an augmented risk, because the most effective therapy cannot be used.

Although the importation of chloramphenicol-resistant *S. typhi* is undesirable it need not arouse alarm in Britain, because opportunities for the spread of typhoid in this country are negligible. Nevertheless, if epidemics caused by chloramphenicol-resistant *S. typhi* occur on a sufficiently large scale in countries of high typhoid incidence, such organisms may spread to other countries often enough to present an irksome problem, and the possibility of residual carriers of these imported strains cannot be discounted. The British cases of typhoid infected in Mexico, and the epidemic which caused them, are a warning of this, and are a reminder that if antibiotics such as chloramphenicol are to retain their efficacy for important diseases, their use should be largely if not entirely restricted to those diseases throughout the world.

**MEDICAL MEMORANDA**

**Tuberculous Ulcer of the Skin**

HAMID SAHEBJAMI, DONALD MASSARO

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The presence of a pulmonary infiltrate and an ulcerating chest wall lesion usually leads one to consider the diagnosis of actinomycosis. Tuberculous skin lesions, especially skin ulcers, are extremely unusual (Duncan, 1968; Olurin and Itayemi, 1970). When present they are usually ascribed to haematogenous dissemination from a pulmonary or extrapulmonary focus (Kleid and Rosenberg, 1970). We report the present case because of the rarity of tuberculous skin ulcers and because it suggests the possibility of contiguous spread of pleuropulmonary tuberculosis to the skin.

**Case Report**

A 58-year-old labourer was admitted to the Veterans Administration Hospital, Washington, complaining of a productive cough and weight loss for one year. Several months before admission he noted a "lump" in his right lateral chest wall which gradually enlarged and ulcerated and drained blood-tinged purulent material. He did not recall any trauma to this area. He had been exposed to heavy cement dust for 15 years and had smoked one packet of cigarettes a day for 40 years.

Physical examination showed nothing abnormal except for rales over both lungs and a round pinkish grey ulcer over the right midaxillary line at the fourth intercostal space. The ulcer was deep and indurated with raised edges and had vegetations and crusts (Fig. 1).

A chest roentgenogram showed bilateral infiltration and cavitation and pleural thickening over the right lateral chest. His intermediate-strength purified protein derivative was positive and his sputum contained acid-fast bacilli on smear and grew *Mycobacterium tuberculosis* on culture. Biopsy of the chest wall ulcer showed a caseating granulomatous abscess (Fig. 2). Special tissue stains did not show acid-fast bacilli or fungi. Serological studies and sputum cultures for fungi were negative.

He was treated with isoniazid, para-aminosalicylic acid, and streptomycin. The skin ulcer healed after six weeks of therapy and his sputum became negative for *M. tuberculosis* after two months of therapy.

**Comment**

The major purpose of this report is simply to call attention to the presence of tuberculous skin lesions. In the present

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**References**


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The pathogenesis of the skin ulcer in this case is not clear. Most extrapulmonary tuberculosis represents haematogenous spread from a primary focus in the lung. Tuberculous lesions of the skin were once thought to arise from this lymphatic spread along the path of an intercostal nerve tracking out at the lateral branch. Two recent cases of tuberculosis involving the skin of the chest at the site of blunt trauma were attributed to silent primary tuberculosis and bacilaemia (Stead and Bates, 1971). In these cases the only clinical evidence of pulmonary involvement was hilar adenopathy, there being no roentgen manifestations of parenchymal or pulmonary involvement. The present patient had cavitary tuberculosis including pronounced pleural thickening immediately subjacent to the tuberculous skin ulcer. For this reason we suggest that this skin ulcer might have been caused by contiguous spread from the pleura.

References
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Ethambutol and a False-positive Screening Test for Phaeochromocytoma

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Case Report
A 46-year-old woman presented with renal tuberculosis. Urogenital tuberculosis was first diagnosed in January 1968, but despite chemotherapy and left nephrectomy tubercle bacilli continued to grow from her urine. On examination in February 1971 she weighed 55 kg and her blood pressure was 130/80 mm Hg. The blood urea was 46-60 mg/100 ml, and the creatinine clearance 40-48 ml/min. Tubercle bacilli recovered from the urine were sensitive to streptomycin, para-aminosalicylic acid, ethambutol, and rifampicin and highly resistant to isoniazid. Treatment was begun with streptomycin 500 mg, ethambutol 1,400 mg (25 mg/kg body weight), and rifampicin 600 mg daily. In May the ethambutol was reduced to 800 mg daily (15 mg/kg body weight). The blood pressure was 140/80 mm Hg. Streptomycin was discontinued in July owing to a persistent rash.

In August the blood pressure was found to be persistently raised at 170/120 mm Hg and a retinal haemorrhage was present. No history suggestive of a phaeochromocytoma was obtained. Renal function was unchanged. The hypertension was investigated. Phentolamine (Rogitine) 5 mg intravenously produced a marked depressor effect lasting more than 30 minutes (see Fig.). Glucagon provocation test negative, plasma volume 2.7 litres, total 24-hour exchangeable sodium 2,228 mEq (normal for weight 2,250 mEq), maximum urine osmolality after intramuscular vasopressin (Pitressin) 508 mosm/L, maximum urine pH after ammonium chloride loading (1 mg/kg body weight) 5.4, vanillomandelic acid excretion on two occasions not increased, plasma adrenaline, noradrenaline, and catecholamines within normal limits. After these investigations ethambutol and rifampicin were withdrawn. Over a two-week period the phentolamine test became negative. The hypertension was then controlled with guanethidine given by mouth.

Six weeks later, after temporary withdrawal of the guanethidine, the phentolamine test remained negative, but three days after reinstituting treatment with ethambutol 800 mg and para-aminosalicylic acid 12 g daily a depressor response to intravenous phentolamine was found. The drugs were withdrawn and after two weeks the phentolamine test was again negative. Para-aminosalicylic acid combined with rifampicin 600 mg/day did not result in a positive phentolamine test. When ethambutol was added to this drug regimen the phentolamine test became positive within three days.

Comment
False-positive phentolamine tests in uraemia are well known (Sheps et al., 1966), but although this patient had a damaged kidney her blood urea did not rise above 60 mg/100 ml over the whole period of observation. A phaeochromocytoma is a difficult tumour to exclude and may be present in the absence of raised catecholamine excretion (Litchfield and Peart, 1956). In this patient the timing of positive phentolamine tests in relation to ethambutol administration and the normal plasma adrenaline and noradrenaline levels (taken immediately before a positive phentolamine test) made the existence of a chromaffin cell tumour most unlikely.

Ethambutol excretion is about 80% through the kidneys (Place and Thomas, 1963). The plasma concentration in this patient 24 hours after 800 mg (15 mg/kg body weight) was 2.1 μg/ml, implying impaired excretion (Strauss and Erhardt, 1970).

It is assumed that ethambutol in high concentration reacts in some way with phentolamine. The nature of the interaction is obscure.