Transcutaneous Partial Oxygen Tension and Lung Mechanics During Methacholine Inhaled Challenge

To the Editor:

The possibility of assessing the response to inhaled methacholine by means of transcutaneous oxygen tension (tcPO2) variations has been recently proposed.1 For this reason, it was with great interest that we read the article by Fontana and coworkers,2 which appeared in the May 1993 issue of Chest.

Their findings suggest a link in adult asthmatics between changes in airway caliber and blood gas exchange, which do not appear in normal subjects up to a methacholine concentration of 64 mg/ml. We also extensively studied for several years the significance of tcPO2 variation during methacholine challenge, and preliminary data have been presented.3

The current results regarding 112 consecutive outpatients (58 men and 54 women, mean age 32 ±4 years) admitted to our laboratory, are similar to those of Fontana et al.2 The drug cumulative dose threshold causing a 20 percent decrease in FEV1 (PC20FEV1) and a 15 percent decrease in tcPO2 (PC1502) were calculated from the dose-response curve. Statistical study revealed a significant correlation coefficient (r=0.696, p<0.01) between these two indices. Furthermore, we similarly calculated the dose threshold causing a 35 percent decrease in specific airway conductance (PC35Gaw) by means of body plethysmographic method. For this index, we found a closer correlation coefficient when compared with PC1502 (r=0.866, p<0.001).

In 28 subjects (8 normal men), blood oxygen (PaO2) was randomly obtained, from radial artery puncture, to confirm transcutaneous data at maximum fall. Differently from Fontana,2 we did not notice any clear underestimation of transcutaneous values in our sample (mean PaO2 83 ±3, mean tPO2 80 ±5 mm Hg). Mean PC35Gaw was significantly lower, in asthmatic responders, than the corresponding response to methacholine calculated with FEV1 (380 ±88 µg and 487 ±102 µg for PC35Gaw and PC20FEV1, respectively).

We can similarly conclude that transient oxygen fall is clearly related to the bronchial obstruction induced by methacholine. In addition, we suppose that our relationship when comparing tcPO2 with FEV1 variations is quite different because of the lower cut off in oxygen PC significance. A higher correlation was found with the Gaw variations, which, therefore, appears a more sensitive index to evaluate the test even when tcPO2 variations are smaller.

Further investigations, however, are needed to better understand the physiopathologic link between mechanic and vascular modifications.

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REFERENCES

α1-Antitrypsin Phenotypes and Bronchial Asthma

To the Editor:

We read with interest in the March, 1993, issue of Chest the article by Colpa et al.1 We would like to make the following clarifications on the data in their Table 3 for findings obtained in Spain for α1-antitrypsin phenotypes. Although the authors do not cite the source (nor do they in another of their articles;2 they do in an earlier one3), referring to a study of ours.4 The number of patients with atopic dermatitis was 55 and not 35, and we did not find any SZ phenotype as they report.1-3

Take into account all of our data4-6 shown in Table 1, we con-

Table 1—α1-Antitrypsin Phenotypes in Healthy People and Atopics Patients

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>N</th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>MM</td>
<td>102</td>
<td>19</td>
<td>21</td>
<td>25</td>
</tr>
<tr>
<td>MS</td>
<td>36</td>
<td>3</td>
<td>1</td>
<td>12</td>
</tr>
<tr>
<td>FM</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>MZ</td>
<td>0</td>
<td>8</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>ZZ</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>n</td>
<td>200</td>
<td>30</td>
<td>25</td>
<td>38</td>
</tr>
</tbody>
</table>

N=healthy people.
Group 1 = patients suffering from both atopic dermatitis and extrinsic bronchial asthma.
Group 2 = patients with atopic dermatitis without bronchial asthma.
Group 3 = patients with extrinsic bronchial asthma without atopic dermatitis.

n=number of subjects in each group.

Included that (1) no significant differences exist between any population groups for MM and FM phenotypes; and (2) there are significantly higher quantities of MS phenotypes in group 3 than in group 2 (p<0.02), MZ phenotypes in group 1 than in healthy people (p<0.00001) or in group 3 (p<0.01), and ZZ phenotypes in group 2 than in healthy people (p<0.005).

