Communications to the Editor

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. Specific permission to publish should be cited in a covering letter or appended as a postscript.

T-lymphocyte Subpopulations in Patients with Respiratory Allergy

To the Editor:

We have read with great interest the study done by Kus et al.,1 in which they find that suppressor lymphocytes are decreased in number in a patient with Churg-Strauss syndrome. They hypothesize on the possible role that these T-lymphocyte subpopulations might play in the pathogenesis of this syndrome.

We have studied the average percent of T-lymphocyte subpopulations in peripheral blood using monoclonal antibodies in 152 patients with bronchial asthma and/or extrinsic allergic rhinitis and in 30 healthy subjects (control group). We have found significantly decreased OKT8 levels in patients with respiratory allergy as compared to the control group (allergic 25.35, control 30.46; p<0.001). This suggests that a pathogenic role is played by these T-lymphocyte subpopulations in allergic disease, as other authors have affirmed.2 Tse et al.3 have also found a significant decrease in suppressor lymphocytes in patients with bronchial asthma (without Churg-Strauss syndrome).

Our study also demonstrates, as does Hsieh,4 a decrease in helper lymphocytes (OKT4) in patients with respiratory allergy (Table I).

It is possible that the patient studied by Kus et al., who had pathologic levels of IgE, presented changes in suppressor lymphocytes as a manifestation of a respiratory allergy.

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REFERENCES

Table I—Results of T-lymphocyte subpopulations in the 30 healthy subjects and 152 patients affected by respiratory allergy

<table>
<thead>
<tr>
<th>Subpopulation</th>
<th>Healthy</th>
<th>Allergic</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Helper (OKT4)</td>
<td>50.56±4.55</td>
<td>41.71±6.32</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Suppressor (OKT8)</td>
<td>30.46±2.14</td>
<td>25.35±5.22</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Helper/suppressor</td>
<td>1.66±0.19</td>
<td>1.69±0.37</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>


To the Editor:

We agree that the literature cited demonstrates changes in T-cell subpopulations as suggested with decreased OKT8, although unlike Jutribo et al., other authors4 do not observe decreased OKT4 and therefore report an increased OKT4/OKT8. We would further agree that this appears to be markers of respiratory allergy and note the evidence that, with treatment of the allergy, improvement toward normal can be observed in these parameters.1

We would note, nonetheless, the marked degree of OKT8 reduction (and high ratio), more marked than usually reported in cases with respiratory allergy. It is possible, nonetheless, that this may be associated with the allergic condition underlying our patient's illness, albeit of a more marked degree than is usual.

Thank you for your letter.

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Effect of Food on Hemodynamics

To the Editor:

Wilson and colleagues ("The effect of eating on thallium myocardial imaging," Chest 1986; 89:935-96) present fascinating and potentially valuable material on the effect of eating on thallium myocardial imaging. The general issue raised by their needed investigation is the neglect of the effect of food consumption on the results of diagnostic and therapeutic modalities (reflected by the paucity of literature on the subject). In 1983 we reported a double-blinded, prospective, randomized trial of two long-acting nitrates against placebo, including a standardized meal at four hours from baseline.1 We obtained the expected effects on systolic time intervals (STI): increases in pre-ejection period (PEP) and ratio of pre-ejection period to ejection time (PEP/LVET), and decreased ejection time index (ETI), lasting up to the fourth hour intervention—consumption of food. At that point, PEP/LVET plunged sharply back to control levels, which did not change until the sixth hour endpoint of the investigation. (Our previous studies had documented a six-hour effect on these agents).1 This reversal of nitrate effects might not have occurred without consumption of the standard meal, suggesting that
food should interfere with nitrate effects.  
Recent hemodynamic studies in coronary patients indicate increased cardiac and stroke index without changes in mean arterial or pulmonary wedge pressures, which is consistent with the effect of food on STI in our study. Previous studies of normal conscious dogs were also in agreement, indicating an enhanced isotropic state during the postprandial period. These findings could have had a bearing on the altered thallium myocardial kinetics reported by Wilson and colleagues, perhaps through alterations in flow, and as the authors suggest, myocardial metabolism.

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To the Editor:

Dr. Spodick's comments concerning the effect of eating on cardiac hemodynamics are greatly appreciated. With specific reference to our study, they point out the possibility that the increased clearance of thallium-201 by the myocardium may have been due in part to a decrease in coronary resistance and, therefore, an increase in coronary blood flow (since arterial pressure also increased), as observed by Vatner et al. However, previous studies by us failed to show an increase in coronary blood flow during intravenous infusion of glucose-insulin-potassium solution, even though thallium-201 myocardial clearance increased. We cannot exclude an increase in coronary blood flow in the patients who ate, but suspect that an independent effect of insulin on the myocardial handling of thallium is also involved. Further studies are needed to elucidate the relative importance of flow and metabolism in thallium kinetics.

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Effects of Beta-Adrenergic Agents on Hypokalemia

To the Editor:

We read with interest the paper of Rohr et al entitled, "Efficacy of parenteral albuterol in the treatment of asthma" (Chest 1986; 89:348). The authors discuss different metabolic effects of adrenergic agents parenterally administered. They note that these drugs could produce hypokalemia, potentially dangerous especially in patients with previous borderline or low serum K+ levels. This effect was previously described with different β-adrenergic agents administered either by inhalation or per os. In this context, it is interesting to note our findings in a group of 12 patients with COPD to whom we administered a β2-adrenergic agent (albuterol, 15 mg p.o.). None was on steroid or digitalis therapy. Aminophylline and diuretic therapies were withdrawn 24 hr before study. In these patients, we measured plasma K+ by means of a KNA sodium-potassium analyzer and arterial blood samples for gas analysis obtained through an indwelling catheter placed in the humeral artery. The samples were obtained before and at 60, 120 and 150 min after albuterol administration. Control plasma K+ (x±SEM) was 4.25±0.14 and 3.76±0.07, 3.71±0.07 and 3.78±0.11 mEq/L at 60, 120 and 150 min after albuterol administration, respectively. These values were significantly different as compared with control values (p<0.001).

FIGURE 1. Correlation between control plasma K+ values vs Δ max K+ after albuterol administration.

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