made of an obstetric patient in this article.

As an intensivist who also, coincidentally, happens to deliver pregnant patients as well as having a strong interest in the care of the obstetrically ill critically ill patient, I would be willing to develop a registry of obstetric patients who develop a critical illness. Included in the data would be the category of critical care diagnosis, such as respiratory failure, eclampsia, cardiac arrest, or miscellaneous (ie, the procedures performed of a critical care nature such as intubation, Swan-Ganz catheterization, arterial line, as well as the obstetric procedures such as cesarean-section, dilatation, and curettage, etc.) Of course, demographic data such as age, race, gravidity, para, week of pregnancy, size of off-spring, maternal/fetal outcome, all would be germane to this registry.

For those readers of Chest who are interested in such a registry, you may feel free to forward discharge summaries (without names of patients if desired) with all data incorporated into it. My address is St. Mary’s FP Center, 2243 N Prospect, Milwaukee, WI 53202.

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REFERENCES


To the Editor:

I thank Dr. Scarpinato for his comments and applaud him for his interest in starting a registry. Although pulmonary artery catheters and arterial lines were placed more frequently in the pregnant patients than in the general medical ICU patients, the reasons remain speculative. My bias is that these patients are often brought to the ICU for management of respiratory failure (60 percent of our patients) and arterial lines are needed to accommodate frequent blood gas measurements. Additionally, hypertensive disorders accompanying pregnancy (20 percent of our patients) may be best managed with continuous BP monitoring via an arterial line. Pulmonary edema is seen frequently in the critically ill pregnant patients (35 percent of our patients) and placement of a pulmonary artery catheter assists in both diagnosis and management.

Although I recognize that the pregnant patient in ICU raises the anxiety level of physicians who see these patients infrequently, I feel that these patients have more procedures because of the illnesses prompting ICU admission rather than physician insecurities.

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Obstructive Sleep Apnea and Pharyngeal Wall Collapsibility

To the Editor:

Despite extensive research of the last decade, much is still to be understood in the intimate mechanisms of obstructive sleep apneas (OSA), as critically reviewed recently by Hudgel.1 In the Summary of this excellent overview, the author writes, “Intrinsic tissue abnormalities have not been shown that might be responsible for this collapsibility.”

Our attention, however, has been recently attracted by the interesting findings of Smirne et al.2 These Milanese authors found a significant difference in distribution of pharyngeal constrictor muscular fibers between four snorers and nine nonsnorers: the former had significantly more (mean ± SD 75.2 ± 2.4 percent vs 41.3 ± 9.1 percent, p<0.001) type 2a fibers, whereas type 1 and 2b were less represented. Two hypotheses were put forward to explain their findings: either a constitutionally determined reduction in slow alphamotor neurons, or an adaptation of the muscle to anatomical characteristics of the upper airway associated to habitual snoring.

As far as snoring could be considered a first step in the natural history of obstructive sleep apnea,3 it would be interesting to test the hypothesis that changes in muscle fiber distribution play a role in obstructive sleep apnea by changing the pharyngeal wall collapsibility.

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To the Editor:

The possibility of intrinsic abnormalities of respiratory muscles as a pathophysiologic mechanism in obstructive sleep apnea (OSA), brought to attention by Teculescu and Vespignani, was first raised in a study by Vincken et al in 1987.1 These investigators calculated that the tension-time index generated by the diaphragm during an obstructive apnea was above the fatigue threshold. Although their study was aimed at defining the mechanism of apnea termination, the findings stimulated the notion that respiratory muscles, including upper airway inspiratory muscles, might be fatigued, contributing to hypotonia or poor contractile function and upper airway collapse. Respiratory muscle fatigue was an attractive theory as the cause of OSA, but no data were generated to directly test this possibility in upper airway muscles until the last 2 years.

