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 caesarian-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, please 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 alpha motor 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 become 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 al5 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 al7 presented data showing that sternohyoideus 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-