To the Editor:

I thank Dr. Winck and colleagues for their interesting letter on the objective salutary effect of warming nasal continuous positive airway pressure (CPAP) air on their patient’s nasal symptoms. They raise the interesting question of whether nasal symptoms are the result of the low temperature, the low relative humidity (rH), or both. Unfortunately, published studies on this subject do not currently provide the answer, since none specifically address the condition of warm air and low humidity. For instance, Richards and colleagues examined nasal airway resistance in patients with mouth leaks using nasal CPAP air conditioned in a variety of ways. This included the following: cold dry air (temperature, 22.4 to 23.2°C; rH, 61 to 67%); cold humidified air (temperature, 22.8 to 23.6°C; rH, 97 to 100%); warmer humidified air (temperature, 30°C; rH, 97 to 100%); and body temperature humidified air (temperature, 37°C; rH, 97 to 100%). They demonstrated that most of the fall in nasal resistance from baseline occurred when rH was increased even if the air remained cool, while further heating of the air while maintaining the same, almost fully saturated, humidity did not result in much additional benefit. Unfortunately, they did not test the condition of heated, relatively dry air as suggested by Winck and coworkers.

Togias and colleagues have outlined a mechanism by which nasal mucosal drying could cause an inflammatory response resulting in nasal symptoms. They challenged subjects with cold dry air and measured the osmolality of nasal secretions, nasal symptoms, and the concentration of inflammatory mediators. They were able to demonstrate that osmolality correlated with the level of inflammatory mediators in those subjects who developed nasal symptoms in response to the challenge. Other workers have demonstrated that isolated mast cells exposed to a hyperosmolar medium will release inflammatory mediators. Workers have demonstrated that isolated mast cells exposed to a hyperosmolar medium will release inflammatory mediators.3,4 Togias and colleagues outlined a mechanism by which nasal mucosal drying could cause an inflammatory response resulting in nasal symptoms. They challenged subjects with cold dry air and measured the osmolality of nasal secretions, nasal symptoms, and the concentration of inflammatory mediators. They were able to demonstrate that osmolality correlated with the level of inflammatory mediators in those subjects who developed nasal symptoms in response to the challenge. Other workers have demonstrated that isolated mast cells exposed to a hyperosmolar medium will release inflammatory mediators.3,4 Finally, a large body of evidence has emerged suggesting that a similar phenomenon, that of exercise-induced asthma, is the result of water loss from the respiratory mucosa rather than heat loss, although this is still somewhat controversial.5,6

While I applaud the results obtained by Winck and colleagues in treating their patient with warmed, but not humidified, CPAP air, I remain skeptical that this method will be universally applicable. Certainly, there is no harm in asking patients complaining of nasal symptoms from their CPAP to take the simple measure of placing the circuit under the bedclothes; this procedure is, in fact, commonly recommended by durable medical equipment suppliers in our area. However, if symptoms do not resolve readily, then elimination of mouth leaks and heated humidification should be pursued. In addition, since Dr. Winck and coworkers have the technology to pursue objective evidence that warmed, unhumidified CPAP air reduces nasal symptoms, I would encourage them to continue this line of investigation. They should also measure the CPAP air temperature at the mouth to provide evidence that routing the CPAP circuit under the bedclothes does indeed provide significant warming.

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REFERENCES

Clinical Application of a Prognostic Model for Severe Community-Acquired Pneumonia

To the Editor:

The study by Pascal et al (February 2000)1 is one more step toward defining prognostic factors in the subset of patients with severe community-acquired pneumonia who require mechanical ventilation. By reason of retrospective analysis, the authors conclude that a prediction model—based on extent of lung injury assessed by a hypoxemia index, age > 80 years, immunosuppression, medical comorbidities with a median prognosis for survival < 5 years, and number of nonpulmonary organ failures—classifies patients at the threshold of 95% mortality rate, with a positive predictive value of 100% and specificity of 100%. A few observations in the study merit further analysis. In the univariate analysis, survivors had a mean duration of hospital stay prior to mechanical ventilation of 0.8 days compared with 2.8 days (p = 0.016) for nonsurvivors. The logical query is that, in the absence of standardized criteria for mechanical ventilation, did timing of mechanical ventilation impact on prognosis? Because the prognostic model was based on assessment of parameters after the initiation of mechanical ventilation, the assessment of the hypoxemia index would depend on the timing of mechanical ventilation, with the possible corollary that earlier intubation improved outcome. This suggests that the clinician’s assessment of the timing of intubation has a great impact on prognosis. The importance of the clinician’s role is further emphasized by the disparity in prognosis in the patients who had survived an initial trial of mechanical ventilation and were later given a do-not-resuscitate/do-not-intubate status; in these instances, clinical assessment overestimated rates of mortality compared with the prognostic model.

