To the Editor:

We thank Dr. RuDusky for his comments. He raises important issues in dealing with young adults, having to do with the lack of a standardized criterion for age inclusion. Family history of premature coronary artery disease is considered positive if it was diagnosed in a first-degree relative at age 55 or younger for men, and age 65 or younger for women. As premature coronary artery disease becomes more appreciated, it will become necessary to have a consistent definition, as Dr. RuDusky suggests.

A second issue concerns the unfortunate presentation of myocardial infarction in the very young. In the very young population that Dr. RuDusky discusses, myocardial infarction may occur in the absence of angiographically proven coronary artery disease. The differential diagnosis may include thromboembolism, metabolic and endocrine disorders, illicit drug use, coagulopathies, or infections. However, there are disease processes separate from coronary artery disease, as angiography fails to reveal underlying atherosclerotic lesions. Our study deals with people with coronary artery disease in whom the clinical presentation occurred at an early age. Traditionally, it has been thought that myocardial infarctions are relatively rare at this age.

Another difference between our population and the population that Dr. RuDusky talks about is that our subjects have high rates of traditional risk factors, including hypertension, smoking, obesity, and a family history of premature coronary artery disease. Our message is that young adults with cardiovascular risk factors are at risk for early presentation of myocardial infarction. Contrary to popular belief, a normal lipoprotein profile in a young adult with risk factors does not imply freedom from coronary artery disease.

Concerning novel risk factors, we believe it is important to understand all contributing causes of coronary artery disease. However, sufficient evidence exists to support the aggressive management of all modifiable risk factors. Ironically, most of these risk factors are preventable in the first place. Yet we do poorly in controlling them.

We must not necessarily wait for new answers to start taking premature coronary heart disease seriously. A good beginning is to translate what is already known into clinical practice, focusing on elimination of all modifiable risk factors.

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Diurnal Hypercapnia in Patients With Obstructive Sleep Apnea Syndrome

To the Editor:

We read with interest the article by Akashiba et al (February 2002), which reported on their study of the determinants of chronic hypercapnia in patients with obstructive sleep apnea syndrome (OSAS). We have recently conducted a study to assess the prevalence and mechanisms of diurnal hypercapnia in patients with OSAS, and we think that our results support the findings of Akashiba et al.

We retrospectively studied the records of 175 consecutive patients in whom OSAS had been diagnosed in our center. All patients underwent anthropometric evaluations and forced spirometry using a bell spirometer with a water seal. Diurnal arterial blood gas sampling while breathing room air was obtained from the radial artery. Polysomnography was performed and interpreted following standardized procedures. Patients with an apnea-hypopnea index (AHI) ≥ 10 received a diagnosis of OSAS. COPD was diagnosed in patients with FEV1 values < 80% of the predicted value and FEV1/FVC ratios < 70%. For analyzing the data, we first classified the patients into the following two groups: those with diurnal PaCO2 ≥ 46 mm Hg (ie, hypercapnic OSAS [H-OSAS]); and those with PaCO2 < 46 mm Hg (ie, normocapnic OSAS [N-OSAS]). The main characteristics for both groups were compared, using unpaired t tests and χ2 tests, when applicable. As a second step, correlations among diurnal PaCO2 and spirometric parameters (ie, FEV1, FVC, and FEV1/FVC ratio), gasometric parameters (ie, PaO2, PaCO2, and pH), polysomnographic parameters (ie, AHI), demographic parameters (ie, age), and anthropometric parameters (ie, body mass index [BMI]) were searched for all patients, using the Pearson correlation coefficient. Finally, multiple regression analysis was performed, introducing diurnal PaCO2 as the dependent variable and those parameters that previously had been found to correlate with PaCO2 using the Pearson correlation coefficient, as independent variables. The results were expressed as the mean ± SD, unless otherwise indicated.

One hundred seventy-five patients were studied (56 men and 19 women). AHI was 42 ± 24 kg/m2. Thirteen patients (7%) were moderately obese (BMI ≥ 40 kg/m2); 22 patients (13%) had COPD, and 24 patients (14%) had diurnal hypercapnia. H-OSAS and N-OSAS differed significantly in FEV1 (64 ± 26% predicted vs 96 ± 20% predicted, respectively; p < 0.0001), FVC (70 ± 23% predicted vs 101 ± 16% predicted, respectively; p < 0.0001), BMI (35 ± 7 vs 31 ± 5 kg/m2, respectively; p = 0.002), and the percentages of patients who were morbidly obese (21% vs 4%, respectively; p = 0.0068). There were no differences between both groups regarding age, sex, FEV1/FVC ratio, AHI, or the percentage of patients with COPD. Using the Pearson correlation coefficient, PaCO2 correlated with PaO2 (r = 0.22; p < 0.0001), PaO2 (r = 0.41; p < 0.0001), FVC (r = 0.46; p < 0.0001), and BMI (r = 0.24; p < 0.0015). PaCO2 did not correlate with gender or FEV1/FVC. Correlation with AHI was weak but almost significant (r = 0.14; p = 0.053), so we decided to include AHI in the multiple regression analysis. Only FVC was found to correlate independently with PaCO2 in multiple regression analysis (p = 0.0075).

The prevalence of diurnal hypercapnia in our patients was similar to that found in other studies and was somewhat lower than the findings of Akashiba et al. Our results suggest that the main mechanism promoting chronic alveolar hyperventilation in patients with OSAS is the presence of restrictive ventilatory defects. Several reports have emphasized the association between chronic hyperventilation and heavier weight in patients with OSAS. Although BMI in our study was different in patients with H-OSAS and N-OSAS and correlated with PaO2, it was not found to be an independent predictor of hypercapnia in multiple regression analysis. This suggests (in agreement with the findings of Akashiba et al) that the association between both parameters is related to impaired ventilatory mechanics in patients who are overweight, because FVC and BMI correlated significantly in our patients (r = -0.29; p = 0.0001). In agreement with Akashiba et al, we did not find a clear association between ventilatory obstruction and PaCO2 in our patients, unlike the findings of previously reported studies. However, forced spirometry may be relatively insensitive to detecting obstructive ventilatory obstructions if it is not combined with other lung function testing methods, such as whole-body plethysmography or gas dilution methods. Therefore, we cannot definitively exclude a role for airways obstruction in the development of alveolar hyperventilation.