Gastroesophageal Reflux Common in Patients With Sleep Apnea Rather Than Snorers Without Sleep Apnea

To the Editor:

In a recent issue of CHEST (June 2002) Valipour and coworkers reported that symptomatic gastroesophageal reflux (GER) is common in subjects with a breathing sleep disorder, but that there was no difference between those with obstructive sleep apnea (OSA) and subjects who snore.

We agree with the authors that GER is common in subjects with a breathing sleep disorder. However, we do not agree with the second conclusion that the symptoms of GER are not different between a patient with OSA and a snorer without OSA.

First, because a pathologic link between GER and OSA has been suggested by us and others, the interrelationship between GER and OSA is not totally due to snoring, but primarily to sleep apneas.

Second, the authors assessed the severity of GER by the degree of GER-related symptoms alone. However, outcomes after the treatment of GERD are usually measured by symptoms, pH monitoring, and health-related quality of life. Thus, the current study did not complete the assessment of the severity of GER in both OSA patients and snorers. Furthermore, conventional GER is effectively treated by, but is not cured by, continuous positive airway pressure (CPAP). This suggests that the disease state of GER in OSA patients is not equal to that of GER alone.

Third, we have already reported that GER symptoms in OSAS patients were reversed by nasal CPAP treatment. Kerr and coworkers also reported that treatment with nasal CPAP at night can correct sleep apnea-related GER in patients with OSA. These results indicate that OSAS is more associated with the symptoms of GER than with snoring alone.

Fourth, Senior and coworkers have reported that the treatment of GER with omeprazole improves the apnea index and respiratory disturbance index in patients with obstructive sleep apnea.

Fifth, when snoring patients have a spectrum of symptoms, featuring upper airway resistance syndrome (UARS), the GER symptoms may be similar between snoring patients with UARS and patients with OSA.

Considering the points listed above, GER seems to associate with sleep apnea rather than snoring alone.

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To the Editor:

We acknowledge the comments by Teramoto et al regarding our recent article in CHEST (June 2002). We agree that there might be an interrelationship between gastroesophageal reflux (GER) and sleep-disordered breathing. Lifestyle features associated with sleep apnea, including obesity and alcohol ingestion, are also risk factors for GER, and apnea-induced transdiaphragmatic pressure swings might promote the reflux of gastric contents. However, it remains less clear whether hypoxia is related to GER episodes. While some have suggested that hypoxia induces a protective mechanism against reflux by increasing the lower esophageal sphincter tone, others observed increased GER episodes during hypoxia due to an impaired swallowing function. In our report, we did not observe a relationship between nocturnal oxygenation and the occurrence of symptomatic GER in patients with sleep apnea.

In their correspondence, Teramoto and colleagues suggested that GER is less common in patients who snore than in those who have obstructive sleep apnea. While this might be true for asymptomatic “silent” reflux episodes, it seems not be true for symptomatic GER, as our data show. However, this also might be due to the approach used in the diagnosis of GER. In our report, a validated questionnaire developed by the Mayo Clinic and designed to identify symptomatic GER, was used. We decided to use this diagnostic tool based on the following reasons: (1) pH monitoring is an invasive procedure that is reserved for atypical symptoms of GER; (2) in daily practice, the diagnosis of GER is based on the recognition of symptoms, and a response of reflux symptoms to empirical treatment is considered diagnostic, with a sensitivity for and specificity comparable with pH monitoring.
monitoring; and (3) pH recordings have some considerable limitations with poor reproduibility data.8

However, from our findings we cannot rule out that asymptomatic GER was more prevalent in patients with sleep apnea compared to patients who only snore. Only 5 of 15 patients with sleep apnea and GER, which were documented by abnormal findings of pH monitoring studies, had symptomatic GER in the study by Penzel and colleagues.9 It also might be considered that patients with a respiratory condition have a different perception of sensing reflux events compared to healthy subjects.10 If this is the case, do patients with sleep apnea and asymptomatic GER require separate GER treatment? Furthermore, if therapy with continuous positive airway pressure improves nocturnal GER in the absence of sleep apnea,11 is GER then necessarily related to apneas? Since these questions are as yet unanswered, new studies are necessary in order to investigate the link between sleep-disordered breathing and GER more thoroughly.

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Success Rates for Nortriptyline

To the Editor:

We would like to congratulate da Costa et al (August 2002)1 on extending the database on nortriptyline for tobacco dependence. However, we would like to comment on several issues regarding their study. First, we would caution against comparing results across studies. Comparing efficacy for given treatments is best done using direct comparative data. In the absence of such data, results from similarly designed studies may be compared as long as limitations of the comparison are noted.

In the present study, abstinence rates appear to be based on patient self-report without biological confirmation, which is the standard for determining efficacy in smoking cessation studies. Additionally, efficacy is reported as the 1-week quit rate at the end of treatment. No data on quit rates at other time points during treatment or on continuous quit rates are presented. The limited quit data reported and the lack of biological confirmation of quitting do not allow for an informed comparison with bupropion studies. In addition, this study employed relatively intensive group therapy administered by psychiatrists. Such therapy would be expected to elevate quit rates as opposed to quit rates in studies that used a less intensive behavioral intervention.

Regarding a separate issue, and in contrast to a statement by the authors, the cardiovascular profile for bupropion is well-established. Bupropion therapy has been evaluated in multiple depression and smoking cessation studies, and has been shown to be associated with minimal cardiovascular risk.2,3 In a study of bupropion therapy in smokers with cardiovascular illnesses,4 the safety profile was similar to that seen with bupropion therapy in a general smoking population. In the present study of nortriptyline therapy, 16% of smokers accepted into the study were excluded due to ECG alterations, which further raises questions about comparability.

In conclusion, it is inappropriate to conclude that success rates obtained with nortriptyline therapy in this study are comparable to those established with bupropion. In fact, when available evidence of efficacy and safety were reviewed by Agency for Health Care Policy and Research, only bupropion and nicotine replacement therapy were recommended as first-line treatments.5

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