pectin exposure. His claim was accepted by the Ontario Worker's Compensation Board.

**DISCUSSION**

Pectin is a food additive that is found in the *pectinellus* of unripe fruit. Lemons, orange peel, and apple pomace are the sources commonly used for the commercial extractions. In the presence of 50 percent sugar and a low pH, pectin forms a gel in water solution and it is used as a thickening agent in the commercial production of jams and jellies. It is a hydrophilic celluloidal carbohydrate consisting chiefly of partially methylated polygalactose acid units. The molecular weights of the pectin vary between 100,000 and 200,000 kd. Breakdown of the pectins into components yields galactonic acid, methyl alcohol, and galactose.  

There has been only one previous report, to our knowledge, suggesting occupational asthma to pectin and in that case report, the skin test response was negative, unlike this patient.

We conclude that pectin should be added to the high molecular weight allergens that cause occupational asthma. Treatment includes avoidance of such products if possible in sensitive individuals. The recognition of pectin as an allergen raises the possibility of allergic responses to pectin secondary to ingested fruits, jams, or jellies. To date, this has not been reported and our patient did not report food-related allergic symptoms. The heating of pectin during preparation of jams and jellies may alter its integrity in these products. Assessment of pectin in patients with documented allergic responses to foods containing pectin may merit further investigation.

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**Delayed Pulmonary Perforation**

**A Rare Complication of Tube Thoracostomy**

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Tube thoracostomy is a standard therapy for a number of pulmonary disorders. The procedure is associated with a certain incidence of morbidity related to the technique of insertion, the patient population selected, and the length of time the tube remains in place. Complications of tube placement previously described include empyema, residual pneumothorax, lung perforation, placement of the tube in

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Insertion of a chest tube into the pleural space is an accepted treatment for a variety of pulmonary disorders. Indications include but are not limited to pneumothorax, penetrating chest injuries, hemothorax, empyema, chylothorax, bronchopleural fistula and pleurodesis. The most common complications of chest tube insertion are empyema and residual pneumothorax after chest tube removal. The technique of chest tube insertion has been shown to influence the incidence of certain complications, with the trocar insertion method being associated with a much higher incidence of perforation of the lung, diaphragm and intra-abdominal viscera. Other complications appear to occur without regard to method of tube placement but are directly related to tube location after placement, such as cardiac shock secondary to right atrial compression or Horner syndrome secondary to pressure on the inferior cervical ganglion. The following case report describes the development of a pulmonary lesion and the apparent migration of a chest tube from the pleural space into the lung parenchyma over a three-day period following chest tube insertion using the blunt dissection method for a spontaneous pneumothorax. A literature search has failed to locate a previous account of this rare complication.

**CASE REPORT**

A 68-year-old man with a history of three episodes of right-sided spontaneous pneumothorax requiring chest tube drainage at several institutions over the six months prior to presentation. His past medical history also was notable for the development of re-inflation pulmonary edema in the past following chest tube thoracotomy. One week after discharge from an outside institution where he had undergone his third chest tube evacuation of a pneumothorax, he presented to his internist complaining of general malaise and shortness of breath. Physical examination by the internist disclosed decreased breath sounds on the right side. The patient was sent to the emergency room at our institution for evaluation and treatment. Physical examination in the emergency room indicated the absence of breath sounds in the right lower lung fields and a marked decrease in breath sounds in the right upper lung fields. There were several right-sided scars from past chest tube thoracostomies, including a recently healed wound in the fourth interspace in the mid axillary line. The remainder of the physical examination was normal with the exception of guaiac-positive stool test. A chest radiograph obtained in the emergency room demonstrated a moderate pneumothorax on the right side. A No. 28 French straight thoracic catheter was placed via the fifth interspace in the anterior axillary line using the blunt dissection technique previously described. The patient tolerated the procedure well and there were no complications noted at insertion. The chest tube was connected to the Pleur-evac suction device and suction was applied at -20 cm H₂O. There was no immediate fluid drainage from the chest tube, although a large air leak was noted. The patient reported a subjective decrease in his shortness of breath, and a chest radiograph obtained

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immediately following the procedure showed complete resolution of the pneumothorax with the tube in satisfactory position (Fig 1). The patient was then admitted to the hospital for observation and management of the chest tube. His admission laboratory studies were all within normal limits with the exception of a white blood cell count of 12.4 thousand per cubic millimeter.

A follow-up chest radiograph the following day showed no evidence of pneumothorax and an infiltrate in the right lower lobe which was originally interpreted as re-inflation edema vs pneumonia. The patient remained afibrile without a change in the leukocytosis. The prior air leak was noted to have resolved. Chest tube suction was discontinued and the tube left to water seal drainage. A chest radiograph on the following day showed a small right-sided pneumothorax, and the chest tube was placed back on ~20 cm of H2O suction. That night, the patient had a spiking temperature of 38.4°C with a concomitant rise in the white blood cell count to 17,400/cu mm. The following day, the chest radiograph showed straightening of the chest tube course with an inflammatory response along the chest tube tract, along with a hydropneumothorax (Fig 2). On an empiric basis, therapy with broad-spectrum antibiotics for presumed pneumonia was started. Computer tomographic imaging of the chest cavity, previously scheduled in order to rule out bullous disease, demonstrated an intraparenchymal chest tube (not shown).

