Communications for this section will be published as space and priorities permit. The comments should not exceed 500 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.

Smoking, Carboxyhemoglobin, and Oxygen Therapy

To the Editor:

The article by Foster et al1 on therapy with supplemental oxygen in polycythemic smokers leaves several important questions unanswered.

How much carboxyhemoglobin should one accept as secondary to cigarette smoking? When we purchased a spectrophotometric oximeter (Instrumentation Laboratories CO-Oximeter), we studied 70 consecutive patients who were hospitalized for a long period in whom arterial blood gas levels were determined, and we correlated the levels with the smoking history. A good relationship to the smoking history was found, as shown in Table 1.

The levels of carboxyhemoglobin in a large group of blood donors from 18 major US cities were reported by Stewart et al2 in 1974. They also found that the upper limit for the level of carboxyhemoglobin in smokers was 10.9 percent. It seems that any level larger than this should be suspected of having an additional cause. Newly admitted patients are noted to have levels of carboxyhemoglobin that are in excess of this upper limit occasionally. Stewart et al2 point out that smokers seem to have an additive effect, with urban location, occupation, and wind speed as significant factors influencing levels of carboxyhemoglobin.

At what level of oxygen pressure (P02) should patients be maintained on therapy with supplemental oxygen? This is a very important economic and practical consideration. Since therapy with supplemental oxygen has not been observed to prolong life or prevent complications of chronic pulmonary disease, shouldn’t we use it when general medical management fails? Recent studies by Milhner et al4 have suggested that this is not usually associated with mixed venous hypoxia. The general population at an altitude such as Salt Lake City or Denver lives long lives with an arterial P02 of 65 mm Hg, such as Foster et al1 provided with therapy with supplemental oxygen. Optimal levels for maintenance therapy with oxygen are at this point somewhat arbitrary, since cardiac output and mixed venous P02 are not readily available. I would recommend that patients with stable chronic obstructive pulmonary disease be maintained at a range of P02 from 50 to 60 mm Hg, as management and symptoms warrant. Maximum therapy for chronic bronchitis will often make administration of supplemental oxygen unnecessary; however, to accomplish this improvement, one must weigh the costs of two weeks of hospitalization against the costs of continuous therapy with oxygen. It seems to me that the former is preferable to the latter from the patient’s point of view.

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Table 1—Smoking History and Levels of Carboxyhemoglobin

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of Patients (Present Study)</th>
<th>Mean Level of Carboxyhemoglobin (Range), percent</th>
<th>Stewart et al (1969-1972)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-smokers</td>
<td>17</td>
<td>1.8 (1.0-3.2)</td>
<td>1.59</td>
</tr>
<tr>
<td>Ex-smokers</td>
<td>17</td>
<td>2.5 (1.0-4.8)</td>
<td>4.17</td>
</tr>
<tr>
<td>Smokers</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1 pack per day</td>
<td>5</td>
<td>4.0 (2.4-6.1)</td>
<td>6.10</td>
</tr>
<tr>
<td>1-2 packs per day</td>
<td>21</td>
<td>5.5 (2.8-10.9)</td>
<td>...</td>
</tr>
<tr>
<td>&gt;2 packs per day</td>
<td>10</td>
<td>6.7 (4.4-10.0)</td>
<td>6.35</td>
</tr>
</tbody>
</table>

REFERENCES


To the Editor:

Mitchell asks, "How much carboxyhemoglobin should one accept as secondary to cigarette smoking?" She states that Stewart et al1 found that the upper limit for the level of carboxyhemoglobin in smokers was 10.9 percent. In reviewing the article by Stewart et al1 we noted that the 90-percent range, rather than the true range, was used. Thus, 10 percent of the cigarette smokers were outside of the given range. In addition, the level of carboxyhemoglobin was above 10.9 percent in Chicago, Milwaukee, and Miami. Thus, Stewart et al1 as well as others, have had some cigarette smokers with levels of carboxyhemoglobin above 10.9 percent. A level higher than this should not necessarily make one suspect an additional cause. It is also well known that cigar smokers have higher levels of carboxyhemoglobin than cigarette smokers, and recently a patient with a level of carboxyhemoglobin of 38 percent has been described.45 We would agree that urban atmosphere, occupation, wind speed, and other variables may be significant factors that may influence levels of carboxyhemoglobin, but undoubtedly the most significant factor is the type and amount of tobacco smoke.

