The procedure may be considered by a skilled endoscopist if the patient is cooperative and airway obstruction is not imminent. Although the fiberoptic bronchoscope has the added advantage of facilitating tracheal intubation in certain circumstances, these are unlikely to apply to the patient with epiglottitis. In situations where this method of intubation is chosen, it is achieved by threading an endotracheal tube, of external diameter appropriate to the patient's airway, over the fiberscope before instrumentation is commenced and checking that it slides freely thereon. Once the oropharynx has been entered and topical anesthesia of the larynx achieved, the instrument is advanced through the larynx, thus allowing the endotracheal tube to be slipped over it into the trachea.

Fiberoptically facilitated intubation may be the approach of choice in situations where access to a normal caliber airway is difficult or hazardous using conventional laryngoscopy, but its role, if any, in the facilitation of intubation in patients with epiglottitis, is limited. In this, as in other instances of airway narrowing, there is an obvious potential for inducing complete airway obstruction during the time that the bronchoscope has been advanced through the narrowed glottis and consequently exacerbating an already perilous situation.

Review of reported cases reveals that the tracheal intubation rate, using either tracheostomy or translaryngeal intubation, is 50 percent. The mortality for epiglottitis in adults is 53 percent. Perhaps the risk of unexpected, complete airway obstruction warrants the adoption of a policy of intubation on diagnosis, as is practiced in children.

If tracheal intubation is chosen, we recommend that it should be expertly performed using conventional anesthetic techniques, with the facility for cricothyroidotomy or tracheostomy to hand. Although the fiberoptic bronchoscope offers the obvious theoretical advantages of being a combined diagnostic and, potentially, therapeutic device, it is not appropriate in the distressed patient with incipient complete airway obstruction. Finally, because of the known tenousness of the airway in epiglottitis, even without any aggravating instrumentation, it is advisable that any attempts at modifying the conventional approach to diagnosis and, in particular, to airway management should be performed cautiously and by persons both skilled at endoscopy and expert in conventional airway care.

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Recurrent Re-expansion Pulmonary Edema*

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A unique patient is described who experienced two episodes of pulmonary edema following re-expansion of two left pneumothoraces occurring months apart. Successful surgical treatment was carried out at thoracotomy. Factors regarding pathogenesis are discussed.

Unilateral development of pulmonary edema following evacuation of ipsilateral pneumothorax has generally been regarded as an uncommon occurrence. Our patient is believed to be the first of recurrent re-expansion pulmonary edema following evacuation of two successive pneumothoraces which occurred months apart in the same hemithorax. In view of previous reports implicating excessive negative intrapleural pressures in the pathogenesis of re-expansion pulmonary edema, it is emphasized that only water seal drainage in conjunction with left tube thoracostomy was used for re-expansion of the left lung prior to the second episode of pulmonary edema in the case presented. The problem of recurrent pneumothorax was successfully managed in this case by left thoracotomy with resection of an apical pleural bleb and pleurodesis.

CASE REPORT

The patient is a 23-year-old Caucasian woman hospitalized for treatment of pneumothorax. She had been well until 13 days prior to admission, when she awoke with a cough and pleuritic left-sided chest pain. Over the next three days she developed shortness of breath with exertion. Medical attention was sought because of persistence of symptoms.

On admission, examination revealed normal respirations at a rate of 24/min with the patient at rest. Auscultation revealed decreased breath sounds on the left and the chest x-ray film revealed complete collapse of the left lung without shifting of the mediastinum (Fig 1). A number 12 French chest tube was placed on the left side and connected to 20 cm of suction by means of a Pleur-evac device. No air leak was noted. Post-insertion chest x-ray examination 45 minutes later showed complete resolution of the pneumothorax. Approximately three hours following chest tube insertion, the patient became apprehensive, tachycardic (pulse rate of 112/min), and complained of shortness of breath at rest. Respiratory rate was increased 36/min with shallow, rapid excursions and the skin ashen. Blood pressure was 100/70 mm Hg. Arterial blood gas determinations showed pH 7.37, Pco2 32; Po2 44; O2 saturation, 75 percent; bicarbonate of 19 base excess, -5.7, with the patient receiving oxygen at 2L/min by nasal prongs. With institution of 100 percent oxygen by means of a nonrebreathing mask, the patient's symptoms lessened and oxygenation progressively improved. A followup chest film four hours after chest tube insertion revealed moderate opacification in the left lung zone and reconfirmed complete expansion of the left lung. Furosemide, 20 mg, was administered intravenously to promote diuresis. The chest tube was placed to water seal. Gastric distention was relieved through the use of a nasogastric tube overnight.

