obstructed, accounted for the timing, small caliber and high transmural pressures, for the character.

I advocate the term "chirping rales" for this singular finding. (One must caution against the possibility of confusing the term "chirping rales" with "chirpes," the latter designating a well-known and regrettable common disorder of amorous songbirds.) More euphonious than "squawk," the onomatopoetic "chirp" more closely emulates the actual sound; it has the sanction of prior usage1 and encompasses both auscultatory components. Chirping rales of EAA are distinguished from the findings in bronchiolitis obliterans by later occurrence during inspiration, diffuseness, clinical setting, and absence of airway obstruction.2

It is a melancholy experience for those who would popularize a sign, when they see no way to substantiate its existence by means of three, four, or six imaging techniques. Having no other recourse available, I will now, therefore, humbly propose my own thoughts (the author wishes to acknowledge with deepest gratitude the "Modest" contribution of J. Swift, D.D., to this portion of the manuscript), which I hope will not be liable to the least objection: A manually operated, sonically activated cylindrical auscultating tool (SCAT), convenient for chest scanning, may be helpful in suggesting EAA in patients with diffuse interstitial lung disease. Its many other applications in this field I omit, being studious of brevity. I profess, in the sincerity of my heart, that I have not the least personal interest in endeavoring to promote the use of stethoscopy: being the practitioner of a cognitive specialty, my emoluments are too small to finance their manufacture, and my wife is in similar exiguous circumstances.

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Undetected Superior Laryngeal Nerve Injury Presenting as Noncardiogenic Pulmonary Edema

To the Editor:

Upper airway obstruction is occasionally associated with noncardiogenic pulmonary edema.1,2 Recently we had the opportunity to see a patient in whom noncardiogenic pulmonary edema was associated with superior laryngeal nerve injury.

Four months before admission, the patient, a 16-year-old boy, was admitted to our unit with severe cranial trauma after a car accident. A computed tomographic scan showed a right parietal hemorrhagic contusion and brain edema. Intracranial hypertension was controlled with hyperventilation and barbiturates. Mechanical ventilation and orotracheal intubation were used for 10 and 13 days, respectively. The patient was extubated without problem and was discharged 7 days later with hemiparesis on the left side.

Four days before admission the patient began smoking 20 cigarettes daily. He experienced increasing cough and was brought to the emergency room in obvious respiratory distress and stridor. Ootracheal intubation was performed, and mechanical ventilation was initiated. Light vocal cord edema was appreciated at intubation. Chest radiography showed interstitial edema in the right lung. A Swan-Ganz catheter was inserted. The pulmonary artery pressure was 23/12 mm Hg, and the pulmonary capillary pressure was 7 mm Hg. Gram stain of a sputum specimen showed no polymorphonuclear leukocytes and no organisms. Cultures of blood and bronchial secretions were done, and therapy with antibiotics and bronchodilators was initiated.

Twenty-four hours later the chest radiograph was normal, and arterial blood gas values while breathing O2 at a rate of 5 L/min were as follows: Pa02, 163 mm Hg; PaCO2, 43/L mm Hg; pH, 7.39; bicarbonate, 20 mmol/L. The patient was extubated. An otolaryngologist performed a direct laryngoscopic examination and found minimal vocal cord edema. Bronchoscopy disclosed no abnormalities in the trachea and bronchial tree.

Twenty-four hours later the patient had another episode of stridor and respiratory distress. Ootracheal intubation and mechanical ventilation were again initiated. The pulmonary artery pressure was 34/15 mm Hg, and the pulmonary capillary pressure was 12 mm Hg. A chest x-ray film showed bilateral alveolar edema (Fig 1). No organisms grew in blood and sputum cultures.

Twenty-four hours later the patient was again extubated. A chest x-ray film was normal. Laryngoscopic examination was again performed, and bilateral cord paralysis was observed at phonation, suggesting superior laryngeal nerve paralysis. The patient was discharged 2 days later.

Superior laryngeal nerve injury can be associated with minimal symptoms.3 In our patient it was undetected until a laryngoscopic examination was performed at phonation. Both episodes of pulmonary edema were associated with normal pulmonary capillary pressures, and chest radiographs were normal after 24 h. Initially, the possibility of pneumonia was entertained, but the rapid chest radiographic resolution, the absence of fever and leukocytosis, and the negative sputum and blood cultures excluded that possibility. The vocal cord changes associated with increasing smoking in the previous 4 days seemed to be the precipitating factor for the upper airway obstruction. The superior laryngeal nerve is a branch of the vagus nerve supplying the motor fibers to the cricothyroid muscle.

FIGURE 1. Chest x-ray film obtained on the second day shows bilateral interstitial edema.
which maintain the tone of the vocal cords. In our patient, the cranial trauma could have produced some injury to the vagus in the brainstem nuclei or in the course through the cranial vault.

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MR Imaging in the Diagnosis of Partial Eventration of the Diaphragm

To the Editor:

Eventration of the diaphragm is characterized by elevation of that structure.1,2 Clinically, eventration is classified into complete and partial types depending on the extent of diaphragm involvement.3 In the diagnosis of right diaphragmatic partial eventration, lung or liver tumors should be differentiated. It is important to show that elevation of the diaphragm is related to local bulging of the liver. This has been diagnosed with computed tomography, ultrasonography,4 and liver scan using technetium-99m phtyate.5

We encountered a case of partial eventration of the right hemidiaphragm diagnosed by magnetic resonance (MR) imaging. The patient was a 59-year-old man without significant symptoms or abnormalities in laboratory values. On a coronal T1-weighted MR image, local supradiaphragmatic bulging of the liver was clearly shown with a hepatic vessel (Fig 1). Good visualization of hepatic vessels without the use of contrast medium is one of the benefits of MR imaging. However, the muscular component of the diaphragm was not detected. Therefore, hepatic hernia could not be completely excluded.6

In conclusion, MR imaging seemed to be the most diagnostic noninvasive imaging method for this disorder.

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Use of Amikacin and Amoxicillin-Clavulanic Acid Against Mycobacterium tuberculosis

To the Editor:

Amikacin—to the best of my knowledge first used in Hungary for the treatment of multidrug-resistant pulmonary tuberculosis1—may be combined with amoxicillin-clavulanic acid.2 To obtain more data about this drug combination, we performed checkerboard titration on Sula media.

We prepared twofold dilutions of amikacin from 0.8 μg/ml to 0.0125 μg/ml and of amoxicillin-clavulanic acid from 64 μg/ml to 1 μg/ml. We inoculated Sula media with bacterium suspensions grown on Lowenstein-Jensen media. The inocula contained $8 \times 10^6$, $6 \times 10^6$, $3 \times 10^6$, $2 \times 10^6$, and $0.8 \times 10^6$ colony-forming units. $H_3Rv$, two sensitive, and two multidrug-resistant Mycobacterium tuberculosis strains were examined. We recorded the growth of the cultures weekly from the second to the sixth week.

Since we have not seen any antagonistic effect, we believe that the combination of amikacin and amoxicillin-clavulanic acid could be used in the treatment of multidrug-resistant tuberculosis.

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