In group 1, we also found a relation between the presence of MZ phenotype and another genetic marker, the goniometric tla angle in the right hand.6

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To the Editor:

We are grateful to Dr. Monteseirin and colleagues for their correction of the typographical errors in our papers and also for providing clarification of his data. Of most interest, the increased prevalence of the S variant for α1-antitrypsin in asthmatics is again indicated, this time in a population from Spain.

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More on Asthma Prevention

To the Editor:

Dr. Bailey’s response to Dr. Unger’s critique1 on the former’s2 not necessarily benign neglect on preventive measures in controlling asthma is like preparing a pot of chili with only bell pepper.

Even if the pulmonologists choose to ignore allergen immunotherapy as an effective form of antiallergy treatment, the general lack of understanding on effective measures to reduce allergen exposure amounts to poor medical practice.

None of us would argue Dr. Bailey’s fear that restricting a patient’s life-style excessively is undesirable. We allergists have come to the same conclusion by providing our patients the information as well as the tools to handle their indoor allergen exposure. There is simply no excuse for anyone treating patients with allergic asthma to be ignorant of the recent advances in house dust mite and cat allergen avoidance measures. Our ultimate goal, of course, is to serve our patients better.

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REFERENCES

To the Editor:

Thank you very much for your letter regarding my response to Dr. Unger’s critique (Chest 1993; 104:326). While I would not want to leave the tabasco sauce out of a pot of chili, I would not want to put in too much either. While I believe that allergy therapy has a distinct place in the treatment of asthma, it should be in a supporting role rather than as the main character, which currently for most patients is the proper use of inhaled steroids.

I am in complete agreement with you that there is no excuse for anyone treating patients with asthma to be ignorant of the recent advances in house dust mite and cat allergen avoidance measures. These are of vital importance and should be used aggressively.

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Talc Slurry for Pleurodesis

To the Editor:

There is continuing concern about the best palliation for malignant pleural effusions.3 This is especially true since tetracycline is no longer available as a sclerosing agent.4 Talc has become recognized as an excellent sclerosing agent; however, it is often not recognized that thoracoscopy is not required for satisfactory talc sclerosis. Recent review articles suggest that general anesthesia and thoracoscopy are necessary.3 During the last year, we have had excellent results from a talc slurry injected through a simple chest tube.

The techniques of the use of talc slurry have been well described by Weissberg.5 Briefly, an adequate bore chest tube is inserted and the pleural cavity drained dry. Two grams of sterile talc in a slurry with 50 ml of normal saline solution is injected followed by an additional 25 to 50 ml of saline solution to clear the tube. The tube is clamped for 1 to 2 h and the patient repositioned frequently. The tube is then reattached to underwater seal with suction. We do not know if repositioning is absolutely necessary. The suction is maintained until the daily chest tube drainage is less than 50 ml or for 4 days.

The outcome in our first eight patients is shown in Table 1.

Table 1—Results of Talc Slurry

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Cancer</th>
<th>Recurrence of Effusion</th>
<th>Survival, wk</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Small cell, lung</td>
<td>None ipsilateral; contralateral (4 wk)</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>Breast</td>
<td>None</td>
<td>7</td>
</tr>
<tr>
<td>3</td>
<td>Non-small cell, lung</td>
<td>Partial (10 wk)</td>
<td>13</td>
</tr>
<tr>
<td>4</td>
<td>Small cell, lung</td>
<td>None</td>
<td>Alive at 27 wk</td>
</tr>
<tr>
<td>5</td>
<td>Non-small cell, lung</td>
<td>None</td>
<td>6</td>
</tr>
<tr>
<td>6</td>
<td>Breast</td>
<td>None</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>Adenocarcinoma, primary unknown</td>
<td>None</td>
<td>Alive at 11 wk</td>
</tr>
<tr>
<td>8</td>
<td>Piriform sinus</td>
<td>None</td>
<td>Alive at 9 wk</td>
</tr>
</tbody>
</table>