Role of Upper Airway Occlusion as a Predisposing Factor for Systemic Hypertension

To the Editor:

Controversy exists over the role of upper airway occlusion as a predisposing factor for systemic hypertension.\(^1\)\(^2\) In the July 1992 issue of *Chest*, Hanly and associates,\(^3\) using Doppler and twodimensional echocardiography, reported finding no abnormalities of left ventricular (LV) structure or function in snorers with or without obstructive sleep apnea (OSA), as compared with normal values. They were careful to exclude patients with hypertension from the control group (snorers without OSA) and included hypertensive patients in the OSA group. However, the duration of hypertension was not addressed, nor was the effect of antihypertensive medications. Of 11 patients with hypertension and OSA studied, 10 were treated with calcium-channel blockers, angiotensin-converting enzyme inhibitors, or beta-blockers—all of which have been shown to cause regression of LV mass index and wall thickness and improvement in LV diastolic function.\(^4\)\(^5\)

Further studies that include untreated hypertensive and normotensive patients with OSA will be needed to assess the effects of upper airway occlusion on cardiac function.

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REFERENCES

1. Parish JM, Shepard JW. Cardiovascular effects of sleep disorders.
*Chest* 1990; 97:1200-26

To the Editor:

We agree with Dr. Kosinski that the role of OSA as a risk factor for systemic hypertension remains controversial. However, our study was not designed to address this issue. We wanted to determine whether untreated OSA without daytime hypoxemia or hypcapnia impaired left ventricular (LV) function. We included 11 hypertensive OSA patients on the assumption that hypertension was part of their sleep apnea syndrome. We wanted to avoid the potential bias of excluding OSA patients who were most likely to have LV dysfunction. We agree that their antihypertensive medication may have prevented, or even corrected, LV dysfunction due to elevated blood pressure. However, we would not expect these medications to improve LV dysfunction due to OSA. Consequently, we feel that normal Doppler echocardiographic results in these patients indicate that isolated OSA does not impair LV function or induce LV hypertrophy. In addition, we reanalyzed our Doppler echocardiographic data excluding hypertensive patients from the OSA group and found no significant differences between OSA patients and nonapneic snorers.

We do not feel that further comparative studies of cardiac function between untreated hypertensive and normotensive OSA patients are likely to determine the effect of upper airway occlusion on the heart. It is already well established that untreated hypertension without OSA causes both LV dysfunction and LV hypertrophy. Consequently, we can anticipate that OSA patients with untreated hypertension will have abnormal Doppler echocardiographic findings. Such a study would be unlikely to clarify whether OSA impairs LV function since the presence of untreated hypertension is a confounding variable. We feel that more sensitive techniques to assess cardiac function are more likely to improve our understanding of the interaction between OSA and the heart.

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Postprandial Serum Cholyglycine as a Marker of Antituberculous Drug Hepatotoxicity in HIV-Infected Patients

To the Editor:

Patients with AIDS have a high incidence of adverse reactions to drugs that are commonly used in the treatment of opportunistic infections. As a result of the high rate of adverse reactions, drugs that are currently available are often limited in their use.\(^6\)\(^7\) The measurement of postprandial serum cholyglycine (CG) by radioimmunoassay is a very sensitive test in detection of hepatic alterations,\(^8\) which has been proposed for early detection of hepatic injury due to industrial products and drugs.\(^9\)

We evaluated the presence of hepatotoxicity due to isoniazid and rifampin in a group of 16 patients with HIV infection and pulmonary tuberculosis. In each patient we determined conventional hepatic measurements (glutamic oxaloacetic transaminase, glutamic pyruvic transaminase, alkaline phosphatase, and y-glutamyltransferase [GGT]) and postprandial serum CG. The study included tuberculosis patients who had no history of hepatointestinal alterations, cholecyctectomy, or exposure to hepatic toxic agents and whose analytical hepatic parameters had been within normal limits before treatment.

To compare the results with those in a group of 100 tuberculosis patients not infected with HIV. In HIV-infected patients the CG level detected a greater number of statistically significant alterations compared with transaminases and alkaline phosphatases. In the control subjects, there was a greater association between CG and alkaline phosphatase and GGT levels than between CG and transaminase levels, suggesting that hepatic alteration evidenced by bile acid determinations would be predominantly cholestatic. Cholyglycine, alkaline phosphatase, and GGT were altered more frequently in HIV-infected patients than in those not infected with HIV (Table 1), which was attributed to administration of rifampin. Also, it was necessary to suspend treatment in a large number of

Table 1—Proportion of Patients With Altered Hepatic Values During Study Period

<table>
<thead>
<tr>
<th>Altered Hepatic Value</th>
<th>Duration of Antituberculous Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 Week</td>
</tr>
<tr>
<td>SGOT</td>
<td>7/25</td>
</tr>
<tr>
<td>SGPT</td>
<td>17/25</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>10/25</td>
</tr>
<tr>
<td>GGT</td>
<td>13/37</td>
</tr>
<tr>
<td>CG</td>
<td>47/75</td>
</tr>
</tbody>
</table>

*Values are percentages of the two subgroups of subjects, expressed as HIV-negative patients/HIV-positive patients. SGOT = serum glutamic oxaloacetic transaminase; SGPT = serum glutamic pyruvic transaminase.