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Chest x-ray examination the following morning showed increased opacification of the left chest even though the patient's clinical course and arterial blood gas levels continued to improve. The chest x-ray film that afternoon showed even further opacification of the entire left hemithorax (Fig 2), with air bronchograms visualized. Blood gas levels were satisfactory on FIO2 of 40 percent, with an arterial P O2 of 86. The patient had no further dyspnea. Blood cultures gave negative findings and sputum cultures showed only normal flora. The chest tube was discontinued on the fourth day of hospitalization, after which time chest x-ray film findings were normal. The lungs were clear to auscultation. The final arterial P O2 was 88 mm Hg, with the patient on room air. The patient was discharged on the fifth hospital day, asymptomatic, ambulatory, and receiving no medication.

Twenty-eight months later, the patient was seen with a complaint of shortness of breath of seven days' duration. A small left pneumothorax was documented radiographically and progressed to complete collapse of the left lung. A No. 12 Fr chest tube was placed on the left and connected to water seal. No suction was used in re-expansion. The chest film two hours following tube thoracotomy revealed extensive opacification of the left lung, consistent with unilateral pulmonary edema (Fig 3). The patient manifested hypoxemia and mild shortness of breath, but the clinical course was not characterized by respiratory distress. Satisfactory oxygenation was achieved with administration of oxygen by mask. With the left lung fully expanded and the radiographic evidence of left pulmonary edema resolved, the chest tube was removed and the patient discharged on the sixth hospital day.

Recurrence of an approximately 40 percent pneumothorax the next day required readmission and re-insertion of a chest tube on the left. The chest tube was connected to water seal, and complete expansion occurred without clinical or radiographic evidence of left pulmonary edema. The patient underwent exploratory left thoracotomy on September 25, 1981, at which time wedge resection on an apical pulmonary bleb and pleural abrasion were performed. Histologic analysis of the specimen revealed a focal area of bullous cysts lined partly by mesothelial cells. The postoperative course was uncomplicated and the patient was discharged on the ninth postoperative day. She has done well since then, with no further respiratory difficulty.

DISCUSSION

Unilateral occurrences of pulmonary edema have been reported following evaluation of pneumothoraces1415 or pleural effusions.15161718 The case presented here is unique in
that three episodes of pneumothorax occurred in the same hemithorax. Chronicity of pneumothorax has been delineated as an important factor regarding development of re-expansion pulmonary edema following evacuation of pneumothorax, and varying durations of collapse are illustrated by the case presented. The first two episodes of pneumothorax were associated with the occurrence of re-expansion pulmonary edema. From the history it is thought that the initial pneumothorax existed for a number of days, conceivably up to ten days prior to hospitalization. The second pneumothorax, occurring months later, may have been present up to seven days prior to hospitalization. The third pneumothorax was obviously present for less than 24 hours, prior to hospital admission. Re-expansion pulmonary edema did not occur following the third pneumothorax, which was comparatively brief in duration. Duration of pneumothorax greater than 72 hours has been documented in the vast majority of reported cases of ipsilateral re-expansion pulmonary edema following pneumothorax.

A variety of other factors associated with the development of re-expansion pulmonary edema have been reported. High negative intrapleural pressure used in re-expansion has been implicated in clinical reports, as well as animal studies. In the case presented here, only 20 cm of suction were used prior to the first episode, and water seal drainage without suction was used prior to the second episode.

Rapid re-expansion may accompany the use of high negative intrapleural pressure; however, complete re-expansion may occur relatively soon following tube thoracostomy without the use of suction. In a review of 15 cases of re-expansion pulmonary edema by Brennan and Fitzgerald, in approximately half of the cases no suction was used in re-expansion.

The degree of collapse of the lung is also an important factor in ipsilateral re-expansion pulmonary edema which has followed collapse due either to pneumothorax or pleural effusion. Complete collapse of the lung has been radiographically documented preceding most of the cases of re-expansion pulmonary edema following pneumothorax.

Re-expansion pulmonary edema following thoracocentesis has generally followed evacuation of pleural effusions which were large.

From the cases reviewed and the present report, gradual re-expansion of the lung that has undergone collapse due to pneumothorax or pleural effusion appears particularly appropriate when the duration of collapse has been greater than 72 hours, and the degree of collapse is large. The use of water seal alone, with avoidance of suction, may not be sufficient in preventing re-expansion pulmonary edema following pneumothorax. Perhaps consideration should be given to intermittent clamping of the chest tube to allow for even more gradual re-expansion than afforded by the use of water seal. Re-expansion over at least several hours may be necessary.

Adding further variability to the clinical spectrum of patients in whom re-expansion pulmonary edema has been described are two cases characterized by collapse of lung presumably only several hours in duration. One case followed collapse due to pneumothorax, and the other followed collapse related to intraoperative atelectasis. Decrease in surfactant, bronchial occlusion, partial re-collapse of lung, and increased capillary permeability have all been considered in the pathogenesis of re-expansion pulmonary edema.

From the wide variety of factors involving the clinical setting, etiology, details of treatment, and degree as well as duration of lung collapse, generalizations with regard to pathogenesis of re-expansion pulmonary edema have been difficult. It is possible that individual factors, as yet undefined, may be of fundamental importance.

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