from such patients, which gives to AP the same BAL cellular profile as hypersensitivity pneumonitis.\(^4\)

To further analyze the immunologic processes involved in this disease, we evaluated the presence of both IgG and IgM in the BAL fluids from patients with AP. Immunoglobulins were quantified by ELISA (enzyme linked immunosorbent assay). Albumin content was separately determined by immunonephelometric study. The assays were initially performed in four male patients (age 70 ± 6 years) treated for arrhythmia or angina pectoris with oral amiodarone (1,000 mg/week) for a mean duration of 66 ± 69 months (range, 22 to 156 months; mean cumulative dose, 373 ± 250 mg). In these patients, AP was diagnosed on the basis of diffuse interstitial and/or alveolar opacities on chest roentgenographic films, associated with a mild-to-severe dyspnea and hypoxemia and an inflammatory syndrome. Every other etiology, especially acute cardiac failure, was ruled out by appropriate investigations (including cardiac catheterization). All patients subsequently improved after withdrawal of the drug associated with steroids, further confirming the diagnosis.

In these patients, an important elevation of IgG and, moreover, IgM (Table 1) in BAL fluid was found when compared to normal subjects. However, this phenomenon was accompanied by severe exudative lesions suggested by a high albumin level in BAL fluid from patients with AP. This exudation may explain in part the increased immunoglobulin levels, but IgG- and IgM-to-albumin ratios were significantly higher than in normal subjects, suggesting the hypothesis of a local production (or active transportation) of immunoglobulins in such patients.

To evaluate the relevance of these findings in AP, we also determined immunoglobulin levels in BAL fluid from subjects treated with amiodarone, but exhibiting no pulmonary abnormality, neither radiologically nor clinically, and with normal pulmonary function test results. Immunoglobulin and albumin values were assayed in BAL fluid of 6 amiodarone-treated subjects (age, 62 ± 12 years) receiving amiodarone (1,000 mg/week, except in one case, 3,000 mg/week) for a mean duration of 35 ± 26 months (range, 6 to 60 months; mean cumulative dose, 162 ± 100 g). All data are shown on table I.

Our results show an elevation of both IgG and IgM levels in BAL fluid of amiodarone-treated subjects, but at a lower degree than in patients with AP. However, the most striking difference between these two groups is the absence in the former group of albumin level increase in BAL fluid. This fact suggests that, when there is no obvious pulmonary involvement, there is no exudative alveolar structures, only increased immunoglobulin levels. Thus, our findings may be interpreted as enhanced local immunoglobulin production in the alveolar structures. It is noticeable that this increased production also predominates on the IgM fraction, for only IgM-to-albumin ratio is significantly different from normal subjects in these cases. This point reflects another similarity with hypersensitivity pneumonitis, where IgM increase in BAL fluid has been reported.\(^3\) We conclude that immunological abnormalities do exist in the lungs of patients treated with amiodarone but exhibiting no pneumonitis. These abnormalities are attested to by immunoglobulin (mainly IgM) level elevation in BAL fluid, and may represent the first immunological marker of an evolution towards hypersensitivity pneumonitis.

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**REFERENCES**


**Association of Ulcerative Colitis and Sarcoidosis?**

To the Editor:

We recently encountered three patients with classical ulcerative colitis who developed histologically-proven sarcoidosis, stage III, in the course of their disease. In all three patients, no granuloma was seen on histologic examination of the biopsy specimen taken from the colonic mucosa, sulphasalazine was not administered prior to establishment of a diagnosis of sarcoidosis, and the chest X-ray findings were not consistent with the unexplained bronchopulmonary disease sometimes associated with ulcerative colitis.\(^4\) Although this association may be coincidental,\(^3\) we suggest a possible common initiating factor(s) in both disorders, in light of current knowledge about the immunoregulatory defects postulated for both disorders, namely: 1) heightened activity of circulatory killer and natural killer lymphocytes; 2) excess of helper T-lymphocytes in sites of disease activity in the intestinal mucosa and alveolar wall, respectively; and 3) the presence of circulating immune complexes and autoantibodies.\(^4\)

