Predictors of mortality in chest wall disease treated with noninvasive home mechanical ventilation

Sergi Marti a,b, Mercedes Pallero a,b,c, * Jaume Ferrer a,b, Jose Rios d,e, Esther Rodriguez a,b, Ferran Morell a,b,c, Xavier Munoz a,b,f

a Respiratory Medicine Department, Hospital Universitari Vall d'Hebron, Barcelona, Spain
b CIBER Enfermedades Respiratorias (CIBERES), Spain
c Departament de Medicina, Universitat Autònoma de Barcelona (UAB), Barcelona, Spain
d Laboratory of Biostatistics & Epidemiology, Universitat Autònoma de Barcelona (UAB), Barcelona, Spain
e Statistics and Methodology Support Unit, IDIBAPS, Hospital Clinic, Barcelona, Spain
f Departament de Biologia Celular, Fisiologia, Immunologia, Facultat de Medicina, Universitat Autònoma de Barcelona (UAB), Barcelona, Spain

Received 10 June 2010; accepted 26 August 2010

KEYWORDS
Predictors of mortality; Chest wall disease; Noninvasive mechanical ventilation; Mortality

Summary
Rationale: The long-term evolution of patients with chest wall disease and chronic respiratory failure treated with noninvasive home mechanical ventilation (NIHMV) is poorly known.
Objectives: The aim of this prospective observational study was to analyze the variables associated with mortality in a cohort of chest wall disease patients with chronic respiratory failure undergoing long-term follow-up after starting treatment with NIHMV.
Methods: Chest wall disease patients who began NIHMV between 1996 and 2005 were followed up, with death as the primary outcome. The patients’ clinical characteristics, lung function, and arterial blood gases were recorded at the start of treatment. Patients were seen and evaluated 1 month after starting NIHMV. The prognostic value of clinical and functional variables were assessed by Cox regression analyses.
Main results: We included 110 patients, 61 with tuberculosis sequelae and 49 with kyphoscoliosis. By the end of follow-up, 34 patients (28%) had died. The 5-year survival was 69% in those with tuberculosis sequelae and 75% in kyphoscoliosis. PaCO2 ≥ 50 mmHg at 1 month of home ventilation and comorbidity (Charlson Index ≥ 3) were independent predictors of mortality.
Conclusion: Our results suggest that PaCO2 levels ≥ 50 mmHg at 1 month after starting noninvasive home mechanical ventilation and the presence of comorbid conditions are risk factors for mortality in patients with chest wall disease. The importance of early detection of suboptimal home ventilation as well as comorbidities is highlighted.

ª 2010 Elsevier Ltd. All rights reserved.
Introduction

Noninvasive home mechanical ventilation (NIHMV) is indicated in chest wall diseases (CWD) developing chronic respiratory failure (CRF). Therefore, in a European epidemiologic survey (Eurovent), CWD accounted for approximately one-third of the indications for home mechanical ventilation.

After implementation of NIHMV in CWD patients with CRF, improvements in hypoventilation symptoms and arterial blood gases (ABG) and a reduction in hospital admissions due to respiratory complications have been shown. Despite these short-term favorable outcomes, the long-term evolution of NIHMV in these patients is poorly known. Survival in patients with CWD receiving NIHMV has been estimated in 2 series. Leger et al. found that nearly 80% of patients continue NIHMV at 3 years. In the study by Simmonds et al., this figure differed slightly depending on the cause of CWD: 94% and 79% of patients with tuberculosis sequelae and kyphoscoliosis, respectively, continued NIHMV at 5 years.

The relationship between clinical and respiratory function variables and mortality in CWD patients undergoing NIHMV remains to be defined. In a 10-year analysis from the ANTADIR Observatory, prognostic factors of mortality were evaluated in patients with tuberculosis sequelae and kyphoscoliosis. However, less than 30% of these patients were receiving NIHMV, and the authors did not perform a separate analysis of this subsample. Taking into account all patients, regardless of the therapy received, female sex, younger age, a high body mass index, and higher PaO2 and PaCO2 values were all favorable independent prognostic factors. Survival of patients with kyphoscoliosis and tuberculosis sequelae was also evaluated in the 2 Swedish studies mentioned above. In both studies, survival was associated with the therapy applied (NIHMV or LTOT) and this fact likely precluded the finding of associations between other independent variables and mortality. This paucity of evidence highlights the need for further studies focused on CWD patients undergoing NIHMV and followed-up on a long-term basis. Knowledge of prognostic factors for mortality has clinical interest, since it may help to improve the management of these patients.

