DEATH DURING THERAPEUTIC STARVATION

SIR,—The fatal outcome in 2 out of 12 patients treated by Dr. Spencer [1] by a fasting regimen may be an unfortunate coincidence, but the events could conceivably have been precipitated by other factors which may be avoidable.

I have treated close to 150 subjects by extended fast periods lasting from two to four months. Weight-losses usually ranged around 100 lb. (45 kg.). No serious cardiac complications were encountered. 1 patient experienced a transient episode of supraventricular tachycardia.

Fasting in itself produces a pronounced diuresis of water, potassium, sodium, magnesium, and calcium. Fasting patients and others on severely restricted diets have been shown to be particularly sensitive to the action of diuretics, responding with large sodium and potassium losses.[2] The additive effects of fasting plus the diuretic may result in depletion of tissue-potassium. Plasma-levels generally do not reflect this, as has been noted with fasting,[3] as well as other instances of long-continued, gradual mineral loss. I have found the use of diuretics unnecessary and hazardous. The normal potassium supplements do not prevent a negative balance in the first two to three weeks of fasting. If given as enteric-coated tablets, the potassium is often not absorbed.

The administration of digitalis in grossly obese subjects with pronounced œdema is often of questionable value. Even proven congestive failure, if the result of the Pickwickian syndrome, does not respond well to digitalis alone, but seems to improve without it with rapid weight-loss and diuresis of fasting. Digitalis, in the presence of an unsuspected mineral depletion of tissue, may more readily precipitate arrhythmias. I have avoided the use of digitalis in these patients whenever possible. The persistence of brawny, hard œdema in the legs over long periods is not a sign of persistent failure and tends to clear slowly with progressive weight-loss.

Wadsworth Hospital, V.A. Center,
Los Angeles, California 90073, and
U.C.L.A. School of Medicine,
Los Angeles, California 90024. ERNST J. DRENICK.

AUTOIMMUNE DISEASE IN NIGERIANS

SIR,—In his letter last week (p. 513) Dr. Shrank challenges my preliminary report of the infrequent occurrence of autoimmune diseases in Nigerians. Whilst agreeing with Dr. Shrank that considerable caution must be applied in the use of hospital data as an indication of disease prevalence, I believe that the figures obtained for rheumatoid arthritis are sufficiently striking to suggest strongly that clinical rheumatoid arthritis does not occur as frequently in Nigerians as in Caucasians. Dr. Shrank suggests that my hospital figures are invalidated by the fact that large numbers of young people with acute infectious diseases are admitted to hospital in Nigeria but not to hospital in England and Wales. He will, however, have noted from the table in my paper (Aug. 17, p. 380) that the difference between the number of observed and expected admissions with rheumatoid arthritis is most striking among patients aged 45–64 years, who are less often admitted to hospital with acute infectious diseases. For female patients in this age-group, rheumatoid arthritis accounts for 70 times as many admissions to hospital in England and Wales as to University College Hospital, Ibadan. Full details of the population survey, which supports the view that clinical rheumatoid arthritis is not common in Nigerians, will be published in due course.

Dr. Shrank criticises my use of rheumatoid arthritis as a marker for autoimmune disease. My study also includes data showing that Hashimoto's thyroiditis, thyrotoxicosis, myxœdema, and pernicious anæmia are all rarely seen in hospital practice in Ibadan. Taylor [4] has presented strong evidence that autoimmune thyroid disease is uncommon in Ibadan.

Dr. Shrank rightly points out that discoid lupus erythematosus is sometimes seen in the outpatient clinics at University College Hospital, Ibadan. Nevertheless the systemic form of the disease, in which immunological abnormalities are a much more prominent feature, is a very rare cause of admission to this hospital. The situation is perhaps analogous to my findings (to be published) that when rheumatoid arthritis does occur in Nigerians the disease is usually mild, and that patients with the disease rarely develop rheumatoid factor, nodules, or vascular lesions. Both these findings suggest that autoimmune phenomena are uncommon in Nigerians.

M.R.C. Rheumatism Research Unit,
Canadian Red Cross Memorial Hospital,
Taplow, Maidenhead, Berks. B. M. GREENWOOD.

RADIATION DOSE IN ISOTOPE ENCEPHALOGRAPHY

SIR,—In recent issues of *The Lancet* estimates of the radiation dose to the central nervous system following intrathecal injection of 100 μC of ^{131}I-labelled human serum-albumin (^{131}I H.S.A.) have been given by Bannister,[1] Sear and Cohen,[2] Bull,[3] Brocklehurst,[4] and McAlister.[5] The values range from approximately 1 rad to 100 rads, with a value of 600 rads estimated for an extremely abnormal situation. Presumably these values refer to the average dose to the central nervous system, in particular to the spinal cord. Unfortunately, none of these workers has clearly presented the anatomical and physiological features relevant to the calculation of radiation dose.

By using a formula which gives the radiation dose to negligibly thin structures within a volume of radioactive solution, Sear and Cohen [2] calculated the radiation dose to the spinal cord. They essentially assumed that the spinal cord was negligibly thin compared with the half-absorption thickness (approximately 0·02 cm. in tissue) of the β radiation from ^{131}I. In fact, the spinal cord is a column of nervous matter of about 1 cm. average diameter [6] covered by a thin protective layer of tissue. It is positioned within a sheath of the arachnoid mater within the vertebral column. Cerebrospinal fluid (C.S.F.) fills the space between the spinal cord and the arachnoid mater. Nerve-roots, each consisting of bundles of nerve-fibres, span the gap between the spinal cord and arachnoid mater at intervals throughout the entire length of the spinal cord. The volume of the spinal subarachnoid fluid has been given as 90 ml. (Lindgren [7]), 30 ml. (Weston [8]), and 75 ml. (Lups and Haan [9]). For a given patient the volume of spinal subarachnoid fluid is, therefore, likely to be somewhere in the range 30–90 ml. It would be useful to estimate the radiation dose to the spinal cord for this range of volumes.

It is clear at once from the geometry of the situation that the β-radiation dose to the centre of the spinal cord is zero, since the maximum range of the predominant 0·61 MeV β-radiations from ^{131}I is 0·21 cm. (assuming that negligible amounts of ^{131}I reach the central canal of the cord, since the tracer does not normally reach the ventricular regions). At the surface of the cord, the radiation dose depends on the amount of absorption due to the protective layer. Part of this layer consists of a fine network of blood-vessels and fibres, perhaps with C.S.F. filling the interstices. Therefore, in the following calculations, the extreme cases, when the effective thickness is zero and when it is equal to the actual thickness of the layer, will be considered. The radiation dose to the nerve-roots depends on the thickness of the filaments of which they consist. If a filament is negligibly thin, the radiation dose will be given by the formula used by Sear and Cohen.[2] The same applies to any other thin nerve-fibres surrounded by C.S.F.

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