air lateral to or above the displaced lung, is not usually present on a portable supine chest film until the pneumothorax becomes large (Fig 1). A relative hyperlucency of the right or left upper quadrant, visualization of the anterior costophrenic angle, and a hyperlucency between the medial aspect of the lung and the anterior mediastinum are specific radiologic signs of a pneumothorax on a supine chest film. A posteriorly placed chest tube might not decompress the anteromedial component of the pneumothorax and this may require an anterior tube (Fig 1). Decubitus or cross-table films by visualization of the visceral pleura confirm the diagnosis of pneumothorax in doubtful situations.

If the patient described had pneumothorax associated with her pneumoperitoneum, then a well placed chest tube, at the proper time, would efficiently have resolved both the pneumothorax and the pneumoperitoneum and probably in a shorter time, thus eliminating the need to resort to more unusual solutions such as insertion of an abdominal catheter to drain the air out.

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Sudden Respiratory Arrest from Asthma

To the Editor:

Gabay's letter (Chest 1982; 82:387), which described the abrupt onset of respiratory arrest occurring within ten minutes in a previously well-controlled asthmatic patient, was both interesting and informative.

We agree with the author that the occurrence of such severe bronchospasm in a previously "healthy" asthmatic subject is possible. Moreover, we have seen five of these patients during last year, and we have failed to identify their etiologic factor(s). We wish to describe one of the patients as an example of how quickly airways can constrict.

The patient, a 59-year-old white woman who had been suffering from asthma for the last 34 years, suffered during the last two years ten sudden respiratory arrest crises which developed within minutes. She required mechanical ventilation on all occasions. Following the administration of bronchodilators and steroids, the patient usually resumed spontaneous ventilation, allowing weaning in a few hours. Indeed, two of the crises took place during hospitalization while being treated with oxygen, intravenous aminophylline and high doses of corticosteroids (60 mg/6 hours of prednisone).

As pointed out by Williams and Levin, it is possible to find patients with a relatively low degree of bronchial obstruction presenting sudden and life-threatening bronchospasm; in fact, our patient's peak flow rate, only ten minutes before her last arrest, was 180 L/min. All studies performed gave normal results except for the existence of a high bronchodilator response (+ 60 percent) and a slightly reduced FEV1, response to hypercarbia.

We agree with Dr. Gabay that airways could constrict within minutes, at least in some asthmatic patients as reflected by the high bronchodilator response test, the presence of a relatively good peak flow rate only a few minutes before the crisis, and the excellent therapeutic response.

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REFERENCE

ARDS After Local Lidocaine Administration

To the Editor:

A recent article by Howard et al (Chest 1982;81:644-5) prompted our interest in writing a brief summary of a similar case of a lidocaine-induced adult respiratory distress syndrome that occurred in a patient who presented to our emergency department approximately at the same time as their case of lidocaine-induced adult respiratory distress syndrome.

Our case involved a 19-year-old woman who came to our emergency department principally for the purpose of suturing a ¼ inch laceration of her left toe which she sustained during a heated struggle with her boyfriend. The emergency physician injected 5 ml of lidocaine (Xylocaine 1 percent) without epinephrine for local anesthesia. Five minutes after the injection, the patient complained of nausea and faintness. Ten minutes after the lidocaine injection, the patient complained of a generalized burning sensation. Blood pressure at this time was 700 mm Hg. Presuming the patient was having an anaphylactic reaction to the lidocaine, we placed her in the reverse Trendelenburg position and started two IV lines for medication and fluid administration. Crystalloid solutions, steroids, and vasopressors were administered. Auscultation of the lungs revealed wheezing and ronchi in both lung fields. Aspiration was thought to have occurred and a pharmacologic dose of 6 mg/kg of dexamethasone was given to possibly inhibit "a shock lung" situation from occurring in this patient. Dopamine was started and allowed to run wide open to affect an adequate diastolic pressure to maintain an adequate perfusion pressure to the major organ systems. This only transiently affected blood pressure and an epinephrine drip was started at a rate of 8-12 µg/min to maintain a diastolic pressure of 60-70 mm Hg. The patient lost consciousness about 30 minutes into the arrest. The anesthesiologist was called to intubate the patient. Upon intubation, a large quantity of frothy, rusty sputum was returned upon suctioning. The patient became bradycardic (20 bpm) shortly thereafter and CPR was started. Almost immediately after CPR was started, the patient had a grand mal seizure that quickly resolved without any anticonvulsant medication. Within minutes, the patient went into electromechanical dissociation at which time 2 mg of atropine was given IV push. The patient returned to a sinus tachycardia about five minutes after administration of atropine. Sodium bicarbonate (50 mEq) and epinephrine 1:10000 (1 mg) were administered every five minutes for two doses, at which time the patient became conscious and began struggling. Blood gas levels at the completion of cardiopulmonary resuscitation were pH-7.12, Pco2=46; Pco2=51; CO2=15.9; and a base deficit of 15.8. Sodium bicarbonate was given for these additional doses over the next 30 minutes which raised the pH to 7.35 with a base deficit of 5.3. Results of the initial portable chest x-ray films obtained immediately after endotracheal intubation revealed noncardiogenic pulmonary edema with more confluence of density in the left upper lung field.