The aim of the present study was to analyze the variables associated with mortality in a cohort of patients with CWD and CRF undergoing long-term follow-up after starting treatment with NIHMV.

Patients and methods

All adult patients (≥18 years) with CWD who started NIHMV in a teaching hospital (Vall d’Hebron Hospital, Barcelona, Spain) were considered for inclusion in this prospective, observational study. Patients were enrolled between January 1996 and December 2005. The indication for NIHMV was based on Spanish and international guidelines, including clinical symptoms (dyspnea, fatigue, orthopnea, or morning headache) and one of the following criteria: 1) stable patients with chronic hypercapnia (PaCO2 >45 mmHg), 2) patients admitted due to acute hypercapnic respiratory failure requiring noninvasive mechanical ventilation, and 3) patients with oxygen saturation <90% during ≥30% of the night.

Patients with hypoventilation due to other respiratory diseases (neuromuscular, chronic obstructive pulmonary disease [COPD], and obesity-hypoventilation syndrome) were not included in the study. Having a tracheostomy for airway access and discontinuation of NIHMV due to noncompliance were exclusion criteria. The flow chart for selecting patients is shown in Fig. 1.

The study protocol was approved by the hospital ethics committee and written informed consent was obtained from all patients.

Variable measurements

Patients’ characteristics were systematically recorded in a computer database. Forced spirometry and static volumes (MasterLab Pro, Jaeger GmbH, Wuerzburg, Germany) were obtained according to the European Respiratory Society guidelines. In stable patients, baseline respiratory function values were defined as the most recent ones obtained prior to initiating NIHMV. In patients who started NIHMV during acute respiratory failure, these values were obtained once the patient had stabilized before hospital discharge.

Samples for ABG testing were taken with the patient breathing room air and processed with a pH and blood gas analyser (IL-1306; Instrumentation Laboratories, Milan, Italy). Comorbid conditions were recorded using the Charlson Index. A score of 1–6 was assigned to each disease, depending on its associated risk of death. In this study, all patients had a minimal score of 1, since CWD was considered a chronic pulmonary disease.

Mechanical ventilation

NIHMV was initiated in the respiratory ward. Custom-made or commercial nasal masks were used. The choice of

![Figure 1 Flow chart of patient enrollment.](image-url)
ventilator type was based on the criteria of the attending physician.18 The ventilator parameters were set, and the efficacy of ventilation was evaluated taking into account the patient’s tolerance, oxygen saturation, ABG while awake at 1 h after starting ventilation, and nocturnal pulse oximetry values.14 Patients were instructed in the management of the ventilator and counseled in its progressive use, at least during the entire night.

Follow-up

All patients were seen in the hospital outpatient clinic 1 month after starting NIHMV, and at intervals of 3–6 months thereafter. At the 1-month visit, room-air ABG were measured. Compliance with the prescription was reported by the patient. A commercial supplier was in charge of providing technical support at the patients’ home.

Patients remained in the study from the start of NIHMV up to closure of follow-up, in December 2007. Closure was earlier in patients who moved out of the study district. These patients were censored, but the information generated up to the time of closure was used in the study. The vital status and cause of death were obtained from the patients’ medical files, relatives, or primary care physicians.

Statistical analysis

Assessment of baseline homogeneity between survivors and deceased patients was performed with the unpaired t-test for continuous variables, the Mann–Whitney test for ordinal variables, and Fisher’s exact test for qualitative variables.

Results are expressed as the mean ± SD for quantitative variables, and as frequencies and percentages for qualitative and ordinal variables. The Kaplan–Meier method was used to estimate the survival functions and the Cox proportional hazards model was used to perform the adjusted analysis.

The selection of independent variables for multivariate Cox model was based on statistical significance obtained in the univariate analysis (p < 0.10). A forward stepwise procedure was used and results are expressed as hazard ratios with 95% confidence intervals (CI).

Analyses were performed with SPSS for Windows, 15.0 (Statistical Package for the Social Sciences, Chicago, IL, USA). Statistical significance for all tests was set at a two-tailed p-value of ≤0.05.

