epinephrine and dexamethasone in the immediate postbronchoscopy period. The patient did well and was discharged 24 h later. Follow-up by his private pediatrician has revealed no sequela to this episode.

Potpourri is frequently used as an air freshener, and many commercially available containers can be refilled and therefore opened by small children. Its pleasant smell, small size, and ready availability make potpourri a likely object for aspiration. Parents and physicians need to be aware that this household product is potentially hazardous.

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The Atrial Natriuretic Peptide Paradox
A Hypothesis

To the Editor:

At physiologic and pathophysiologic levels, small rises in atrial natriuretic peptide (ANP) level are often accompanied by diuresis, while constantly elevated levels in the same range are not. We would like to hypothesize that the receptor system may be responsible for this apparent paradox.

Atrial natriuretic peptide is cleared rapidly by the large number of receptors in the circulation. These receptors, which are of two types, biologically active and silent, compete with each other for ANP. Since the overwhelming majority are silent receptors, the rate of active receptor stimulation will be minimal. However, we postulate that in the dynamic setting of rapid clearance, for even a small rise in ANP level to occur, a quantum of ANP sufficient to at least temporarily saturate all the receptors must have entered the circulation. Such a quantum would not only saturate the silent receptors but also increase the rate of stimulation of active receptors. Conversely, it is suggested that constantly elevated levels do not saturate the receptors and, therefore, the rate of stimulation of active receptors is not significantly increased.

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Blunt Chest Trauma Causing Isolated Single Papillary Muscle Dysfunction and Mitral Regurgitation

To the Editor:

Isolated mitral valve injury is a rare consequence of blunt chest trauma. We report a case of a patient with single papillary muscle contusion presenting with mitral regurgitation (MR) and later with congestive heart failure (CHF).

A healthy 21-year-old man was hospitalized for high-velocity blunt chest trauma. Auscultation revealed a II/VI nonradiating systolic murmur over the mitral area. A chest radiograph showed multiple rib fractures. An echocardiogram showed normal wall motion, moderate MR, and an enlarged posterolateral papillary muscle. On the tenth hospital day the patient underwent an emergency splenectomy. Intraoperatively he developed CHF. Worsening MR and refractory CHF necessitated valve replacement. Intraoperative inspection revealed only contusion of the posterolateral papillary muscle, which was resected. Pathologic examination revealed that only its distal third was damaged, with histopathologic features typical of infarction.

In the largest review of nonpenetrating cardiac injuries, isolated papillary muscle contusion without laceration or rupture was unreported. Subsequent case reports describe either multiple papillary muscle contusions or single-muscle contusion with associated mural cardiac injury. Our case highlights isolated single papillary muscle dysfunction as a cause of mitral regurgitation after nonpenetrating chest trauma.

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Pulmonary Barotrauma in Mechanical Ventilation

To the Editor:

Pulmonary barotrauma, while not truly iatrogenic, is a consequence of our therapy for patients requiring mechanical ventilation. Anyone involved in the care of ICU patients would like to reduce its incidence. Therefore, it was with great interest that I read the report by Gammon et al,¹ which appeared in the August 1992 issue of Chest. Their ICU had an incidence of barotrauma of 24.5 percent (34/139) in patients requiring mechanical ventilation for over 24 h. All except 11 patients were ventilated in the assist-control mode. This makes their results very similar to those of Mathru et al,² who reported a 22 percent incidence of barotrauma in patients ventilated in the control mode, while only 7 percent of the patients ventilated with intermittent mandatory ventilation (IMV) suffered barotrauma. The patients in the control-mode group were ventilated at significantly lower peak inspiratory pressure (PIP) and positive end-expiratory pressure (PEEP) levels, yet still had three times the incidence of barotrauma. The data of Mathru et al suffer because they are 16 years old and because the patients on which they were based were both medical and surgical; therefore, a direct comparison cannot be made. However, the issue of whether it is the PIP itself or a high PIP combined with the rate at which PIP is attained still needs to be addressed. Gammon et al did not separate the patients on IMV. It is doubtful that the data on the small number of patients ventilated with IMV would lead to meaningful statistics, but it would be interesting to know the incidence of barotrauma in that subgroup.

