Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Please include a cover letter with a complete list of authors (including full first and last names and highest degree), corresponding author’s address, phone number, fax number, and e-mail address (if applicable). An electronic version of the communication should be included on a 3.5-inch diskette. Specific permission to publish should be cited in the cover letter or appended as a postscript. CHEST reserves the right to edit letters for length and clarity.

Proteinuria in COPD Patients With and Without Respiratory Failure

To the Editor:

We read with great interest the article by Abid et al (December 2001), demonstrating the predictive value of microalbuminuria in the identification of more severe ICU patients. Microalbuminuria is reported to be an early feature of acute inflammatory conditions due to the rapid increase in renal permeability induced by inflammatory mediators and oxygen free radicals. In our opinion, hypoxemia also could play a role on capillary endothelial permeability as shown by much research, both in vitro and in vivo. In fact, studies at high altitude suggest that systemic hypoxemia may cause a generalized increase in capillary permeability, while in vitro studies have shown that cultured endothelial cells exposed to low oxygen concentrations become larger, and small intercellular gaps appear. These phenomena are reversible.

We therefore aimed to investigate the presence and degree of proteinuria in a group of patients with chronic lung diseases with or without respiratory failure by retrospectively examining the clinical records of all patients with chronic lung diseases admitted to our department (January 1999 to January 1999) due to a worsening of their respiratory conditions. Data from 177 patients were examined. Seventy-five patients were excluded due to the presence of other factors potentially inducing proteinuria (acidosis, renal failure, infections, systemic hypertension, diabetes, hypokalemia). Data from 102 patients (65 male and 37 female) are reported (Table 1). A significant correlation was found between urinary proteins and PaO2 (p < 0.01). When the patients were classified into two groups, normoxemic and hypoxemic, using the value of 60 mm Hg (8 kPa) of PaO2 as cut-off, we didn’t find any significant difference, except for the degree of proteinuria.

Patients with respiratory failure seem to have a significantly increased proteinuria as compared to a similar group of patients without respiratory failure. As serum levels of proteins were similar in the two groups, we suppose that the observed proteinuria is an acute phenomenon, yet prolonged hypoxemia could lead to a reduction in serum total proteins that could contribute to a poor outcome. Monitoring urinary and serum levels of proteins should therefore be recommended in all patients with respiratory failure.

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REFERENCES


<table>
<thead>
<tr>
<th>Variables</th>
<th>All Patients</th>
<th>Hypoxemic Patients</th>
<th>Normoxemic Patients</th>
<th>Hypoxemic vs Normoxemic Patients</th>
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<tr>
<td>Patients, No.</td>
<td>102</td>
<td>47</td>
<td>55</td>
<td></td>
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<tr>
<td>Age, yr</td>
<td>71.96 ± 0.79</td>
<td>71.8 ± 1.2</td>
<td>72 ± 0.9</td>
<td>NS</td>
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<tr>
<td>PaO2, mm Hg</td>
<td>63.8 ± 1.03</td>
<td>55.2 ± 0.7</td>
<td>71.12 ± 1</td>
<td>&lt; 0.01</td>
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<tr>
<td>Paco2, mm Hg</td>
<td>42.79 ± 0.67</td>
<td>43 ± 1</td>
<td>42.5 ± 0.8</td>
<td>NS</td>
</tr>
<tr>
<td>pH</td>
<td>7.42 ± 0.003</td>
<td>7.42 ± 0.005</td>
<td>7.41 ± 0.004</td>
<td>NS</td>
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<tr>
<td>Urinary proteins, mg/dL</td>
<td>26.13 ± 6.19</td>
<td>47 ± 12</td>
<td>8.27 ± 2.8</td>
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<td>Serum proteins, g/dL</td>
<td>6.55 ± 0.07</td>
<td>6.6 ± 0.109</td>
<td>6.5 ± 0.083</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Data expressed as mean ± SE. NS = not significant.

