right middle lobe aspiration pneumonia occurred after a suicide attempt with ingestion of paint thinner. In this case, the patient was unconscious and probably vomited and aspirated when lying prone.

Aspiration leading to abscess formation in the right middle lobe has been reported, but is rare. One patient with poor dentition aspirated infective material when bent forward carrying sacks of coal. Another patient fell into a harbor when inebriated and presumably aspirated oily material when swimming prone, to the dock.

In summary, the recognition that an isolated right middle lobe aspiration may occur when in the siphonage position will help in making this diagnosis, particularly if the patient chooses to conceal this information.

David H. Carlson, M.D., Department of Radiology, Newton-Wellesley Hospital, Newton Lower Falls, Massachusetts

REFERENCES

PiM Subtypes in COPD

To the Editor:

The recent article by Bencze and colleagues on alpha1-antitrypsin Pi M subtypes in patients with chronic obstructive pulmonary disease (Chest 1980; 77: 761-763) and the accompanying editorial by Mittman and Taylor (1980; 77:721) require some comment.

Readers who have followed the recent literature on alpha1-antitrypsin may be confused by the nomenclature used by Bencze et al. What they call M5 is M5 in the nomenclature recommended by the international Pi committee1 and accepted by most workers in this particular area. M5 of Bencze et al is M5 in the recommended nomenclature. In the following, I shall, therefore, use the recommended nomenclature and give that of Bencze et al in parentheses.

Not every observer will find it easy to distinguish all of the phenotypes shown in the isoelectric focusing gel in their Figure 2. It seems that in particular types M1 M3 (M1 M3), M5 (M5), and M5 may be difficult to distinguish. Misclassification may be a real possibility. One way to test this is to check the agreement between the actual phenotype frequencies with those expected under Hardy-Weinberg equilibrium using the allele frequencies of the control group. Table 1 shows this comparison. The chi2 for the phenotype frequencies with expected values of more than 5 is 17.7, P<.001 (3 DF). It should be noted that the significant deviation from Hardy-Weinberg equilibrium is solely due to the discrepancy between the observed and expected values for the phenotypes M5 (M5) and M5 (M5). Such a discrepancy would be expected if these two phenotypes and M5 were not always correctly classified because M5 and M5 (M5) are very close in their respective isoelectric points. The group of patients (B) is not necessarily expected to satisfy the conditions of Hardy-Weinberg equilibrium because by definition it was not collected randomly, and because of its small size.

There are, of course, other explanations for deviations from Hardy-Weinberg equilibrium, but the possibility of misclassification has to be seriously considered. Since the message of the paper under discussion depends on the different frequencies of phenotypes, M5, M5, M5 (M5), and M5 (M5) in groups A and B, reliable typing is clearly an important issue. Methods that can distinguish M5, M5, and M5 have been described.2,4

As pointed out by Mittman and Taylor, one should not arrive at any hasty conclusion concerning job placement, risk calculations and drastic measures, based on information contained in this article. The continuing uncertainty about the risk of COPD in M2 heterozygotes (for a recent review)3 has made most of us more cautious in our conclusions.

Friedrich Kueppers, M.D., Professor of Medicine, Temple University School of Medicine, Philadelphia

REFERENCES

Table 1—Pi Phenotype Frequencies in Control Group A

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</thead>
<tbody>
<tr>
<td>Observed</td>
<td>132</td>
<td>4</td>
<td>12</td>
<td>62</td>
<td>36</td>
<td>8</td>
<td>14</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Expected</td>
<td>129</td>
<td>6</td>
<td>4.7</td>
<td>55.5</td>
<td>49.1</td>
<td>10.6</td>
<td>11.4</td>
<td>2.5</td>
<td>2.2</td>
<td>5.3</td>
<td>1.0</td>
<td>1.0</td>
<td>2.0</td>
</tr>
<tr>
<td>x^2</td>
<td>.07</td>
<td>.67</td>
<td>11.56</td>
<td>.76</td>
<td>3.5</td>
<td>1.9</td>
<td>.59</td>
<td>—</td>
<td>—</td>
<td>.32</td>
<td>—</td>
<td>—</td>
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</tr>
</tbody>
</table>

x^2 for phenotype prevalence with expected number >5. = 17.7, P < .001, 3 DF

*as recommended
†as used by Bencze and colleagues


CHEST, 80: 2, AUGUST, 1981 COMMUNICATIONS TO THE EDITOR 247
To the Editor:

In order to clear up the misunderstanding, it appears appropriate to supplement a few points. In 1980, almost concurrent with the release of our article in Chest, the international Pi committee published its 1978 resolution concerning the nomenclature of the \( \alpha \)-antitrypsin.\(^1\) Previously, the nomenclature of Frants and Eriksson\(^2\) was vague under the argumentation "reflecting the actual physiochemical properties of \( \alpha \)-antitrypsin on IEF . . . The most cathodal type was called \( \text{PiM}_4 \), while the intermediary type received the logical designation \( \text{PiM}_2 \)." Prior to the official recommendation of the committee, we employed the above referenced nomenclature. References to nomenclature within this communication follow the international Pi committee publication.

The methods we employed\(^3\) yielded a different distribution when compared to the results of other authors (Fig 1).

