Einstein's assertion that God does not play dice with the universe, his successors, such as Bohr and Heisenberg, became convinced that there is a basic uncertainty or indeterminancy in the behaviour of matter. Thus the universe may owe its present form to an extraordinary blend of chance and intention.

The origin of life and the ultimate arising of beings conscious of themselves and their Maker may likewise have resulted from an intrinsic property of matter imposed upon it in the original act of creation, and may thus have been as inevitable as the formation of the stars and planets. On the other hand, the place where life first arose, either planetary or celestial, and the subsequent processes of evolution may be subject to the same principles of uncertainty and indeterminancy as those affecting the evolution of the inorganic universe.

In addition to reconciling the doctrines of evolution and creation, this concept accounts for the flaws and imperfections and their attendant sufferings in nature despite the perfection and omnipotence of its Creator, and also the existence of free will within a determined universe. Furthermore, it is compatible with the existentialist concept of the randomness of our own creation and being – our 'thrown-ness' or *Geworfenheit,* as Heidegger termed it. The view expounded here does not, however, exclude the possibility of divine intervention within the created universe. Such an exclusion would deny the power of prayer and the Church's healing ministry.

Yours faithfully

JOHN M GRANGE

18 July 1983

**Contemporary problems in philosophy**

*From Professor Sir Alfred Ayer*

*London W1*

Sir, Mr Wright (September *Journal,* p 798) has misunderstood me. In criticizing the doctrine of holism, I was not denying that Popper's criterion of falsifiability corresponded to scientific practice. All I was maintaining was that a given experiment puts only a section of our beliefs at risk, not the whole corpus. In other words, we test only a limited number of hypotheses at any one time. This is surely obvious.

Incidentally, Popper's theory is not altogether new. It was anticipated in the 19th century both by William Whewell and by the American pragmatist, C S Peirce.

Yours faithfully

A J AYER

27 July 1983

**Back pain research**

*From Dr John W Todd*

*Farnham, Surrey*

Sir, I am puzzled by Dr J A Mathews' statement (May *Journal,* p 343) 'that studies of empirical methods of treatment are of value if carefully controlled'. An empirical method of treatment is one based not on theory but on 'the results of observation and experiment only' (Shorter Oxford Dictionary). The only satisfactory way of obtaining empirical evidence about the value of treatment for a condition so difficult to assess as back pain is by 'carefully controlled' studies. When Dr Mathews refers to an empirical method of treatment, does he not mean its antithesis – i.e. a method of treatment based on theory?

Yours faithfully

JOHN W TODD

7 June 1983

A copy of this letter was sent to Dr Mathews, whose reply follows:

Dear Sir, The definition of 'empirical' that Dr Todd quotes from the Shorter Oxford English Dictionary is the first-mentioned of four and is related to medicine. I willingly agree with his use of it in this way. The second definition, however, is 'That practises physic or surgery without scientific knowledge', and it is in this sense that I used the word and thus entreat workers to further study of *ad hoc* treatment methods.

When Dr Todd upbraided me on this same point in 1976, he enclosed an excellent reprint of his paper 'Plain Words in Medicine' (1964, *Lancet* ii, 1258–1259) which I commend to your readers. I am most grateful to him for keeping a watchful eye on my grammar and envy his abilities as a purist.

Yours faithfully

J A MATHEWS

21 July 1983

**Nicotine and mycobacteria**

*From Mr B W Allen and Professor D A Mitchison*

*MRC Unit for Laboratory Studies of Tuberculosis*

*Royal Postgraduate Medical School, London*

Sir, Kotian, Shivananda and Rao reported in your June issue (p 530) that the speed of growth of *Mycobacterium tuberculosis* and other mycobacterial strains on Lowenstein – Jensen was increased by the addition of tobacco dust extract and by pure nicotine, particularly at 50 μg/ml.

We have attempted to reproduce their findings using 19 strains of mycobacteria (Table 1), including 8 strains of *M. tuberculosis* of varying phage type, guinea-pig virulence and lipid content. Of these, 6 have been described by Mitchison *et al.* (1963) (12646, 79499 & 79665),

*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 $\mu$l 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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**Mitchison D A, Selkon J B & Lloyd J**
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## Discovery of Horner's syndrome

*From Dr John A Ross*
*Frankby, Merseyside*

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*
*Royal Devon and Exeter Hospitals*

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