Table 1—Reactor Status According to Coffee Drinking Habits of 323 Men Surveyed in 1985

<table>
<thead>
<tr>
<th>Coffee drinking (cups/day)</th>
<th>%</th>
<th>OR*</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>10.24 (29)†</td>
<td>1.00</td>
</tr>
<tr>
<td>1</td>
<td>14.29 (42)</td>
<td>1.44</td>
</tr>
<tr>
<td>2</td>
<td>9.80 (51)</td>
<td>0.88</td>
</tr>
<tr>
<td>3</td>
<td>17.74 (62)</td>
<td>1.72</td>
</tr>
<tr>
<td>4</td>
<td>17.02 (47)</td>
<td>1.54</td>
</tr>
<tr>
<td>5</td>
<td>11.43 (35)</td>
<td>1.07</td>
</tr>
<tr>
<td>≥6</td>
<td>18.97 (58)</td>
<td>1.87</td>
</tr>
</tbody>
</table>

*Estimates from multiple logistic regression model. Allowance was made for age, alcohol consumption, smoking habits, and nocturnal work.
†The number of subjects in the group is given in parentheses.

Results—may be absorbed by the drinking of both beverages.* For each subject, the prevalence of asthma was determined during the three months following entry in the survey. Analysis was performed in the sub-sample of 16,220 subjects aged more than 15 years. Fourteen percent of subjects did not drink coffee or tea, 30 percent drank one, 30 percent two and 26 percent 3 or more cups per day. Mean three-month point-prevalence of bronchial asthma was estimated at 0.6 percent, which is consistent with other studies. In contrast with Pagano et al., no relation between the level of coffee or tea consumption and asthma prevalence was noticed. Compared to those who did not drink coffee or tea, the estimated odds-ratios were 1.03 for one cup, 0.86 for two, and 0.96 for three or more cups per day.

The lack of association between asthma prevalence and coffee or tea consumption might be due to the use of three-month point-prevalence. However, lifetime prevalence of bronchial asthma (8 percent of men) was not related to coffee drinking in another French sample of 323 policemen, aged 27 to 57 years, who participated in 1985 in an epidemiologic survey on risk factors of bronchial hyperresponsiveness* (4±3 cups in asthmatic subjects vs 3±3 in others, NS). Bronchial reactor status (defined as PDE<sub>20</sub> ≤ 6 mg to methacholine) (15 percent of men) was no more related to coffee consumption. Figures for the odds-ratios after taking into account age, smoking habits, alcohol consumption, and nocturnal work (possible confounders) are shown in Table 1. The result persisted when excluding the asthmatic subjects, who might drink more coffee because of this disease. The lack of relationship between coffee consumption and the objective measure of bronchial reactor status does not support the hypothesis that coffee consumption has a preventive effect on bronchial hyperresponsiveness.

The discrepancies between French and Italian data might in part be due to national differences in the coffee used in these countries like the type (blend, roast, preparation, etc) and/or the consumption habits (cup size, hours of intake, etc), which could not be taken into account in both studies. Caution is therefore necessary in assessing the possible effect of coffee on asthma from epidemiologic studies based on such simple questionnaires.

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References

To the Editor:

The discrepancy between French and Italian data, beside the reasons given by Annesi et al in their letter, can be simply explained by the play of chance. The apparent protection emerging from our data, in fact, was on the order of 10 percent for one cup per day and 20 percent for two or more cups; studies with (approximately) 100 cases of bronchial asthma (such as the French National Health Survey), or epidemiologic surveys on a lower number of cases, have only little power to detect such a moderate association. Nevertheless, we would like to stress again that the apparent protection of coffee on bronchial asthma observed in our study should at present be considered an hypothesis.

Adriano Decarli, M.D., and Carlo La Vecchia, M.D., Institute of Medical Statistics, University of Milan, Milan, Italy

Treating Asthma

To the Editor:

Jonsson and colleagues* conclude that the oral administration of methylprednisolone and theophylline is as effective as intravenous therapy in treating hospitalized patients with moderate exacerbations of asthma. However, their study result support a far different conclusion: asthmatic patients destined to recover without steroid therapy who present with near therapeutic range theophylline levels are unlikely to recover much more rapidly after continuous intravenous infusion than after oral administration of these medications.