The chest tube was removed and therapy with intravenously administered antibiotics was continued. He subsequently deferred and the hydropneumothorax remained stable as the inflammatory reaction at the original chest tube site slowly resolved. He never suffered any respiratory embarrassment and was eventually discharged six days later on a regimen of oral antibiotics. At that time, the white blood cell count was 11,200/cu mm and the hydropneumothorax was resolving. Close follow-up with his internist was arranged and he has remained asymptomatic since discharge. A follow-up chest radiograph 12 days following discharge demonstrated continued resolution of both the inflammatory process and the resolving hydropneumothorax.

**DISCUSSION**

This report describes a clinical event characterized by fever, leukocytosis, and a change in the radiographic appearance of what was originally thought to be an intrapleural chest tube. An intraparenchymal position was later confirmed with CT scan. Possible explanations for the clinical and radiologic findings include initial intraparenchymal placement which was manifest in a delayed fashion or actual migration of the chest tube from the pleural space into the lung parenchyma. Although perforation of the lung is a well documented complication of chest tube insertion, the incidence using the blunt dissection technique in an adult is low. In a series of 447 patients who underwent chest tube thoracostomy, Millikan et al. report technical complications in four, only one of which consisted of damage to the lung parenchyma. Daly et al. present a similar incidence of parenchymal damage in their series: one perforation in 164 thoracostomies. Although Fraser reports an incidence of lung perforation of 11 percent in patients who underwent placement of chest tubes and subsequently came to autopsy, his results are less useful in that his total population was limited to 18 select patients whose chest tubes had been placed using the trocar technique.

In this case, the chest radiograph taken immediately post-chest tube placement demonstrates complete reexpansion of the lung with the chest tube gently curving along the chest wall. This information, along with the patient's relatively smooth clinical course until the third hospital day, supports the latter hypothesis that the original chest tube placement was intrapleural. The fact that there was a distinct clinical event during the night preceding the third hospital day and that the chest tube was observed to change in position following this event lends further credence to the interpretation that the chest tube did in fact migrate from the pleural space into the lung parenchyma. If this interpretation is correct, the mechanism of migration would be erosion of the plastic chest tube through an inflamed pleura, a complication which has not been previously described. What contribution the original position and subsequent
manipulations of the chest tube (regarding suction) made toward inducing the change in position can only be surmised, as can the importance of the patient's underlying disease. The change in position was marked by distinct clinical and radiologic changes, and the patient responded well to removal of the chest tube and therapy with broad-spectrum antibiotics.

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Pulmonary Sporotrichosis Treated With Itraconazole*

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A 62-year-old woman had chronic cavitory pulmonary sporotrichosis refractory to medical management over an 8-year period. She was treated with oral itraconazole and had an apparent microbiologic and clinical response; however, the patient succumbed to progressive pulmonary hypertension. The early use of oral itraconazole for treatment of pulmonary sporotrichosis is advocated.

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Pulmonary sporotrichosis is a rare disease. Rippon reported about 150 cases in the literature in 1988. We describe a case of chronic cavitory disease caused by pulmonary sporotrichosis that responded to itraconazole after being refractory to medical management for 8 years.

CASE REPORT

A 62-year-old woman first sought medical attention in 1982 for a 2-year history of hemoptysis and dyspnea on walking up 4 flights of stairs. Her last reported chest x-ray film in 1975 was normal, but her initial chest radiograph showed bilateral apical cavitory disease. She had smoked 2 packs per day since the age of 14 years. Her father died of pulmonary tuberculosis. A first and second strength purified protein derivative of tuberculin test was negative in 1982, and three smears for acid-fast bacilli were also negative. Bronchoscopy was performed in October 1982, and pulmonary sporotrichosis was diagnosed. Between October and December, the patient received amphotericin B in a total dose of 1,981 mg, with sputum cultures remaining positive. She was placed on therapy with a saturated solution of potassium iodide (SSKI), 75 drops per day, from 1983 until 1986 and received ketoconazole concurrently with SSKI in 1985 and 1986. Cultures of sputum remained positive, and therapy with SSKI and ketoconazole was stopped, since the patient had become permanently hypothyroid.

The patient referred herself to Brigham and Women's Hospital in 1990. At that time, she had lost 25 percent of her ideal body weight and had a chronic productive cough and low-grade fevers. Physical examination showed 4+ clubbing of the digits, a loud pulmonary component of the second heart sound, and amphoric breath sounds at both lung apaxes. The chest radiograph revealed marked apical cavitation and a nodular infiltrate (Fig 1). Chest computed tomography confirmed the fibrosis and retraction of the remaining lung (Fig 2). A culture of sputum was positive for Sporothrix schenckii.

The patient was placed on therapy with itraconazole (Janssen), 200 mg per os twice daily; and after 1 month a serum level of 4.5 μg/ml was measured 2 hours after the dose. She was treated for 12

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FIGURE 2. Computed tomogram of chest in 1990, showing large apical bullae and fibrotic scarring of remaining lung with nodular infiltrate.