Results

Clinical characteristics

Among 115 CWD patients starting NIHMV during 1996–2005, 110 were included in the study (Fig. 1). Characteristics of the patients as a group and according to their vital status at follow-up closure are shown in Table 1. At baseline, the study cohort included 58% men with a mean age of ~66 years. Spirometry showed a severe restrictive ventilatory impairment with associated obstruction (FEV1/FVC <0.7) in 52 patients (47.3%). Obstruction was more prevalent in the group with tuberculosis sequelae (60.7%) than in those with kyphoscoliosis (30.6%) (p = 0.002). Over 75% of participants had at least one comorbid condition in addition to respiratory disease (i.e., Charlson Index ≥2).

Noninvasive home mechanical ventilation was started during an admission for respiratory exacerbation in 17 patients (15.5%). Among the stable patients, NIHMV was indicated with PaCO2 <45 mmHg in 6 cases (5.5%). Ninety-three patients (84.5%) were treated with volume-cycled ventilators and 17 (15.5%) with pressure-cycled ventilators. Assist-control mode was used in 59 patients (53.6%), and control mode in the remaining 51 (46.4%). In order to overcome oral leaks, a chin strap was prescribed in 4 patients (3.6%). Twenty-four patients (21.8%) needed oxygen coupled to the ventilator. Baseline PaO2 and PaCO2 indicated hypercapnic respiratory failure (PaO2 55.6 ± 11.2 and PaCO2 56.4 ± 9.0 mmHg) (Table 1). During mechanical ventilation, PaCO2 returned to normal levels 1 h after starting therapy (41.3 ± 7.6 mmHg); 1 month later, with the patient using home ventilation, diurnal PaCO2 was maintained at 46.4 ± 4.9 mmHg while breathing room air.

Follow-up and mortality

Patients were followed-up for a median of 4.6 years (interquartile range, 2.4–6.0). In 2 survivors who moved to a different region, follow-up ended before December 2007, at 7 and 23 months after starting NIHMV, respectively. Another patient presented acute respiratory failure 9 years after initiating NIHMV via nasal mask, and ventilation was continued via tracheostomy. No patients were lost to follow-up. The mean 1-month patient-reported compliance with therapy was 9.3 ± 1.9 h/day (9.6 ± 1.8 in survivors versus 8.8 ± 2.0 in non-survivors p = 0.055).

By the end of follow-up, 34 of 110 patients had died (28.1%). Deaths were mainly due to respiratory causes (64.7%) (Table 2). In the univariate analysis, patients who died had higher comorbidity, total lung capacity, residual volume, and PaCO2 at 1 month, and lower FEV1, FEV1/FVC ratio, and PaO2 at 1 month (Table 1). There were no differences in ventilation characteristics, such as ventilator type (volume- vs. pressure-cycled) or mode (assist-control vs. control), between survivors and non-survivors (p = 0.776 and p = 0.303, respectively). There were no differences in survival between patients with tuberculosis sequelae and those with kyphoscoliosis (Table 1). The 5-year survival rate was 0.69 (95% CI 0.55–0.83) in patients with tuberculosis sequelae and 0.75 (0.60–0.89) in those with kyphoscoliosis.

Multivariate Cox analysis identified comorbidity and PaCO2 at 1 month as independent predictors of mortality (Table 3). The risk of death at least doubled with a Charlson score ≥3, assuming the lower limit of the 95% CI as the minimum risk associated with the present data. Regarding PaCO2 value at 1 month, we defined several meaningful cutoff points, such as 40, 45, and 50 mmHg (Table 4). A 1-month PaCO2 of ≥50 mmHg yielded a more than 3-fold increase in the probability of death. Kaplan–Meier survival curves for all-cause mortality according to PaCO2 at 1 month are shown in Fig. 2.

Discussion

In this study, a PaCO2 value of ≥50 mmHg at 1 month after starting ventilation and the presence of comorbid conditions as assessed by the Charlson Index were predictive
Chest wall disease is a frequent cause of respiratory failure. The available data have shown considerable long-term mortality in patients with CWD, particularly those treated with LTOT.\textsuperscript{12,13} NIHMV is an effective treatment for ventilatory failure in CWD patients, but few studies have focused on the long-term survival of this population and the factors associated with mortality. The fact that a percentage of our patients died during follow-up despite treatment with NIHMV prompted us to investigate this issue.

Our finding that an elevated PaCO\textsubscript{2} at 1 month after starting NIHMV is associated with mortality seems clinically important. This finding suggests that monitoring PaCO\textsubscript{2} levels may be useful in predicting mortality in patients treated with NIHMV.