The more methodologically sound literature on emergency treatment of asthma suggests that the lower the steroid dose, the higher the pre-treatment FEV<sub>1</sub>, and the shorter the period of observation, the more difficult it is to demonstrate a steroid benefit. Therefore, it is surprising that Jonsson et al chose a methylprednisolone dose of only 80 mg/day—substantially lower than that previously found efficacious—and did not limit their study to patients with severe airway obstruction resistant to conventional bronchodilator therapy. Even more surprising is that patients randomized to intravenous therapy did not receive a steroid bolus. The study results reflect these decisions. Subjects experienced most improvement in FEV<sub>1</sub>, in the first 24 h, long before 80 mg of methylprednisolone by continuous infusion would be expected to have an effect.* Consequently, the role of steroid administration route cannot be meaningfully assessed.

Studies of asthma therapy continue to be plagued by problems of small sample size. The current investigation is no exception. Were
the above-noted methodologic flaws not present, the finding that intravenous therapy was no more beneficial than oral medication would still leave us in the dark given the study's low statistical power to detect anything but large differences in outcome measures.8

In choosing study outcomes and subject groups, clinical investigators must respect that nearly all asthmatic patients presenting for emergency treatment eventually recover. Consequently, studies should focus on meaningful endpoints such as survival in previously intubated patients, duration of hospitalization in those admitted with severe obstruction (FEV1, 25 percent predicted), hospitalization in those presenting with moderately severe obstruction (FEV1, 40 to 50 percent predicted), and relapse in those discharged home from the emergency room. Recent studies9,10 addressing the latter two endpoints are in need of validation given their potential major impact on clinical practice. On the other hand, it is time we stopped studying change in FEV1 as an endpoint unto itself.

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REFERENCES

To the Editor:

Dr. Kern maintains that our patients were destined to recover without steroid therapy, as most asthmatic patients eventually would. Yet in his criticism of our methods he complains that we used too little methylprednisolone and did not give the IV group a bolus. He also feels that the patients responded too quickly for the steroids to have had any major impact, although the steroids were the most significant addition to therapy given the nearly-therapeutic mean theophylline levels on admission and the fact that the patients were all using inhaled or oral beta-agonists.

The above contradictions aside, I feel our major disagreement is the concept that, in order for patients with acute asthma to be hospitalized or receive steroid treatment, they must be screened in the emergency room with vigorous bronchodilator therapy. This approach to treating acute episodes of asthma has become questionable after recent studies have clearly shown the great value of steroids in improving treatment results and preventing relapses of patients thus treated.14 As a result of better understanding of asthma as an inflammatory disease, treatment results are likely to improve with a more liberal use of steroids, both short term in acute asthma and as maintenance therapy in the inhaled form.

In Iceland there have been less restrictive admission criteria for asthmatic patients and this is likely to account for the fact that our hospitalized patients were, on the average, not as severely ill as patients in some other studies. We feel that this in no way compromises our ability to draw conclusions from our data, which was carefully collected and included assessment of dyspnea and wheezing (not just spirometric data as implied by Dr. Kern). The steroid dose proved to be appropriate for the severity of disease and patients with the lowest FEV1 appeared to do quite well on either treatment program. We made no attempt to belittle the value of therapy with a larger IV dose of steroids in severe obstruction which we feel is likely of benefit but stand by our conclusion that oral administration of steroids and theophylline is just as effective as IV use in moderate disease.

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Flow Volume Loop

To the Editor:

It is with great interest that I read the article, "The Shape of the Maximum Expiratory Flow Volume Curve" by Kapp et al (Chest 1988; 94:799-806). I share their opinion that the usefulness of the flow-volume loop could further be enhanced by taking into account the configurational information contained in this relatively simple and widely used test. The difficulty is in expressing this configurational information—which is readily available by visual inspection of the shape of the flow-volume loop—in numerical indices to quantify and compare various shapes. In this respect, the proposed

![Figure: Three MEVF curves, one with a straight descending limb (---) and two curves with opposite sigmoid descending limbs (--- and ---) have been superimposed. The three MEVF curves shown, although having widely different shapes, will yield the same value for angle β because they are determined by similar values for VC, PEF and FEF50. Expiratory flow (Vex) on the y-axis, volume (V) on the x-axis.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21612/)