in stable conditions. Acute effects of N-CPAP on arterial blood gas (ABG) values in a group of our patients with stable COPD are shown in Table 1.

Finally, we agree together with de Lucas et al. that the beneficial effect of N-CPAP in COPD is predominantly related to a better ventilation/perfusion relationship. In one 65-year-old patient with COPD, we performed a lung ventilatory scan (Fig 1) which clearly showed that N-CPAP significantly improves ventilation of the lung.

We think that more studies are needed to examine the effects of N-CPAP in COPD and to better define its role in the clinical setting.

Table 1—Effects of N-CPAP Plus Oxygen on ABGs (M ± SD) in Patients With COPD (N=10)

<table>
<thead>
<tr>
<th></th>
<th>Basal</th>
<th>35-50% Ventimask</th>
<th>N-CPAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>O2 flux, L/min</td>
<td>—</td>
<td>6</td>
<td>1.3</td>
</tr>
<tr>
<td>PaO2 mm Hg</td>
<td>44±8</td>
<td>54.7±8*</td>
<td>66.3±15†</td>
</tr>
<tr>
<td>kPa</td>
<td>5.8±1</td>
<td>5.7±1</td>
<td>8.8±2</td>
</tr>
<tr>
<td>PaCO2 mm Hg</td>
<td>52.1±13.8</td>
<td>52.8±12.5</td>
<td>52.9±10</td>
</tr>
<tr>
<td>kPa</td>
<td>6.9±1.8</td>
<td>7±1.6</td>
<td>7±1.3</td>
</tr>
</tbody>
</table>

*p<0.05.
†p<0.01.

REFERENCES


To the Editor:

We thank Dr. Dottorini and his colleagues for their comments on our paper about the effects of N-CPAP in patients with COPD (Chest 1993; 104:1694-97).

In their study, the use of N-CPAP has improved the oxygenation of COPD patients without any significant change in Pco2 levels. This effect may be attributed to a better ventilation/perfusion relationship, which is in agreement with some other studies. These results contrast with our study, in which, although there was a significant improvement in Pco2, the most relevant finding was the reduction in Pco2.

We consider that this different behavior may be explained by the distinct clinical condition of the patients. It is possible that, when patients are stable, the main effect of N-CPAP is to improve ventilation-perfusion, while, when they are in an acute situation, as it occurred in our study, N-CPAP might decrease the respiratory work. As a consequence, it would improve the respiratory pattern and the alveolar ventilation. A decrease in respiratory work has been previously associated with the use of PEEP in mechanically ventilated COPD patients, and although we did not measure tidal volume, we did indeed find a reduction in the respiratory rate.

We agree with Dottorini et al that more studies are necessary to elucidate the mechanism by which N-CPAP improves gas exchange in COPD patients. We believe that it would be useful to analyze not only the changes in arterial blood gases but also the changes in minute ventilation, tidal volume, and dead space.

Pilar de Lucas, MD, Carmen Tarancón, MD, Luis Puente, MD, Carmen Rodríguez, MD, Emilio Tatay, MD, and...
To the Editor:

We wish to comment on the article “Efficacy of Breathing and Coughing Exercises in the Prevention of Pulmonary Complications After Coronary Artery Surgery” by Stiller et al published in March 1994 Chest.² This remains an important issue for chest physiotherapy and, if the study’s findings can be reproduced, could have widespread implications for cardiac surgical units. We believe, however, that this study’s negative findings cannot be accepted uncritically, because of incomplete documentation and weakness in the trial design.

There is a lack of internal validity in this study on several key points. Whilst group allocation of patients was randomized, no attempt was made to match them. There is a lack of detail about the different levels of independent variables, and multiple dependent variables, eg, chest x-ray film and temperature, are treated as if they were independent.

There was no control for the “history threat,” since events over the 4 days of testing were not mentioned or standardized, and it would have been difficult to control for natural change.

The methods used in measuring the chosen dependent variables were not confirmed for reliability, eg, spirometry, and the appropriateness of using oral temperatures and graded chest x-ray films as measures of change in these patients is questionable. Pulmonary complications can be present without temperature change, and increased temperature can be a sign of either non-pulmonary or pulmonary infection.

Whereas the radiologist had good intratester reliability, no criteria were given to define each grade of chest x-ray films. Also, intertester reliability needs to be evaluated.

In their statistical analyses, the authors do not specify which statistical test was used for which data. Further, univariate analysis was used when multiple dependent variables, eg, temperature and chest x-ray films, were recorded. This “blows out” the error rate for determining any variable’s significance and fails to allow for evaluation of interaction between dependent variables.

There was also the problem of sample size. The patients chosen for this study had only a low morbidity (7.5%) across the groups. If therapy had, in fact, achieved some small reduction in morbidity, say no more than 10%, a sample size of many hundreds would be necessary to prove significance for the effect. There was also a mortality rate which may be significant.

Exclusions from the trial were higher risk patients, but no indication is given of outcomes for this group, who presumably received more definitive forms of physiotherapy.

It is not our purpose to argue for the efficacy of the relatively nondescript, and hard to quantify breathing and coughing exercises used in this study. Our main concern is that if accepted uncritically, the authors’ negative conclusion could be widely misinterpreted with possible prejudice to more definitive forms of chest physiotherapy such as, unilateral basal expansion, forced expiratory technique, and active cycle of breathing technique.

We only hope that the readers will take time to read the whole article and judge for themselves the value of such research findings. In conclusion, partly because of incomplete documentation and in part from inherent weakness in the trial design, it would be difficult for other centers to reproduce this study, either to confirm or refute its negative conclusions. We do not consider that these conclusions, as presented, are validated by the findings.

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REFERENCE


To the Editor:

We regret that Mr. Smith and Ms. Fowler believe that our study (Chest 1994; 105:741-47) demonstrated “... incomplete documentation and weakness in the trial design.” We strongly believe this was not the case and welcome this opportunity to address the specific points raised by the authors.

The comments made by Smith and Fowler regarding sample size are important. The study was designed with a higher incidence of clinically significant pulmonary complications expected for the control group and at least a 20% decrease in the complication rate for the treatment groups. As stated in the letter, to detect smaller differences between groups in the incidence of pulmonary complications, a much larger sample size would have been required. We considered that a difference of at least 20% between the control and treatment groups in the incidence of pulmonary complications would be considered clinically significant. It could be argued that prophylactic treatment for all patients is not cost effective if it only decreases the incidence of complications by, for example, 10%. The sample size is more than adequate for detecting small differences in the other response variables, and we are very confident about the negative findings for these variables.

Smith and Fowler note that higher risk patients were excluded from the study. As stated in the article (Chest 1994; 105:741), patients who were unable to understand written or spoken English (or who refused consent) were excluded. There is no reason to believe that these patients were at higher risk of developing complications. We did not exclude patients from participation in the study on the basis of a perceived higher risk of complications. The seven patients who were withdrawn (not excluded) from the study (because of the need for mechanical ventilation for more than 24 h or because of neurologic complications) could be considered to be at higher risk of pulmonary complications and in actual fact required varying levels of physiotherapy input, including “chest” physiotherapy. Because of these withdrawals due to intraoperative or perioperative complications, we were careful to limit our conclusions to patients after routine coronary artery surgery.

Concerning the internal validity of the study, the authors are