The criteria for diseases with median prognosis for survival of < 5 years included HIV with CD4 < 200 and New York Heart Association class 3 congestive heart failure (CHF). With the
advent of highly active antiretroviral therapy and newer modalities of treatment of CHF, the prognosis for these patients has improved, and this would impact on the prognostic model in these subsets of patients. The comparable number of medical comorbidities (1.9 in each group) and their effects on survival are interesting. It is a frequent clinical observation that patients with advanced COPD, interstitial lung disease, CHF, and/or cystic fibrosis are difficult to wean and would need prolonged treatment with mechanical ventilation; however, this did not seem to be the case in this study. The number of patients with chronic respiratory disease is not clear. Conceptually, the hypoxemia index would already be compromised in these patients, and an added respiratory insult such as pneumonia could skew their prognosis adversely.

Finally, according to the model, as prognostication based on assessment of lung injury is done early in the first 24 h of mechanical ventilation, a poor prognosis implies possible cessation of intensive care. If a similarly accurate prognostication, with a noninvasive assessment of lung injury, could be made before initiation of mechanical ventilation therapy, it would translate into greater advantages for the clinician, patient, and family perspectives, because withdrawing ventilatory support is always more difficult than not initiating it.

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REFERENCES

To the Editor:

In response to the comments by Drs. Mehta and Groth, the observation that survivors had a mean duration of hospital stay of 0.8 days before receiving mechanical ventilation, compared with 2.8 days for nonsurvivors, could be taken to imply that earlier initiation of mechanical ventilation at the first sign of significant respiratory failure in community-acquired pneumonia (CAP) could favorably impact prognosis. In addition to the finding above, our observation, that the hypoxemia index was lower in survivors vs nonsurvivors (37 vs 59, respectively) over the first 24 h of mechanical ventilation, supports one of the following: (1) that survivors were intubated earlier in the course of their respiratory failure, as Drs. Mehta and Groth have suggested; or (2) that overall, survivors had less severe lung injury as a group, independent of the timing of intubation. Because this was a retrospective study, it is difficult to determine which of these two factors was more dominant. Based on our data, therefore, we cannot make the claim that earlier treatment with intubation and/or mechanical ventilation for respiratory failure ultimately leads to a more favorable prognosis in patients with CAP.

Drs. Mehta and Groth also raise a valid point that, with the advent of highly active antiretroviral therapy and newer modalities for the treatment of congestive heart failure, the overall prognosis for such patients has improved. We would urge clinicians to take these factors into account in determining whether a patient fulfills criteria for medical comorbidity with median survival of <5 years.

Finally, we would caution against using this prediction model in patients who are not receiving mechanical ventilation. The rule is not derived from that particular subset of patients with CAP whose prognosis may be influenced more strongly by factors other than the degree of lung injury. Because lung injury overall may not be as severe in that subset, there may be reason to believe that nonpulmonary organ system failure, medical comorbidity, or other prognostic markers may have greater influence and that the weighting of such factors in a model for that population might differ substantially from those in our model. Second, because of the problem of entrained air around an oxygen mask, it is difficult to obtain an accurate assessment of hypoxic lung injury in a patient who is not intubated; typically, the actual airway fraction of inspired oxygen (Fio2) in such patients is less than the Fio2 delivered in the mask. Thus, any calculation of the hypoxemia index (or other measures of lung injury) in that situation could lead to an overestimate of the degree of lung injury and a spurious overestimate of the risk of death in the patient not receiving ventilation.

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REFERENCE

Stage IA Non-small Cell Lung Cancer
A Small Proportion of Cases in the General Population

To the Editor:

A recent article by Patz and colleagues (June 2000) evidenced no correlation between decrease in tumor dimension and improvement in survival among patients with stage IA non-small cell lung cancer (NSCLC). The authors used this result to caution against the use of low-dose spiral CT as an effective tool for early diagnosis of lung cancer.

Lung cancer represents one of the most frequently diagnosed cancers, with a rather poor prognosis, and which accounts for most of all cancer deaths. Stage IA non-small cell lung cancer is, unfortunately, only a small proportion of the total case series. The Tuscany Cancer Registry (RTT) has been active in the provinces of Florence and Prato in central Italy (about 1,200,000 inhabitants) since 1985. In 1995 and 1996, lung cancer ranked first in this area among the most frequently diagnosed cancers in men, with a standardized (World Standard Population) incidence rate of 56 cases for every 100,000 inhabitants; it was seventh