Table 1. Speed of growth of mycobacteria and their inhibition by nicotine

Species	First appearance of growth on control medium (days)	Minimal inhibitory concentration of nicotine (mg/l)
M. tuberculosis, 12646	7	800
M. tuberculosis, 79499	7	800
M. tuberculosis, 79665	7	900
M. tuberculosis, 6558	7	700
M. tuberculosis, 6872	7	800
M. tuberculosis, H37Rv	7	900
M. tuberculosis, B1453	10	1000
M. tuberculosis, 34625	7	600
M. bovis, BCG	10	700
M. bovis, human isolate	7	600
M. microti	14	700
M. avium, NCTC 8559	7	800
M. intracellulare NCTC 10425	7	800
M. scrofulaceum, NCTC 10803	4	300
M. kansasii, NCTC 10268	7	800
M. marinum, NCTC 2275	7	1000
M. xenopi, NCTC 10042	14	600
M. flavescens, NCTC 10271	7	>1000
M. fortuitum, NCTC 10394	4	>1000

Grange *et al.* (1978) (6558 & H37Rv), and Jackett *et al.* (1978) (B1453); while one (6872) is a Teheran strain of 'phage type B included in the study of Goren *et al.* (1982), and another (34625) is a wild strain recently isolated from a Hong Kong patient. These strains were grown in 7H9 Tween-albumin liquid medium (Difco Laboratories) and an inoculum of 10 μl of a fully grown culture was added to each of 2 sets of slopes of Lowenstein – Jensen medium without potato starch (Cruikshank *et al.* 1975). One set contained 0, 1, 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900 and 1000 mg/l nicotine (Sigma Chemical Company Limited), and the other set the same concentrations of a preparation of nicotine sulphate (Sigma Chemical Company Limited), which consisted of 45% nicotine base and 55% nicotine sulphate. The slopes were incubated at 37°C and were read at 4, 7, 10 and 14 days. Table 1 shows the day on which growth first became clearly apparent and the minimal inhibitory concentration of nicotine. No stimulation of the rate or amount of growth of any strain occurred on slopes containing subinhibitory concentrations of nicotine or nicotine sulphate.

It is remarkable that our strains of *M. tuberculosis* had usually grown at 7 days and had all grown at 10 days of incubation, whereas Kotian *et al.* reported that growth on their medium without nicotine took 28 days. While nicotine clearly has no stimulatory action on the growth of mycobacteria on good culture media, the possibility that it could act as a nitrogen source in medium with deficient or absent asparagine might be explored.

B W ALLEN
D A MITCHISON
29 July 1983

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Discovery of Horner's syndrome

From Dr John A Ross
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Dear Sir, Dr Rajni Amin's letter concerning the 'newly recognized syndrome in the neck' (July *Journal*, p 621) prompts me to point out that there is another claimant to the discovery of Horner's syndrome – namely John Reid (1809–1849), Chandos Professor of Anatomy at St Andrews. He described it in 1839, thirty years before Horner. I have made a brief reference to his career elsewhere (Ross 1981).

Yours faithfully

JOHN A ROSS
25 July 1983

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Pure red-cell aplasia secondary to angioimmunoblastic lymphadenopathy

From Dr M Al Hilali and Dr M V Joyner
Department of Haematology
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Sir, Angioimmunoblastic lymphadenopathy with dysproteinaemia (AILD) had been described in association with a number of haematological abnormalities (Pangalis *et al.* 1978). We have recently treated a classical AILD who presented with a pure red-cell aplasia.

A 79-year-old woman was admitted with a three-week history of anorexia, weight loss, nausea and vomiting, severe night sweats and the appearance of lumps in her neck. On examination she was clinically anaemic, there was marked

generalized tender lymphadenopathy and splenomegaly. Investigations on admission revealed a haemoglobin of 6.5 g/dl, MCV 85, total white count 18.0×10^9/l (27% immunoblasts, 66% polymorphs, 4% lymphocytes), platelet count 149×10^9/l, ESR greater than 140. Direct Coomb's test was strongly positive (IgG+++, C3C4++, C3+++, C4±, broad spectrum+++), reticulocytes less than 1%. Protein electrophoresis showed a diffuse increase in gammaglobulins and immunoglobulin quantitation revealed an IgG of 33 g/l, IgA 7.5 g/l, IgM 4.8 g/l. Peripheral blood lymphocyte markers showed 5% E rosettes and 88% SmIg-bearing cells. Biochemical analysis showed a raised blood urea (13.4 mmol/l) and raised urate (0.698 mmol/l). Left axillary node biopsy was performed which showed the classical appearance of AILD.

Bone marrow aspirate showed normal cellularity, normal thrombopoiesis and granulopoiesis, but no red-cell precursors were identified. There was a marked lymphoid infiltrate by a spectrum of cells including plasma cells, plasmacytoid lymphocytes and numerous immunoblasts. Bone marrow trephine revealed increased cellularity and confirmed the aspirate appearances. Diagnosis of AILD in association with red-cell aplasia was made and following transfusion she was started on prednisone 60 mg/daily. This resulted in marked clinical improvement which was maintained for 13 months on 5–15 mg of prednisone daily. Recent recurrence of symptoms and lymphadenopathy was resistant to increased steroids and she died 14 months after presentation. The red-cell aplasia did not recur during this period.

Abnormalities of the blood with a demonstrable autoimmune basis are confined to autoimmune haemolytic anaemia which rarely, however, explains the severe anaemia commonly found in this condition. Our patient, with a morphological picture of a pure red-cell aplasia with a strongly-positive direct antiglobulin test, lends support to the concept that intramedullary haemolysis, in this case including red cell precursors, may contribute significantly to the anaemia (Pangalis *et al.* 1978). The pure red-cell aplasia in our patient may be another manifestation of an automimmune process, in this instance directed against immature and mature erythroid cells. Such an immunological basis has been suggested in pure red-cell aplasia of idiopathic type (Krantz & Kao 1969).

M M AL HILALI
M V JOYNER
27 July 1983

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Book reviews

Psychosurgery: A Scientific Analysis
M A J O'Callaghan & D Carroll
pp 332 £24.95 Lancaster: MTP Press 1982

'I follow a simple prescription, generally, for deciding on all elective medical procedures', declared the neurophysiologist Paul MacLean in an address to the National Institute of Health in the United States. 'First, I ask, is this a procedure I would want done to a friend? If the answer is yes, I ask myself is this a procedure I would want done to a member of my family – my mother, my wife, my child? And if the answer is still yes, I ask is this something I would want done to myself?'

MacLean's eminently sensible remarks are quoted in this very thorough and immensely readable review of one of the most controversial of modern psychiatric treatments. As the historical introduction in the book reminds us, surgery applied to the head of a living human being to relieve mental as well as physical disease has been carried out as far back as prehistoric times. During the 1940s and 1950s, with relatively crude models of brain function upon which to rely, surgeons embarked on what has become known as the first phase of modern psychosurgery, frontal lobotomy being the main procedure favoured for a variety of psychiatric conditions including schizophrenia and chronic depression. The second phase, otherwise rather euphemistically referred to as the 'renaissance' of psychosurgery, is allegedly taking place at the present time, fostered by the development of refined surgical techniques as well as by innovations such as the destruction of brain tissue by