The cause of this discrepancy lies in the different distribution of the ampholyt-species upon the PAG-plate. This problem is referred to in other sources.\(^4\) With the application of the phenotype \( \text{M}_1 \), \( \text{M}_2 \) after every second specimen, the exact position of the protein bands can be ascertained. The judgment is substantiated by the differences in the \( \text{M}_4 \) lines (Fig 2). With the employment of \( \text{M}_4 \), an exact interpretation is ensured seeing that \( \text{M}_4 \) is exactly placed in the split between the double band of \( \text{M}_2 \). The applicability of the Hardy-Weinberg equation to the control group in our care is questionable. Our group was selected through repeated occupational and medical check up and the question of possible relationship between the subjects was not explained.

Table 1 represents the age distribution of subtypes of the \( \alpha \)-antitrypsin in the B group of patients with COPD. The subgroups of group B are too small to substantiate a statistical tendency. How representative the characteristics of a cohort are is dependent, among other things, upon their constellation and size. It is exactly the uncertainty in matters of heterozygotes \( \text{MZ} \) that induced us to publish our results, and on a broader basis to follow the question of predisposition of carriers of \( \text{PiM}_4 \) to COPD. It is possible

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Table 1—Distribution of Age of Patients with COPD (Group B) and PiM Subtypes

<table>
<thead>
<tr>
<th>PiM</th>
<th>Age up to (years)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>10</td>
</tr>
<tr>
<td>( \text{M}_1 )</td>
<td>1</td>
</tr>
<tr>
<td>( \text{M}_2 )</td>
<td>1</td>
</tr>
<tr>
<td>( \text{M}_3 )</td>
<td>1</td>
</tr>
<tr>
<td>( \text{M}_4 )</td>
<td>1</td>
</tr>
<tr>
<td>( \text{M}_5 )</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>1</td>
</tr>
</tbody>
</table>

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To the Editor:

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that a different distribution could have been seen with broadening of the palette of PiM subtypes with $M_i$ and $M_6$.

Independent of this, it would have been advisable to acquaint ourselves more closely with the biochemical properties of electrophoretic variants. The functional changes of the primary structure alone can hardly be the sole factor of pathologic action. A change in the tertiary structure can also be influenced by an inductive effect, for instance, the binding energy of the neuramino acid and therefore the splitting of the ester bond, and thus lead to a different biological action. An indication of this can be the split change of the Pi.

Presently we are informed concerning the structure of the pathogenic Pi variant $Z^{10}$ and $S^{10}$, as well as some of the problems of microheterogeneity.$^{11,12}$ The question of the connection between structure and biological activity is still open.

In the phenotypes V, S, T or Y, Z the Pi values are not larger than $M_i$, $M_5$, $M_6$; however, Z and S have been assumed to be pathologic.

K. Bencze, Ph.D.; and G. Fruhmann, M.D., F.C.C.P., Institut and Poliklinik Arbeitsmedizin, University of Munich, Munich, Germany

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Preoperative Evaluation of Pulmonary Function

To the Editor:

With the advent of noninvasive radionuclide methods of determining regional perfusion and ventilation, there has been increasing interest in evaluating preoperatively the patient who requires pulmonary resection. I have followed recent publications, including the one by Ali et al. (Ches 1980, 77:337-342) who have discovered that following pneumonectomy, pulmonary regional and overall pulmonary functions are relatively stable, and that following subtotal pulmonary resection, there is a decrease in pulmonary function which gradually improve with time. They state that "the quantitative evaluation of the late functional recovery has not been documented before.”

I have$^{1,2}$ shown that following pulmonary resection of seven segments or less, patients suffered restrictive changes which were almost identical, but postpneumonectomy patients, especially those who developed contralateral hyperinflation, do not demonstrate this effect.$^{1,2}$ Almost all patients recovered from restrictive effects in approximately six weeks, in a very predictable manner.

I also note that Boyens’s editorial (Ches 1980, 77:8-7), still places reliance on ventilatory tests in screening patients. We have found that ventilatory tests are of no greater help than clinical impressions in prognostication. Our continued experience supports our original findings$^3$ which indicates that mean pulmonary artery pressures correlate best with prognosis.

Thus far, scintiscanning appears to offer little more than bronchospirometry, being preferred because it is noninvasive. The data of Ali et al, indicate that the technique is not accurate enough to rely upon where subtotal resection is planned.

David V. Pecora, M.D., F.C.C.P., Chief, Surgical Service, Veterans Administration Medical and Regional Office Center, Wilmington, Delaware

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1. Pecora DV. Progressive changes in ventilation following pulmonary resection. Surg Gynec Obstet 1956; 103:455-58

To the Editor:

Dr. Pecora has commented about our discovery that following pneumonectomy, pulmonary regional and overall pulmonary functions are relatively stable. I refer Dr. Pecora to Table 3 of our paper published in Ches (1975; 68:292), which shows that we were the first group to present and publish data emphasizing the stability of regional pulmonary function following pneumonectomy.

Dr. Pecora’s quotation of our statement is incomplete. The sentence in our paper reads “Although this short term discrepancy between the physiologic and anatomic loss following partial pulmonary resection has been noticed by other investigators,$^{17}$ to our knowledge, the quantitative evaluation of the late functional recovery has not been documented.