In all fairness, we should also note that our interpretation of the study by Lever and Jaspan6 that was cited by Dr. Rosival is different from his. The authors do not seem to support the idea of giving sodium bicarbonate to patients with diabetic ketoacidosis. In their retrospective analysis of 95 episodes of severe diabetic ketoacidosis, they concluded that sodium bicarbonate might not confer any special benefits in the treatment of diabetic ketoacidosis. It is difficult to extrapolate these results to lactic acidosis since diabetic ketoacidosis is a different disease that has an established treatment aimed at the etiology, and because this retrospective study has its own limitations.

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Sleep-Disordered Breathing in Middle-Aged Adults Predicts No Significantly Higher Rates of Traffic Violations

To the Editor:

The American Medical Association7 has recommended that guidelines be developed for the driving licensure of persons who have sleep-related disorders that would be based on the extent to which those disorders contribute to traffic accidents and injuries. There is concern that regulators might attempt to restrict the driving privileges of apnea patients because of “lapses of consciousness” as defined by the “inability to respond rationally to driving privileges of apnea patients because of ‘lapses of consciousness’.”2

Kripke et al4 described a large population sample studied between 1990 and 1995, which may be the only randomly selected, ethnically representative objective survey of sleep-disordered breathing in a general US middle-aged population. Included were 190 women and 165 men between the ages of 40 and 64 years. and 65 women between the ages of 20 and 39 years. Home-based pulse oximetry data were collected to determine the incidence of 4% oxygen desaturation (ODH) events each hour during sleep. More than half the male and almost half the female participants had at least five desaturation events per hour of sleep. The prevalence of > 20 events per hour was 9.3% for men and 5.2% for women.

Following the receipt of driving records, which spanned the previous 3 years in most cases, coded violation/accident data were calculated for each subject. Citations were scored from 1 to 3, according to the severity of the violation.

The 303 volunteer records had violation scores ranging from 0 to 7, with 51 scores of > 0. The mean adjusted violation scores for groups with increasing numbers of ODH events were not consistently related to increasing the number of ODH events. Logistic regression for any violation (ie, a score of ≥ 1) showed no significant effect of logODH, when age and gender were controlled. The results of a logistic regression analysis for violation scores of ≥ 3 (n = 20) were not significant, with no predictor meeting p < 0.05 criteria. This survey demonstrated that the relationship of desaturation incidents to sleepiness is very weak,3 as other population samples have also shown.4–7 This study had 80% power to detect if the logODH increases 0.3 log units or if the risk of accident increases 76% (risk ratio, 1.76). Thus, the possibility of any strong risk in our general population can be excluded.

Young et al8 described an increased number of motor vehicle accidents in men with apnea-hypopnea index (AHI) scores of ≥ 5 but also described an anomalous protective association of AHI scores of ≥ 5 in women. There was no significant overall relationship of the odds ratio to the AHI. Aldrich9 found that there was no significantly higher self-reported accident rate among patients with sleep apnea, narcolepsy, or other disorders of excessive daytime sleepiness when compared to a control group. George et al10 found that male subjects 49.3 ± 12.7 years old (SD) who had sleep apnea (AHI score, 73 ± 29) did significantly worse than control subjects on a divided attention driving test. However, there was no relationship between the level of the AHI and poor performance, and a large number of symptomatic volunteers were as good as, or better than, control subjects.

Neither this study nor the preponderance of previous studies supports a strong risk of accidents related to sleep apnea. Although the occurrence of five or more respiratory events per hour is exceptionally common in the population, it does not indicate a driving risk as great as that of single men under the age of 25 years, which we tolerate.

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Pleurodesis and Silver Nitrate

To the Editor:

Vargas and coworkers (September 2000)1 have concluded that intrapleural silver nitrate was more effective than talc in producing a pleurodesis. Bouros and coworkers (September 2000)2 have commented that silver nitrate could become the sclerosant of choice, given its wide availability and inexpensiveness. Brock,3 in 1948, advocated the production of a chemical pleuritis by injecting into the pleural cavity, through an intercostal tube, 5 to 10 mL of a weak solution (2%) of silver nitrate followed by active suction on the drainage tube to rapidly reexpand the lung. This method was effective but had been condemned by some who maintained that it may have caused a fibrothorax.

Robert R. Shaw, MD, Professor of Thoracic Surgery during my cardiothoracic residency training at the University of Texas Southwestern Medical School at Dallas, TX, gave us his 107-page Surgery for Thoracic Disease: An Outline as a teaching guide, in 1969–1971.4 Under the tutelage of Dr. Shaw and Donald L. Paulson, MD, in Dallas, we had routinely used dry t alc poudrage on the ward via 3/4-inch thoracostomy sites, using a combination of latex chest tubes and sterilized rigid bronchoscopes, for bedside t alc poudrage. For > 30 years, I have also successfully used a 1% solution of silver nitrate, 5 to 10 mL, via a chest tube, on highly selected cases, not only for pleural effusions but for patients with prolonged air leaks.

When, rarely, dry t alc pleurodesis via video-assisted thoracic surgery or talc shurry pleurodesis for bedside use via chest tubes fails, patients have been successfully treated with silver nitrate sclerosis. The two major clinical drawbacks have been the occasional febrile response and an infrequent occurrence of a transient (< 24 h) production of a few hundred milliliters of cloudy, gray pleural fluid before pleural symphysis is achieved.

While there may be “nothing new under the sun,” I commend Vargas and coworkers1 and Bouros and coworkers2 in applying modern methods of reevaluation to old methods of effective treatment in thoracic surgical patients.

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Platypnea-Orthodeoxia Syndrome
A Probably Underestimated Syndrome?

To the Editor:

We have read with interest the observation of Acharya and Kartan (September 2000) concerning a case of platypnea and orthodeoxia because of interatrial shunting. As reported by the authors, many causes can be responsible for this syndrome despite no interatrial shunting. We would like to focus on patients with this syndrome and interatrial shunting.

In our experience, we collected 18 patients (mean ± SD age, 71 ± 6 years; range, 50 to 79 years) with similar symptoms.2 The patients had variable dyspnea and/or hypoxemia, but postural character was present in only 55% of them. In fact, this may be underestimated or difficult to diagnose when patient cannot be weaned off the ventilator! All of them had a persistent foramen ovale (PFO) and/or small atrial septal defect (ASD) with normal right-sided pressures, and symptoms occurred in only two patients after right pneumonectomy.

In our opinion, the interatrial shunting clearly results from a combination of both hemodynamic and flow phenomena. The hemodynamic phenomenon is interatrial pressure gradient favoring a right-to-left shunt across the ASD during early diastole. Mean right atrial pressure equal to or lower than the mean left atrial pressure has also been reported.3 In fact, a significant amount of blood flow from the inferior vena cava could probably cross a PFO by kinetic energy without the need of a favorable pressure gradient between the right and left atria. In the same way, decreased right ventricular compliance could produce the same effect. The second aspect is a flow phenomenon, which seems to be essential in our experience. This is well demonstrated by injection of contrast material in the inferior vena cava just at the junction, with the right atrium showing a complete filling of the left atrium through a displaced atrial septum. There are classical causes responsible for atrial septal distortion: pneumonectomy, lobectomy,4 and kyphoscoliosis. In our experience, the most frequent cause was enlargement of the aortic root, which is physiologic with age, or can be also observed with an aortic aneurysmal condition. The consequence is horizontalization of the atrial septum, placing the PFO directly on line with the blood flow from the inferior vena cava. A similar flow phenomenon can