To the Editor:

We appreciate the comments by Dr. Cogo and colleagues, raising the hypothesis that hypoxemia in itself could play a role in the development of hypoaalbuminemia. However, as in many areas of medicine, is this a cause-effect relationship or simply an association, i.e., the inflammatory response is responsible for permeability alterations in the kidney (resulting in hypoalbuminuria) and in the lung (resulting in hypoxemia)?

Moreover, some of our patients required a high fraction of inspired oxygen (sometimes in addition to positive end-expiratory pressure), but we never allow hypoxemia (PaO2 ≤ 60 mm Hg) to persist in critically ill patients. Hence, we do not believe a low PaO2 by itself, could have contributed to our findings.

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Lung Cancer Screening Debate

To the Editor:

With respect to the article by Chirikos et al (May 2002) and the accompanying editorial by Dr. Grannis (May 2002), while the analyses and comments are elegant, they do not address the key issue in the lung cancer screening debate. As noted by Dr. Chirikos, “First and foremost, a cost-effective analysis must assume that effectiveness exists.” Dr. Grannis assumes “a very real possibility that LC [lung cancer] screening, despite high initial cost, can save money as well as lives.” It is problematic that these assumptions are unproven and that considerable data are currently available that, at the very least, suggest that the assumptions are incorrect.

Indeed, in the article following that of Chirikos et al, Lopez-Encuentra et al report relatively uniform results in the group of individuals with nodules ≤ 2 cm despite the heterogeneity of nodule sizes within that group including smaller nodules. While the number of cases was relatively small, similar findings have been reported by Patz et al, although the nodules evaluated were ≤ 3 cm.

I suspect that this is one of many communications that will be directed toward this issue and with justification. Costs engendered by CT screening are large whenever a nodule is detected, regardless of size. Small nodules may be observed, producing large imaging costs, while larger nodules may undergo biopsies, incurring both cost and patient morbidity. Indeed, the unjustified proliferation of independent centers offering thoracic CT screening services needs to be considered in the context of the number of nodules found and the small percentage that ultimately are shown to be neoplastic. Since the concept of lead-time bias has not been resolved in answering questions about the value of such screening, I believe the most pressing issue in this debate is the justification of screening as a modality for decreasing mortality. Until that answer is established, the elegance of analyses such as that by Dr. Chirikos are interesting but not applicable. I believe that efforts to utilize noninvasive techniques such as positron emission tomography and somatostatin receptor imaging (Neotect; Berlex Laboratories; Montville, NJ) have promise in diminishing morbidity and, possibly, cost. Efforts should be directed also at the validation of algorithms with which to evaluate nodules as noninvasively as possible.

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REFERENCES
2 Grannis FW. Lung cancer screening: who will pick up the tab? Chest 2002; 121:1388–1390

To the Editor:

In a very sophisticated analysis (May 2002), Chirikos and colleagues report that lung cancer screening with low-radiation-dose CT is likely to be cost-effective if it proves to be effective in reducing lung cancer mortality rates. While the authors’ assumptions regarding costs may be conservative, their assumptions regarding effectiveness are very optimistic. It is important to note that, as the incremental benefits of an intervention approach zero, the incremental cost-effectiveness ratio approaches infinity. If low-dose CT leads to only marginal improvements in mortality rates, its cost-effectiveness ratio will be highly unfavorable. In contrast, if the health benefits are large relative to the resources consumed, the intervention will be economically attractive. Thus, it is important to determine not only whether low-dose CT is effective, but also to determine how effective it is. Even if > 50% of cancers are detected at a localized stage, we currently do not know enough about the behavior of screen-detected tumors to determine the magnitude of improvements in life expectancy for these patients.

One might also take issue with the claim that the cost estimates reflect a “worst-case” scenario. Although not stated in the article, the analysis assumes that existing resources and facilities are sufficient to support a program of mass screening. In fact, it is likely that health systems will need to make substantial up-front investments in capital equipment and personnel to support the implementation of new screening programs, because of the large numbers of current and former smokers who might be eligible.