Table 1—Study of Percent Changes of Cardiac Arrhythmias during Sleep compared to Wakefulness

<table>
<thead>
<tr>
<th></th>
<th>Patients (%)</th>
<th>Healthy subjects (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature atrial contraction</td>
<td>6.95 ± 2</td>
<td>1.06 ± 6.2</td>
</tr>
<tr>
<td>Premature ventricular contraction</td>
<td>3.9 ± 7.9</td>
<td>1.1 ± 2.2</td>
</tr>
<tr>
<td>Electric aberrances</td>
<td>8.69 ± 1.35</td>
<td>1.44 ± 7.1</td>
</tr>
<tr>
<td>Atrioventricular conduction alterations</td>
<td>1.23 ± 1.9</td>
<td>1.0 ± 1.1</td>
</tr>
<tr>
<td>Sinus arrest</td>
<td>3.17 ± 7.2</td>
<td>1.1 ± 2.0</td>
</tr>
</tbody>
</table>

The results are expressed in Table 1. In normal subjects, the levels of oxygen saturation at 50 percent cumulative time index did not differ significantly between wakefulness and sleep, nor did the incidence or types of cardiac arrhythmia. However, in patients with sleep disorders we demonstrated the presence of multiple oxygen hemoglobin desaturation episodes while asleep (mean increment 6.13 ± 0.84 percent over episodes while awake) plus a statistically significant (p<0.01) increase in the incidence of nocturnal cardiac arrhythmias, as well as all of the types studied (Table 1). In every case there was a close, statistically significant (p<0.01) temporal relationship between the decrease of oxygen saturation at 50 percent cumulative time index and the presence of cardiac arrhythmias.

Our results agree with those of Guilleminault et al and help support the importance of sleep disorders in the origin of alterations in cardiac rhythm.

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Serology and P carinii

To the Editor:

As concerned physicians and/or researchers with long-term experience with Pneumocystis carinii, we read with great interest the lively exchange of views by Drs. Pifer and Hughes concerning the value of serology in the diagnosis of P carinii pneumonia (Chest 1986; 89:764). We are writing to indicate our full support of the data and arguments presented by Dr. Hughes. Based on an analysis of the literature, review of our own published and unpublished data, as well as our collective clinical and laboratory experience, we cannot recommend presently available serum antigen and antibody detection tests for the diagnosis of pneumocystosis in individual patients. These tests simply lack the sensitivity, specificity and reproducibility to be used in that manner. At present, the only reliable method for diagnosing P carinii pneumonia is by morphologic demonstration of the organism.

A simple, reliable, non-invasive and widely available test for the diagnosis of pneumocystosis is urgently needed. Detection of the organism, its antigens, genetic material or metabolic products in respiratory tract secretions or serum would be a logical first step in accomplishing this goal. Tests to diagnose P carinii pneumonia must be validated by carefully-designed prospective multicenter trials which include the following: 1) the use of coded serum specimens from patients with detailed clinical, morphologic and cultural information to confirm the presence of P carinii and/or other causes of pneumonia; 2) collection of at least one serum specimen from each patient before an invasive diagnostic procedure is performed; and 3) testing the same specimens by several institutions to show reproducibility. Until these criteria are met, we recommend that physicians not use serum antigen detection systems for clinical decision-making in the management of patients with suspected P carinii pneumonia.

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To the Editor:

We have stated that clinical decisions concerning P carinii infections should not be made solely on the basis of serology. Ninety-one percent of clinicians submitting sera have indicated that serologic data provided by our laboratory were useful and helped provide a more complete concept of the patient's status. We have encountered no one sufficiently naive to believe that any laboratory test result taken out of context provides a sole and adequate basis for clinical decision-making. Our recent publications emphasize that we are still attempting to understand P carinii serology and its significance.1-11

Since the 13th century and the career of Roger Bacon, it has been customary for scientists to base their opinions on hard scientific data, the foundation of the scientific method. Dr. Arnold Relman, editor of the New England Journal of Medicine, recently stated that: "It is the published evidence that should speak, not the person."

With that advice in mind, I invite the concerned to share the references of their relevant, published, peer-reviewed data with all of us. The only existing published reports are not comparable to our methods and consist of efforts that were less successful than ours.1-13

It has also been customary to refer to unpublished data only when the scientific point to be made is an insignificant one not requiring the scrutiny of peers. Experience and unpublished data may at best be only anecdotal; if the data consist of something more substantial than this, the scientific community would be pleased to review it. The reader should be aware that the unpublished data probably refer to data collected in a study of sera which, when the code was broken, were found to be unacceptable and invalid due to age, lack of tissue documentation, lack of TMP-SMX treatment data, or combined deficiencies.

In my letter to fellow AIDS Working Group members, I specifically invited the submission of coded, tissue-documented cases for

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Allergy Skin-Test Reactions and Chronic Airflow Obstruction in Children

To the Editor:

A high level of total serum IgE in smokers has been related to the impairment of ventilatory function. This finding was confirmed in only one of two recent cohort studies. The authors concluded that elevated levels of serum IgE are probably associated with functional impairment. These studies used percent of predicted FEV1 as an indicator of functional impairment. Nevertheless, the response to inhaled bronchodilator drugs can be a more sensitive method of detecting functional impairment in an early stage, ie, in children.

METHOD

We studied 30 skin-test positive children (23 boys and 12 girls, ages 11 to 14 years), without any respiratory symptom (19 rhinitis, 16 healthy). They were compared with a group of age and sex-matched normal subjects. Respiratory symptoms, family history and smoking habits of parents were investigated by a modified ATS questionnaire. Skin tests were carried out with extracts of grass, Dermatophagoides pt, birch, mugwort and parietaria. Lung function tests (lung subdivision, Raw, Vtg and maximal expiratory flow volume curve [MEFV]) were measured by a constant volume integrated flow plethysmograph (model 2800, Gould, US) using a Hans Rudolph pneumotach. All tests were measured in triplicate except Raw, of which five acceptable curves were recorded.

Lung subdivision, Vtg and MEFV (in triplicate) and Raw (at least five acceptable curves) were measured at baseline. Fenoterol (200 mcg) was administered via a DeVilbiss 564 nebulizer attached to a Mefar (Brescia, Italy) dosimeter. Postdirillator measurements were taken 15 min after the last inhalation.

RESULTS

The two groups did not differ with regard to smoking habits of parents (chi-square test, p > 0.05). One-way variance analysis did not show any difference in the baseline measurements (FEV1, FEV1/FVC, FEF25-75, FEF50-75, Vtg and Scaw). After fenoterol inhalation, changes in FEV1, FEV1/FVC, FEF25-75, Vtg and Scaw were not significantly different in the two groups (p > 0.05). On the other hand, FEF50-75 increased more significantly in skin-test positive children than in control subjects (mean 25.6 ± 10 vs 8 ± 15 percent, p < 0.025). This suggests that an obstruction of the lower bronchial tract is present in skin-test positive children, even if they are respiratory symptom-free. This can result in an increased susceptibility to chronic lower respiratory disease, and cigarette smoking can be the trigger to the development of symptomatic lung disease.

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