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Letters to the Editor

Dear Editor

Successful fibrin glue repair of iatrogenic bronchial rupture

Bronchial rupture is a possible complication of rigid bronchoscopy, and rarely of endotracheal intubation with Carlens' and Robertshow's and even with polyvinylchloride (PVC) endotrachael tubes. Until recently surgical repair has been the treatment of choice (1,2). Fibrin glue, an agent composed of thrombin III and fibrinogen has been used to close the postoperative bronchopleural fistulas (3–5). Its use for repair of iatrogenic bronchial rupture has not been reported in the literature.

A 58-year-old male was admitted to hospital for investigation of a 2-month history of weight loss, exertional dyspnoea, haemoptysis, left back pain and a left hilar mass on his chest radiograph. Rigid bronchoscopy was performed under local anaesthesia with an 8·5 mm, 40 cm standart bronchoscope; while obtaining biopsy specimen with a rotating forceps from the tumour mass in the left upper lobe bronchus, a 5 mm horizontal rupture developed in the left main bronchus. Subcutaneous emphysema and dyspnoea developed quickly and the procedure was stopped immediately. Substernal pain, hoarseness and hypotension were the main manifestations and a chest CT showed pneumomediastinum (Fig. 1). The patient was carefully observed, and 6 h later, bronchoscopy was repeated and 0·5 ml fibrin glue (Tisseel) was directly delivered to the rupture area. Symptoms began to decrease immediately after the procedure, and his subcutaneous emphysema was gone by the 4th day. Chemotherapy and radiotherapy utilized to treat the patient's limited disease small cell lung cancer (T4N2MO, Stage IIIb). During the 9 months since the rupture, no symptoms related to bronchial leakage have been reported.

Fibrin glue was first used for repair of postoperative broncho-pleural fistulas by Hartmann and colleagues in 1977. It has been used in several cases and proved to be useful in experimental studies (6–9). The agent covers the rupture as a plug initially and then inflammation develops, proliferation of the mucosa and fibrosis occurs (10). The size of the plug should be large enough to close the rupture, but should not prevent the

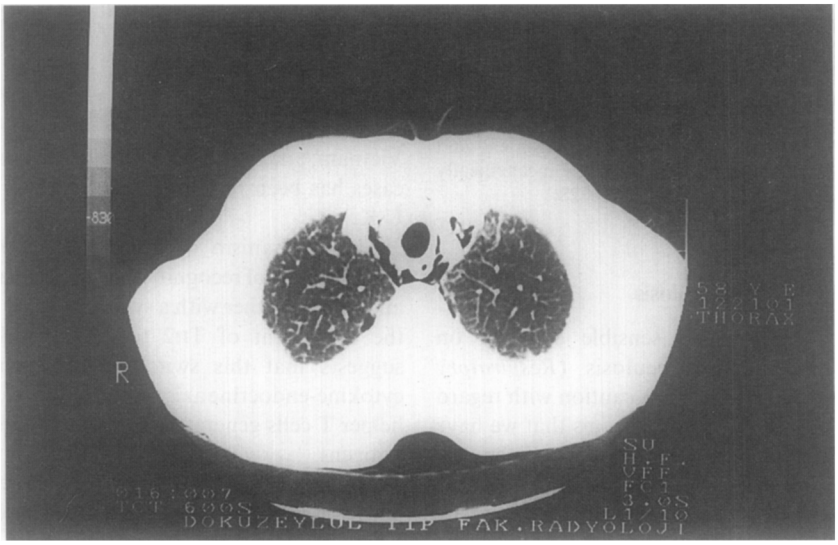

Fig 1 Computed tomogram taken 2 h after the rupture, showed pneumomediastinum.

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drainage of the bronchus. For this reason, 0·5 ml fibrin glue was administered in our case. In the following 6 months, although radiotherapy was applied to the area, no relapse was observed and this reflected the reliability of the procedure.

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Dear Editor

Chemoprophylaxis for tuberculosis

Dr Israel's sensitive and sensible editorial on chemoprophylaxis for tuberculosis (*Respiratory Medicine*, February 1993) sounds caution with regard to isoniazid (INH) and opens questions that we have been considering for some time. Although the oft-quoted American evidence suggests that a short course of INH considerably reduces cases of clinically manifest disease over the 2 years following known contact with an infectious index case, the frequently drawn conclusion that this is evidence of bacterial persistors being destroyed must be questioned. The cases prevented are likely to be those in an early stage of development of disease due to exogenous infection in persons in whom persistor bacilli have not become established. There is no reason to think that INH can prevent cases arising from endogenous activation, except during the time that the drug is being taken. If this is so, then a short course of INH in persons co-infected with HIV may prove disappointing.

Persistor status is a balance between the propensity of the tubercle bacillus to multiply and cause disease, and the ability of T helper cell mediated immunity to hold the organisms in stasis (1). We know that this balance tips in favour of the bacillus very early in HIV infection, apparently being one of the earliest immune functions lost. On theoretical grounds, arming the immune system to destroy bacilli already in stasis should be an option for prophylaxis potentially just as effective in persons infected with drug resistant bacilli as in those infected with drug susceptible organisms. Sadly, we know BCG not to be capable of this, but progress need not end there. Although we have no more than fragmentary evidence that it can actually be done, investigations into the action of immunotherapy with *Mycobacterium vaccae* for clinical tuberculosis suggest that the mechanisms induced should also be active in destroying persistor tubercle bacilli before they give rise to disease. Studies have already started to evaluate this possibility in close contacts of tuberculosis patients and may soon be applied to tuberculin positive HIV seropositive patients.

The studies upon which this proposition is based are just beginning to appear in the literature, and the evidence in support of the value of immunotherapy in the treatment of pulmonary tuberculosis, both drug-resistant and susceptible, is growing almost daily. Studies are completed, or in progress in Kuwait (2), Gambia (3), Iran (4), Nigeria (5), Romania and Vietnam, and anecdotal evidence on small numbers of cases has been received from Sweden, India and the U.K.

The mechanism of its action appears to be an enhancement of recognition of common mycobacterial antigens, together with a switch in T cell function from the equivalent of TH2 to TH1. Work in progress suggests that this switch involves changes in the cytokine-endocrine axis, which modulate the subsets of helper T cells generated in response to mycobacterial antigens.

Enhanced TH1 activity would be precisely what is needed to hold bacilli in persistance, or even to lead to their true elimination. The lack of toxicity or deleterious side effects of autoclaved *M. vaccae* (NCTC 11659) are