The second question asks, "At what level of oxygen pressure (P02) should patients be maintained on therapy with supplemental oxygen?" Currently, we do not use an absolute level of P02, as an indication for who should receive therapy with supplemental oxygen. The patient with persistent hypoxemia and cor pulmonale or secondary polycythemia can...
be assumed to have "significant" hypoxia and, thus, is a candidate for therapy with supplemental oxygen. Our study utilized patients with hypoxemia and secondary polycythemia as their indication for therapy with supplemental oxygen. As stated in our study, all patients had first achieved maximum medical therapy before being considered for therapy with supplemental oxygen. We disagree with Mitchell's contention that therapy with supplemental oxygen has not been observed to prolong life or prevent complications of chronic pulmonary disease. Studies have shown that supplemental oxygen given in the proper circumstances can improve the quality of life, reduce the number of hospitalizations, and reduce the cost of medical care. In addition, in at least one study, such therapy was believed to have prolonged life.

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REFERENCES

Biochemical Screening for Lung Cancer

To the Editor:

In an article in Chest, Kühn et al observed that the activity of cathepsin D is increased in the fluid from bronchial lavage of patients with bronchogenic carcinoma. These investigators concluded that the activity of cathepsin D in the fluid from bronchial lavage is a useful screening test for bronchogenic carcinoma.

It is not surprising that the activity of cathepsin D is increased in patients with bronchogenic carcinoma, since this activity has been demonstrated to be six times greater in smokers than in nonsmokers. Bronchogenic carcinoma is associated with smoking. A useful screening test for bronchogenic carcinoma must be a variable that is independent from smoking.

Lee B. Berman, M.D.
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REFERENCES
1 Kühn SH, de Kock MA, Gevers W: The diagnostic value of lyosomal enzyme patterns in bronchial aspirations of patients with suspected bronchial carcinoma. Chest 74:150-159, 1978

To the Editor:

In reply to the letter of Lee B. Berman, M.D., I would like to make the following comments. When referring to the results obtained by R. R. Martin, Berman failed to point out that the concentrations of all of the acid hydrolyases (acid phosphatase, β-glucuronidase, and cathepsin D) increased as much as six-fold in the aspirates obtained from smokers, as compared to aspirates obtained from nonsmokers.

However, data in our biochemical study (Chest 74:150-156, 1978) revealed elevated concentrations of only alkaline phosphatase or cathepsin D (or both) in bronchial aspirates from patients with bronchial carcinoma. Furthermore, we reported that the activity of acid phosphatase tended to be low in such aspirates, furnishing a control to evaluate the patient.

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REFERENCE
1 Martin RR: Altered morphology and increased acid hydro-

Risk Associated with Digitalis in Respiratory Failure due to Long-Term Air-Flow Obstruction

To the Editor:

Questions have been raised concerning the efficacy and safety of therapy with digitalis in patients with pulmonary disease, notably the recent review suggesting that evidence for enhanced sensitivity to cardiac glycosides may exist in pulmonary disease. This is especially important due to the high frequency of atrial and ventricular arrhythmias now known to exist in patients with long-term air-flow obstruction while in acute respiratory failure. Because our report was one which called attention to the arrhythmias, we became curious about therapy with digitalis, its related morbidity and mortality; and we reexamined the records from our original group of 70 patients regarding therapy with digitalis.

The status regarding therapy with digitalis was identified as either receiving digitalis or not at each particular electrocardiographic entry (774) for the 70 patients. Among the entries for receiving digitalis, further subdivision was made into subgroups when the dosage of digitalis was stable, was being increased, or was being decreased. Determinations of the level of digitalis in the serum were not a common procedure at the time of the study. The causes of death among the 34 patients who died during the original study were noted, including the 28 autopsies that were performed. Testing for statistical significance and determinations of retrospective relative risks were performed according to methods previously described.

Only one of the 28 autopsies showed coronary arterial disease of such significance (70 percent or greater narrowing) that association with death was likely. Certainly, this