao₂ <8.0 kPa) and two patients were hypoxaemic and hypercapnic (Paco₂ >6.5 kPa). The mean Pao₂ and Paco₂ were 9.3 (1.3) kPa and 5.7 (0.7) kPa, respectively.

All patients had at least one period of REM sleep and, in the whole group, 10-8 (4-6)% of the recording time was spent in REM sleep. Of the 34 patients 16 developed episodes with desaturations during the night. In these 16 patients the mean desaturation time and the mean REM sleep time were 40-6 (27-7)% and 11-6 (4-0)% of the total recording time, respectively. In these patients 64-0 (35-2)% of the total REM sleep time was spent desaturated, which represents 18.2% of the total desaturation time. The polysomnographic data are shown in table 1.

Pimax, Pdi, and Pimax were 6.9 (2.3) kPa (87-5 (27)% pred), 9.7 (3-6) kPa, and 8.9 (3-0) kPa (73-1 (22-2)% pred), respectively. In four patients Pdi was not measured because of inability to swallow the oesophageal catheter.

The correlation coefficients between nocturnal saturation data and daytime characteristics are presented in table 2. The highest correlation coefficient was found between daytime and nocturnal saturation.

The correlation between Pimax (% pred) and mean nocturnal arterial oxygen saturation (Sao₂) (%) is shown in the figure. There was a large overlap between patients who did and did not desaturate.

Stepwise multiple regression analysis was used to evaluate the contribution of various parameters in the prediction of the mean nocturnal saturation. The input variables were the daytime parameters shown in table 2. Of the variance in mean nocturnal Sao₂, 75% was explained by a combination of daytime Sao₂ (70.7)% and FEV₁ (5%). A similar analysis was performed to predict daytime Sao₂ with the variables Pimax (kPa), Pdi (kPa), FEV₁ (% pred), FRC (% pred), and Kco (% pred). Pdi was the only predictive variable (r²=0.33).

Discussion

This study shows that maximal inspiratory muscle strength and nocturnal saturation data are significantly correlated in patients with COPD. However, daytime Sao₂ and FEV₁ remain the most important predictors of nocturnal saturation.

An important case of hypoventilation during REM sleep is diminished respiratory activity of the intercostal and accessory muscles14 which increases the workload of the diaphragm. However, strength and endurance of the diaphragm in patients with COPD may be affected by their unfavourable position on the length-tension curve due to hyperinflation.1 It was therefore hypothesised that a relation may exist between nocturnal arterial oxygen saturation and maximal inspiratory muscle strength, and a sig-

<table>
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<th>Table 1 Mean (SD) nocturnal measurements</th>
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<tr>
<td>Baseline Sao₂ awake (%)</td>
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<td>Mean nocturnal Sao₂ (%)</td>
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<td>% time desaturated (%)</td>
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<tr>
<td>Lowest nocturnal Sao₂ (%)</td>
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<tr>
<td>Baseline PTVCO₂ awake (kPa)</td>
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<tr>
<td>PTVCO₂, asleep (kPa)</td>
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<td>Time in bed (min)</td>
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<tr>
<td>Time in non-REM (min)</td>
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<td>Time in REM (min)</td>
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Sao₂ = arterial oxygen saturation; PTVCO₂ = end tidal carbon dioxide tension; REM = rapid eye movement sleep.

<table>
<thead>
<tr>
<th>Table 2 Spearman’s correlation coefficients between daytime characteristics and nocturnal saturation data</th>
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<tr>
<td>Daytime parameters</td>
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<tr>
<td>Pimax (kPa)</td>
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<tr>
<td>Pdi (kPa)</td>
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<tr>
<td>Sao₂ (%)</td>
</tr>
<tr>
<td>Paco₂ (kPa)</td>
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<tr>
<td>FEV₁ (%pred)</td>
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<td>Kco (% pred)</td>
</tr>
</tbody>
</table>

Pimax=static maximal inspiratory mouth pressure; Pdi=static maximal inspiratory trans-diaphragmatic pressure; Sao₂=arterial oxygen saturation; Paco₂=arterial carbon dioxide tension; FEV₁=forced expiratory volume in one second; Kco=carbon monoxide transfer coefficient. *p<0.05; **p<0.01; ***p<0.001.

Relation between maximal inspiratory mouth pressure (Pimax) (% pred) and mean nocturnal arterial oxygen saturation (%) 0.63, p<0.001.
sific correlation was, indeed, shown between these two parameters (figure). How-
never, if patients were divided into those who desaturated and those who did not, a con-
siderable overlap was seen between the two groups. Pimax and Pdi appear to have a low pre-
dictive value and this was confirmed by multiple regression analysis. Daytime Sao2 and
FEV1 were the only independent predictors and explained 75% of the variance in the mean
nocturnal saturation. However, Pdi was the only predictive variable for daytime Sao2 so
an indirect effect on nocturnal saturation via
daytime Sao2 is also possible. The finding that
FEV1 was one of the independent predictors,
in contrast to other studies,1516 may be ex-
plained by the wide range of FEV1 values
(0.6–3.2 l) in our patients.

The significance of daytime Sao2 in pre-
dicting the nocturnal saturation has been de-
scribed previously. Bradley et al16 showed that
daytime Sao2 and Paco2 accounted for 68% of the variability of the nocturnal saturation in
patients with COPD. In another study a high
correlation was found between daytime and
nocturnal Sao2 in 97 patients with COPD.5

The definition of a desaturation as a decrease of more than 4% in Sao2 lasting at least
five minutes was derived from the study of
Block et al7 combined with that of Fletcher et
al8 who defined a nocturnal desaturation as a
fall below 90% lasting at least five minutes or
more. This latter study described desaturations in patients with COPD in whom, in general,
a serious desaturation lasted longer than five
minutes, in contrast to patients with ob-
structive sleep apnoea in whom clinically im-
portant desaturations can last as little as
10 seconds. In addition, desaturations caused
by movement usually last less than five minutes.

The patients in this study spent a long time
awake. This is probably due to the long re-
cording time, defined as the total time patients
spent in bed attached to the polysomnographic
apparatus, because the time spent in REM and
non-REM sleep was comparable to other sleep
studies performed in patients with COPD.5,8,11,16,17 When the total recording time
and the time patients were awake in our study
were compared with the study of Gothe et al17
similar results were found. The total recording
time and the time spent awake were 454 (48)
minutes versus 449 (80) minutes, and 156 (58)
minutes versus 188 (85) minutes in our study
and that of Gothe et al, respectively.

Little is known about the impact of an ex-
perimental situation on sleep stage variability,
Sao2, and breathing pattern in patients with
COPD. Two studies have shown that the mean
and lowest Sao2 and breathing pattern did not differ during two nights.17 18 Based on these
data we presume that the outcome of the pres-
ent study was not influenced by studying
patients for only one night.

In conclusion, this study shows significant
correlations between maximal inspiratory
force and nocturnal saturation data
in patients with COPD. However, 75% of the
variability in mean nocturnal Sao2 was ex-
plained by daytime Sao2 and FEV1.

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