Before discussing results of physiologic studies, it is important to focus on anatomical abnormalities that have been identified in OSA. Stauffer et al2 examined the morphologic characteristics of the uvula in surgical specimens of OSA patients removed at the time of uvulopalatopharyngoplasty. Comparison was made with tissue obtained at autopsy from nonapneic individuals. In OSA, there was increased muscle and fat in the uvular tissue. These findings are consistent with the concept that if upper airway inspiratory muscles have to work excessively hard to overcome an increased collapsing pressure, muscle hypertrophy or hyperplasia might be expected. Recently, Petrof et al3 presented data showing that sternohyoides muscle biopsies taken from English bulldogs known to have obstructive apneas during sleep had more fast fibers than dogs without apneas or control muscles from the ap-
neic bulldogs. In addition, some fibrosis, muscle fiber necrosis, and inflammation were seen in the sternoglossoid muscles of the apneic dogs. As referenced by Teculescu and Vespignani, Smirne et al.4 examined medium pharyngeal constrictor muscle tissue removed from four patients with laryngeal carcinoma who had a history of heavy snoring compared with tissue removed from nine nonsnorers. There are some problems with this study. No subjects had the presence or absence of snoring or the presence or absence of OSAS verified by objective means. The snorers were significantly older than the nonsnorers, and although the subjects in general were not obese, a weight comparison between the groups was not provided. Constrictor instead of dilator muscles were studied. In spite of these design limitations, the results of this study are of interest. It was found that the medium pharyngeal constrictor muscle of snorers had a greater proportion of type 2a fibers and a lower proportion of type 1 and 2b fibers than the same muscle taken from nonsnorers. As these investigators discuss, obesity also is associated with a decrease in the fatigue-resistant type 1 fibers in muscles. Whether or not these changes are constitutional or secondary, the outcome of these changes might be to make obese individuals more susceptible to upper airway collapse in the face of increased pleural pressure necessary to overcome the decreased compliance of the obese, heavy chest wall. Type 2a fibers, however, are somewhat fatigue-resistant so that the end results of these changes is uncertain, unless tested physiologically.

Recently, information has been generated about the functional properties of normal upper airway muscles. The van Lunteren et al.13 article has shown that contraction characteristics, fiber type, and fatigue susceptibility of animal pharyngeal muscles differ among the upper airway muscles themselves and also differ from the characteristics of the diaphragm and limb muscles. Pharyngeal muscles were found to have faster contraction times than the diaphragm. This property allows contraction of the upper airway inspiratory muscles to begin and peak before the diaphragm so that the upper airway is stabilized before the inspiratory intraluminal subatmospheric pressure is generated by the chest wall inspiratory muscles. Salomone and van Lunteren found that fatigue was produced in the genioglossal muscle of anesthetized cats with two minutes of repetitive hypoglossal nerve stimulation only during severe hypoxia (arterial PaO2 < 40 mm Hg), but not during mild hypoxia or hypercapnia. The endurance of cat pharyngeal muscles was greater than that of the diaphragm,17 while in a study of rats, endurance of upper airway muscles was less than that of the diaphragm.18 Interestingly, endurance of pharyngeal muscles in elderly rats was less than that of young rats. This finding could relate to the higher prevalence of OSA in the elderly. The human genioglossus muscle was found to be fatigable, but no comparison was made with the diaphragm in these studies.11,12

Histochemical studies in animals show that pharyngeal muscles have a much lower proportion of slow-twitch, fatigue-resistance fibers than the diaphragm. In contrast, the pharyngeal muscles have a higher proportion of fast-twitch, relative fatigue-sensitive fibers than the diaphragm. As Teculescu and Vespignani point out, these intrinsic properties of pharyngeal muscles may influence the propensity for the upper airway to collapse during sleep. This theory could be tested in an animal model with chronic resistive loading studies.

In summary, new data are becoming available that were not covered in my review article that may eventually show the importance of the intrinsic properties of the upper airway respiratory muscles in the pathophysiology of OSA. Of course, the difficult question to resolve in these studies is whether the changes observed are primary or secondary, and whether in the human, upper airway histologic changes or potential inspiratory muscle fatigue actually play a part in upper airway collapse during sleep.

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Respiratory Drive in Nonhypercapnic Obese Patients With Sleep Apnea

To the Editor:

We have read with great interest the article by Gold et al1 which appeared in the May 1993, issue of Chest. They noticed that patients who are nonhypercapnic and obese with sleep apnea syndrome (SAS) had a lower ventilatory response to hypercapnia (slope V̇E/PaCO2) than patients who were nonhypercapnic and obese without SAS; hypoxic ventilatory response was not different between the groups.

At variance with the authors, concordance of these findings with the previous literature is only partial. Although the ventilatory response to hypercapnia is depressed in hypercapnic patients with SAS, it is doubtful in normocapnic SAS subjects. Lopata and Onal2 have shown normal slope V̇E/PaCO2 in normocapnic SAS patients. Studies by Rajagopal et al3 in nonhypercapnic SAS have shown normal hypercapnic ventilatory response, while White et al4 have shown a reduced ventilatory response to hypercapnia and...