### Table 1: Patient characteristics, univariate analyses.

<table>
<thead>
<tr>
<th></th>
<th>Total series (n = 110)</th>
<th>Non-survivors (n = 34)</th>
<th>Survivors (n = 76)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>65.9 ± 10.5</td>
<td>67.4 ± 11.8</td>
<td>65.3 ± 9.9</td>
<td>0.333</td>
</tr>
<tr>
<td>Sex, male</td>
<td>64 (58.2)</td>
<td>21 (61.8)</td>
<td>43 (56.6)</td>
<td>0.679</td>
</tr>
<tr>
<td>Smoker</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>3 (2.7)</td>
<td>1 (2.9)</td>
<td>2 (2.6)</td>
<td>1.000</td>
</tr>
<tr>
<td>Past</td>
<td>46 (41.8)</td>
<td>14 (41.2)</td>
<td>32 (42.1)</td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>61 (55.5)</td>
<td>19 (55.9)</td>
<td>42 (55.3)</td>
<td></td>
</tr>
<tr>
<td>BMI, Kg/m\textsuperscript{2}</td>
<td>27.2 ± 4.9</td>
<td>26.4 ± 5.3</td>
<td>27.5 ± 4.7</td>
<td>0.252</td>
</tr>
<tr>
<td>Diagnosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tuberculosis sequelae\textsuperscript{a}</td>
<td>61 (55.5)</td>
<td>19 (55.9)</td>
<td>42 (55.3)</td>
<td>1.000</td>
</tr>
<tr>
<td>Kyphoscoliosis</td>
<td>49 (44.5)</td>
<td>15 (44.1)</td>
<td>34 (44.7)</td>
<td></td>
</tr>
<tr>
<td>Comorbidity (Charlson Index)\textsuperscript{b}</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>26 (23.9)</td>
<td>6 (17.6)</td>
<td>20 (26.7)</td>
<td>0.042*</td>
</tr>
<tr>
<td>2</td>
<td>46 (42.2)</td>
<td>12 (35.3)</td>
<td>34 (45.3)</td>
<td></td>
</tr>
<tr>
<td>≥3</td>
<td>37 (33.9)</td>
<td>16 (47.1)</td>
<td>21 (28)</td>
<td></td>
</tr>
<tr>
<td>FVC, % pred</td>
<td>32.4 ± 10.5</td>
<td>30.3 ± 8.5</td>
<td>33.3 ± 11.2</td>
<td>0.161</td>
</tr>
<tr>
<td>FEV\textsubscript{1}, % pred</td>
<td>30.9 ± 10.5</td>
<td>27.1 ± 8.4</td>
<td>32.7 ± 10.9</td>
<td>0.009*</td>
</tr>
<tr>
<td>FEV\textsubscript{1}/FVC</td>
<td>0.71 ± 0.13</td>
<td>0.66 ± 0.13</td>
<td>0.73 ± 0.13</td>
<td>0.006*</td>
</tr>
<tr>
<td>TLC, % pred</td>
<td>62.3 ± 18.5</td>
<td>69.3 ± 19.5</td>
<td>59.5 ± 17.4</td>
<td>0.019*</td>
</tr>
<tr>
<td>RV, % pred</td>
<td>100.4 ± 43.8</td>
<td>115.9 ± 47.3</td>
<td>94.0 ± 41.0</td>
<td>0.028*</td>
</tr>
<tr>
<td>Baseline status</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stable</td>
<td>93 (84.5)</td>
<td>31 (91.2)</td>
<td>62 (81.6)</td>
<td>0.382</td>
</tr>
<tr>
<td>Acute</td>
<td>17 (15.5)</td>
<td>3 (8.8)</td>
<td>14 (18.4)</td>
<td></td>
</tr>
<tr>
<td>PaO\textsubscript{2}, mmHg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>55.6 ± 11.2</td>
<td>53.2 ± 9.6</td>
<td>56.6 ± 11.7</td>
<td>0.146</td>
</tr>
<tr>
<td>One hour ventilation</td>
<td>72.8 ± 13.0</td>
<td>70.2 ± 12.7</td>
<td>73.9 ± 13.1</td>
<td>0.193</td>
</tr>
<tr>
<td>At one month</td>
<td>64.3 ± 9.1</td>
<td>61.4 ± 8.8</td>
<td>65.6 ± 8.9</td>
<td>0.025*</td>
</tr>
<tr>
<td>PaCO\textsubscript{2}, mmHg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>56.4 ± 9.0</td>
<td>56.4 ± 7.8</td>
<td>56.4 ± 9.6</td>
<td>0.990</td>
</tr>
<tr>
<td>One hour ventilation</td>
<td>41.3 ± 7.6</td>
<td>43.4 ± 7.7</td>
<td>40.4 ± 7.4</td>
<td>0.074</td>
</tr>
<tr>
<td>At one month</td>
<td>46.4 ± 4.9</td>
<td>48.4 ± 6.4</td>
<td>45.4 ± 3.9</td>
<td>0.015*</td>
</tr>
<tr>
<td>D(A-a)O\textsubscript{2}, mmHg</td>
<td>25.7 ± 11.7</td>
<td>28.1 ± 8.6</td>
<td>24.7 ± 12.8</td>
<td>0.163</td>
</tr>
</tbody>
</table>