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**Table 1—Comparison of Amiodarone-treated Subjects and Normal Subjects**

<table>
<thead>
<tr>
<th></th>
<th>Albumine</th>
<th>IgG</th>
<th>IgM</th>
<th>IgG/Alb</th>
<th>IgM/Alb</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>μg/ml</td>
<td>μg/ml</td>
<td>ng/ml</td>
<td>μg/μg</td>
<td>ng/μg</td>
</tr>
<tr>
<td>Normal subjects</td>
<td>26.5 ± 11.2</td>
<td>4.9 ± 4.3</td>
<td>11 ± 14.5</td>
<td>0.16 ± 0.09</td>
<td>0.37 ± 0.37</td>
</tr>
<tr>
<td>Patients with</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td>p&lt;0.05*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amiodarone-treated</td>
<td>30.3 ± 9.2</td>
<td>12 ± 6</td>
<td>106.5 ± 145.6</td>
<td>p&lt;0.05</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Comparison to normal subjects.
Embryology of Bronchial Atresia

To the Editor:

We were interested by the report by Williams and Schuster on the association of bronchial atresia and bronchogenic cyst (Chest 1985; 87:396-98). We are aware of two similar cases in previous publications. Case 5 of Tsui et al. showed bronchial mucocele and hyperinflation of the right upper lobe, together with a mediastinal bronchogenic cyst. Case 2 of Brocard and Galloude, had bronchial atresia and mucocele of the posterior segmental bronchus of the left lower lobe and a bronchogenic cyst in paraseptalhepial position, as was confirmed pathologically.

Other congenital anomalies have been associated with bronchial atresia: pericardial defect, strum septum defect and left-sided inferior vena cava, congenital cystic adenomatoid malformation (CCAM) and unilateral agenesis of the kidney, anomalous venous drainage of the left upper lobe, pulmonary sequestration, and fusion of apophyseal joints C2-C3.

These associated defects occur early in embryologic development; eg, the pericardium is normally formed in the fifth week and the interatrial septum is normally complete in the eighth week. CCAM Stocker type III is believed to result from an injury at the time of early budding of the lung. Renal agenesis occurs prior to 31 days. These associations indeed point towards the early phases of budding of the lung (fourth to sixth week) as the origin of bronchial atresia, at least for a number of cases.

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REFERENCES


Hypokalemia from Usual Salbutamol Dosage

To the Editor:

It is well known that both salbutamol and theophylline poisoning produce hypokalemia and hyperglycemia, and that in both cases these changes have been reversed by administration of propranolol. However, it might be possible, we think, that therapeutic concentrations of salbutamol also produce hypokalemia. We describe a case of a 34-year-old male nurse and football player who took 6 mg (0.08 mg/kg) salbutamol half an hour before a 30 min, 100 W exercise test, which was performed as described by Tarssanen et al, in order to study serum potassium changes during exercise with medication.

As we know, potassium concentration normally rises during exercise. However, after taking salbutamol, our patient's serum potassium concentration rised remarkably less than during the exercise test without medication. Serum potassium concentration was 0.5 μmol/l lower than before the exercise and the patient became hypokalemic (3.4 μmol/l) 30 min after the exercise (Fig 1). The patient felt palpitation and he received 40 mg propranolol. About two hours later, he went to play football. Thereafter, he felt unusual palpitation and tiredness that, we suppose, might be due to profound hypokalemia.

Due to our exercise test results, we suggest that hypokalemia may be noticeable also during ordinary salbutamol treatment. Rapid potassium changes, especially hypokalemia, during exercise may achieve dangerous rhythm disturbances.

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FIGURE 1. Serum potassium levels during exercise with salbutamol 6 mg (••••••••) and without medication (- - - - - -).