drug. We consider that this leaves insufficient time for reliable identification of the offending drug or drugs. We agree that the drugs should probably be re-introduced in this order.

A further difficult problem is the re-introduction of chemotherapy after an episode of acute liver failure. Certainly it is our policy, as discussed by Mitchell et al., to change to drugs with no history of hepatotoxicity in patients fortunate enough to have survived this complication.

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AUTHORS' REPLY

The editorial set out recommendations on the management of hepatic reactions after due consideration of both the risks of tuberculosis itself and the risk from the drug treatment. There have been 45 deaths from liver reactions to currently recommended first-line antituberculosis drugs since 1965, with isoniazid implicated in a maximum of 25 of these. Over the same period of time there have been 272,000 notified cases of tuberculosis (all forms), with pulmonary disease—which makes up the majority of cases—carrying an overall mortality of some 5%. The most recently published annual infectious disease statistics show 412 deaths from tuberculosis in 1994, and the level of tuberculosis deaths has been at that level for the last five years, and substantially higher in the earlier part of the period 1963–94. The risk of dying from tuberculosis is therefore clearly at least 200 times higher than that of a fatal hepatic reaction from the treatment, and inadequate treatment must intuitively raise the mortality of the disease still further.

Devlin et al accept that their recommendations that chemotherapy be withdrawn if liver transaminase activity reaches three times normal is not based on firm data. The suggestion that this at least should be withdrawn at this level does not seem logical. A large, mainly prospective, study of reactions to antituberculosis treatment showed that the incidence of hepatotoxic reactions was lowest to isoniazid at 0.3%, being appreciably higher to pyrazinamide (1.25%) and rifampicin (1.4%).

The essential difference between Devlin et al and our editorial is the “balance point” between the risks of treatment and the risks of the underlying disease. To have a level of transaminases of three times normal for modification of treatment may well be unduly harsh. Some patients with such pretreatment levels of transaminases as a result of extensive or disseminated tuberculosis who already face a significant mortality would be denied the most effective antituberculosis drugs, thus potentially increasing further their mortality from the disease. The emergence of multiple drug resistant tuberculosis, which is often due to inadequate treatment and compliance monitoring, is a further reason why standard chemotherapy should not be altered without strong justification.

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Pneumomediastinum following Politzer's manoeuvre

The report by Dr Torres-Melero and co-authors of a case of pneumomediastinum following the use of a high speed air turbine drill during a dental extraction (March 1996; $1:339–40) contains some interesting points about iatrogenic pneumomediastinum.

A 35 year old man was recently admitted as an emergency with acute severe neck and retrosternal pain, dyspnoea, vomiting, and agitation. These symptoms suddenly appeared during Politzer's manoeuvre carried out for the treatment of acousitic problems. Clinical examination showed subcutaneous emphysema in the neck and anterior chest wall with swelling around the eyes and over the cheeks. The patient had no pre-existing lung disease. Blood pressure and pulse, laboratory tests, electrocardiography and arterial blood gases were normal. Chest radiography showed pneumomediastinum, bilateral apical pneumothorax, and subcutaneous emphysema. A large quantity of air was noted in the gastrointestinal tract on the abdominal radiograph. A computed tomographic scan confirmed the presence of air in the soft tissues of the neck, extending through the mediastinum to the diaphragm, with detachment of the mediastinal pleura and the apical parietal pleura bilaterally. The lungs were not collapsed. An oesophageal contrast study was performed to exclude any lesions in the digestive tract; no abnormalities were revealed. An operator's endoscopic study found no lesions in the mucosa of the rhinopharynx. The patient was treated conservatively and his clinical condition improved within 48 hours; he was discharged well six days after admission. A follow up chest radiograph 15 days after discharge showed almost complete disappearance of the air collection.

Our case has to be considered as another cause of iatrogenic pneumomediastinum and should be added to the others previously described.

The Politzer's manoeuvre is a method of restoring the patency of the tubes in middle ear diseases. The aim of the technique is to balance the atmospheric pressure and the pressure inside the eustachian tube by insufflating air through the rhinopharynx with a closed epiglottis. Air can be insufflated manually with a peep-push or mechanically with a conveniently balanced compressor (usually no more than 2000 millibar). Although the exact mechanism of entry of air was not found in our patient, it is likely that malfunction of the machine (or an inappropriate use of the equipment) allowed the output of air at high pressure which diffused down the facial planes to the mediastinum and to the soft tissues of the neck through a small laceration of the rhinopharyngeal mucosa. This suspicion was confirmed by the massive quantity of air in the digestive tract and in the anterior extrapleural space.

Pneumomediastinum must be considered as a rare complication of the use of a jet of compressed air from different medical instruments.

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BOOK REVIEWS


This volume of over 500 pages pulls together many different strands of the anatomy, physiology, and therapeutics of the pulmonary circulation and its disorders. It is particularly strong in the evaluation of the pulmonary circulation in special environments and includes chapters by Jack Reeves on high altitude and high altitude pulmonary
Pneumomediastinum following Politzer's manoeuvre.

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