Values are given as the mean ± SD or n (%), unless otherwise indicated. % pred = % predicted; BMI = body mass index; FVC = forced vital capacity; FEV\textsubscript{1} = forced expiratory volume in 1 s; TLC = total lung capacity; RV = residual volume; PaO\textsubscript{2} = arterial oxygen tension; PaCO\textsubscript{2} = arterial carbon dioxide tension; D(A-a)O\textsubscript{2} = alveolar-arterial oxygen difference.\textsuperscript{a}p < 0.05.\textsuperscript{b}Mainly thoracoplasty and artificial pneumothorax.\textsuperscript{a}n = 109.

### Table 2: Causes of death.

<table>
<thead>
<tr>
<th></th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory</td>
<td>22 (64.7)</td>
</tr>
<tr>
<td>Cardiovascular diseases</td>
<td>3 (8.8)</td>
</tr>
<tr>
<td>Neoplasms</td>
<td>4 (11.8)</td>
</tr>
<tr>
<td>All other causes\textsuperscript{a}</td>
<td>5 (14.7)</td>
</tr>
<tr>
<td>Total</td>
<td>34 (100)</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Sepsis (n = 2), intestinal occlusion (n = 2), and chronic renal failure (n = 1).
of PaCO₂ after starting NIHMV is poorly known. In a recently
published study, 19 the relationship between post-
NIHMV variables and withdrawal of this treatment in
a Japanese cohort of patients with tuberculosis sequelae
was analyzed. The authors found that PaCO₂ levels at 3–6
months were associated with rates of NIHMV continuation.

The main difference between this cohort and ours is the
severity of the baseline hypercapnia at starting ventilation,
which was higher in the former. So, while baseline mean
PaCO₂ in our patients was 56.4, it ranged between 64.6 and
90.2 mmHg in the Japanese cohort. Moreover, 50% of these
patients had PaCO₂ levels higher than 60 mmHg at 3–6
months post-NIHMV. In spite of these differences, both
studies point out the relevance of PaCO₂ measurement in
the control of patients stabilized after NIHMV instauration.

The need to check blood gases early in the follow-up of
these patients seems reasonable, because the initial
response observed during hospital admission should be
reassessed once ventilation is used at home. Hence,
delayed gasometric evaluation of NIHMV efficacy may
better reflect the patients’ response to NIHMV. 20 Some
patients need a longer period of time to adapt to ventila-
tion, and in others delayed problems of leaks, compliance,
or patient-ventilator asynchrony may appear. According to
our results, inability to maintain PaCO₂ <50 mmHg after 1
month of NIHMV may be a reflection of these problems or
simply indicate the limitation of this therapy to revert
respiratory failure in certain patients, thus justifying their
higher mortality. This is supported by the fact that 65% of
the deaths had a respiratory origin. ABG before starting
ventilation were not associated with mortality in our study.

This data likely express baseline severity, but their prog-
nostic value is unclear, because NIHMV was effective in
reverting ventilatory failure in most cases, and therefore,
the problem threatening the patients’ evolution would
have been modified. Our results highlight the value of
short-term surveillance that includes ABG measurement in
the management of CWD patients undergoing NIHMV. This
suggests that home ventilation should be checked early
after it is started and readjusted when hypercapnia is
detected.