Another point not addressed by either Gammon et al or Mathru et al is the timing of the barotrauma. Anecdotally, the occurrence of barotrauma increases as the patient's lungs seem to be improving. A theory to explain this phenomenon involves nonhomogeneous healing of the lung and overinflation of the healthier, more compliant lung areas by the PIP and PEEP, which leads to the barotrauma. Perhaps Gammon and his coauthors could comment on this issue.

Many theories and myths regarding barotrauma will exist until further reports like that of Gammon et al help to prove or disprove them.

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To the Editor:

We appreciate the interest of Dr. Fuhrman and agree that there are many unanswered questions regarding ventilator-associated barotrauma. We did not attempt to compare IMV and assist-control modes of ventilation due to a physician bias in our general medical ICU toward the use of assist-control ventilation in most unstable patients. The 11 patients ventilated with IMV were less ill, and the majority had nonpulmonary diagnoses; no patient with adult respiratory distress syndrome (ARDS) was ventilated with IMV. A slightly lower barotrauma rate was seen in the IMV group (111 patients with pneumothorax), but we believe this reflected the difference in patient population rather than an inherent advantage of IMV.

We were unable to correlate barotrauma risk with patient improvement, and in only one patient with ARDS did a pneumothorax occur when ventilatory pressures were trending down and gas exchange trends were improving. We agree in theory that nonhomogeneous diseases leading to high ventilatory pressures, such as ARDS, may have selective overdistension of less diseased alveoli.

A better understanding of the relationship between ventilatory variables and barotrauma is needed to allow better prediction of immediately life-threatening forms of barotrauma, as well as to devise better ventilatory strategies for high-risk patients. Hopefully, future studies will allow us to separate truth from myth.

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Controlled-Release Oral Salbutamol and Cardiac Arrhythmias in Asthmatic Patients

To the Editor:

Oral formulations of controlled-release salbutamol, used in basic treatment of asthmatic patients, produce more constant plasma levels than standard short-acting tablets of salbutamol. Therefore, controlled-release salbutamol may reduce the incidence of typical secondary effects of this pharmacologic class, above all on the cardiovascular system.¹²

We conducted a study to compare the incidence and type of cardiac arrhythmias in a group of 10 patients (5 men and 5 women) with an average age of 46.4 ± 10.7 years (range, 27 to 61 years) with stable moderate bronchial asthma before and after 6 weeks of treatment with an oral controlled-release formulation of salbutamol (6 mg twice daily). After baseline spirometric and ECG evaluation, the patients received a placebo for 2 weeks. At the end of this period the patients underwent repeat spirometry and 24-h ambulatory ECG (Holter) monitoring. Spirometry and Holter monitoring were repeated again after 6 weeks of treatment with controlled-release salbutamol. Our asthmatic patients had no coronary artery disease and did not display severe cardiac arrhythmias at presentation, and during the study they maintained their potassium plasma levels within normal range; therefore, we can exclude these factors as having considerable unfavorable consequences on the arrhythmic pattern we observed. At the basal evaluation, 70 percent of patients had hyperkinetic arrhythmias, which confirmed that these arrhythmias are a very frequent event in patients with COPD. The incidence rates of ventricular and atrial arrhythmias in our patients (60 percent and 70 percent, respectively) were similar to, although lower than, the values reported in the literature.¹³

After treatment with slow-release salbutamol, the increase in heart rate (8 percent), which occurred gradually, was not quantitatively significant and was rarely noticed by patients. Similarly, the QTc interval showed only a slight increase (2.54 percent). Average values for total supraventricular ectopy (SVE) increased slightly (5.6 percent) after treatment; however, SVE appeared in all patients.

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