We observed a consistent, progressive relationship
between comorbidity expressed with the Charlson Index
and mortality. Comorbidity is likely to influence mortality in
several ways. First, it may have provoked an increase in the
percentage of fatal nonrespiratory complications, since 35% of
deaths were not related to respiratory causes. Second,
the presence of comorbid conditions could make a patient
more susceptible to respiratory exacerbations, 21 which
were the most common cause of death in our series. And
last, the presence of additional diseases might have
depicted a deleterious effect on the efficacy of NIHMV or
make compliance more difficult. In any case, our results indicate
the need for a careful, comprehensive evaluation of
patients with CWD to improve their overall health status
and not only limit the therapeutic effort to reverting
ventilatory failure.

The patients undergoing NIHMV in the present study had
seems restrictive ventilatory pattern, and in 47.3% of
cases, mainly those with tuberculosis sequelae, airway
obstruction was also present. Obstruction has been
described as a functional respiratory complication after
tuberculosis. 22,23 Among our post-tuberculosis patients with
this complication, the functional impairment was likely due
to tuberculosis alone in 44% of patients, while smoking
could have been a contributory factor in the 56% remainder.
We attempted to ascertain the influence of airway
obstruction on mortality in the 110 patients studied.
Although a significant difference was seen in the univariate
analysis, there was no association between obstruction
and mortality in the 110 patients studied. The reason why
obstruction did not influence mortality may be due to the
fact that this impairment was appropriately treated.

The present study has several potential limitations. The
lengthy period of inclusion and follow-up, necessarily
required in a survival study, implies that changes in the
management of the population included may have occurred
over time. For example, the current indications for NIHMV
include patients with obesity-hypventilation syndrome
and some patients with COPD. Nonetheless, the indications

| Table 3 | Prognostic factors according to the Cox model for mortality. |
|-----------------|------------------|-----------------|
| Charlson Index  | HR  | 95% CI          | p          |
| 1               | 1   |                 |            |
| 2               | 3.15 | (0.91–10.96)  | 0.071     |
| ≥3              | 6.61 | (1.96–22.35)   | 0.002     |
| PaCO₂ at 1 month, mmHg | 1.13 | (1.05–1.21)     | 0.001 |

HR = Hazard ratio; CI = Confidence interval.

Table 4 | Hazard ratio for all-cause mortality according to different cutoff points for 1-month PaCO₂ in the Cox model. |
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>PaCO₂, mmHg</td>
<td>HR</td>
<td>95% CI</td>
</tr>
<tr>
<td>≤40</td>
<td>2.78</td>
<td>(0.35–22.13)</td>
</tr>
<tr>
<td>&gt;45</td>
<td>1.8</td>
<td>(0.85–3.82)</td>
</tr>
<tr>
<td>&gt;50</td>
<td>3.42</td>
<td>(1.62–7.2)</td>
</tr>
</tbody>
</table>

HR = Hazard ratio; CI = Confidence interval.
for ventilatory support in CWD have remained stable over the years, and we do not believe this factor posed a problem in the study. Another potentially conflicting point is the type of ventilator used. Classically, patients with CWD have been treated with volume-cycled ventilators, but in recent years there has been an increasing use of pressure-cycled ventilators. In our study, pressure-cycled ventilators were used in only 15% of the patients. The scientific evidence reported to date has not shown differences in the efficacy of ventilation depending on the type of cycling; thus, it is reasonable to assume that this variable would not have an influence on survival. Notwithstanding, we believe the prognostic data obtained in our study should be confirmed in CWD patients who are mainly treated with pressure-cycled ventilators.

In conclusion, our results suggest that PaCO₂ levels ≥50 mmHg at 1 month after starting noninvasive home mechanical ventilation and the presence of comorbid conditions are risk factors for mortality in patients with chest wall disease. We believe these results are relevant for the clinical management of these patients, particularly because they underscore the importance of early detection of suboptimal home ventilation, which may be amenable to correction.

Acknowledgments

The authors would like to thank Teresa Codinachs (Respiratory Medicine Department Hospital Universitari Vall d’Hebron, Barcelona, Spain) for nursing assistance. This work was supported in part by a grant from the Catalan Society of Pneumology (SOCAP 2005), and Catalan Foundation of Pneumology (FUCAP 2006).

Conflict of interest statement

Sergi Marti and Mercedes Pallero contributed equally to this study, which is a part of the doctoral thesis of Mercedes Pallero. Drs. Marti and Muñoz planned the study. Drs. Marti, Pallero and Muñoz contributed to data collection. Mr. Rios performed the analysis. All authors contributed to the writing of the manuscript.

No author reports any financial or other potential conflict of interest.

References


