are other surveys, not quoted in their paper, of radiologically detected lesions of the proximal part of the alimentary tract which are mainly from the developing countries of East Africa⁷ and the West Indies⁸ and also from Scotland³ and South Australia.⁹ Although these surveys do not cover a population as well defined as that of the North-east of Scotland they were made on unselected patients who presented for barium swallow and meal examination in the main teaching hospital in the area concerned, and, in the case of the East African series, the only local hospital at that time which provided a comprehensive radiological service. Therefore, with some limitations, these series are, at least so far as disease of the oesophagus, stomach, and duodenum are concerned, comparable with the survey from the North-east of Scotland. The importance of the comparison of surveys of this type is related to the geographical aspects of pathology, the study of which may provide useful information on the aetiology of disease.—I am, etc.,

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Anticonvulsant Hypocalcaemia

SIR,—Dr. E. H. Reynolds (10 June, p. 656) and Dr. N. M. A. Viukari and others (24 June, p. 768) have argued that factors other than enzyme-induction must be involved in the aetiology of anticonvulsant hypocalcaemia and folate deficiency. They pointed out that phenytoin appears to have a more powerful effect than phenobarbitone on calcium metabolism¹ and folate metabolism,² whereas phenobarbitone is the more potent enzyme-inducer. This latter conclusion, however, is based upon experiments performed in animals (mainly rats) rather than man. The rat liver metabolizes drugs at a very different rate from human liver, and inferences about the enzyme-inducing properties of these drugs in man must be made with caution. Unfortunately, there is no good evidence available from human experiments.

Our suggestion that enzyme-induction may accelerate the metabolism of vitamin D has received firm support from studies in animals^{3,4} and in man⁴ of the rate of conversion of cholecalciferol to its hydroxylated metabolites. In our experience, anticonvulsant hypocalcaemia and osteomalacia responds promptly to administration of 25-hydroxycholecalciferol or whole-body ultraviolet light treatment. None of these observations can be explained by the alternative theories proposed by your correspondents, such as displacement of calcium from its protein-binding sites, calcium malabsorption, or some unspecified influence of folate-

deficiency on bone metabolism. (Incidentally, two of our osteomalacic patients, one of whom has previously been reported,⁵ had been receiving folic acid supplements for some months before their bone disease was discovered.)

None of our patients has shown any improvement in their epilepsy when their serum calcium levels were corrected. We consider that the disturbance of calcium metabolism is merely an occasional nuisance produced by long-term anticonvulsant treatment, and is not a fundamental mechanism by which these drugs may control seizure discharges in the brain. Folic acid deficiency appears to fall into the same category, for other than occasional macrocytosis and rare megaloblastic anaemia there is no satisfactory evidence that any more fundamental disturbance is produced by low folate levels.—We are, etc.,

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Balding Man

SIR,—As a follow-up to the article "The Hairy Story of Four Healthy Heads" (*Sunday Times*, 18 June), I would like to say that I was pleased to comply with the request to examine the persons concerned, because at least the two females involved were terrified by what they had been told at a hair clinic in London. Just as does the presence of too much hair in the conventionally wrong places in women, so too premature, temporary, or permanent hair loss from the accepted normal areas evokes in men, particularly in the younger age group, psychological reactions which are frequently out of all proportion to their clinical state. They will find it extremely difficult and often impossible to weigh for themselves the grave and even terrifying prognostications against the extensive and expensive course of physical treatment stated to be vital to prevent their rapid progress to the billiard-ball image. Could it not be, however, in some cases, a measure of the failure of our own profession to take time to communicate or to offer or obtain the informed medical diagnostic, advisory, and therapeutic services available within the National Health Service for such distressed patients, that they are driven to seek outside help?

As a practising consultant and chairman of the B.M.A. Dermatologists' Group Committee, which represents the opinions of colleagues throughout the country, who have nothing to gain but extra clinic work from this action, I feel that the widest possible publicity should be given to the following facts which are in no way controversial in medical circles but which seem generally unknown to the public at large.

(1) The progression of even premature or

normal male balding through frontotemporal recession to extending loss on the crown and to ultimate coalescence is, with rare exception, extremely slow and the presence or absence of dandruff is totally irrelevant to this. The maintenance of the status quo after three to six months of expensive treatment owes nothing to the activities of the "hair expert" concerned, and it is at this time that we are often first faced with a patient who owes a great deal of wasted money.

(2) Many disorders of hair growth in either sex are the result of or are associated with systemic diseases which require medical recognition, investigation, and treatment. Delay or neglect of such investigations, through non-medical ignorance, may be dangerous to life. Conditions such as thyroid disease or severe anaemia immediately spring to mind in this context.

(3) Research and medical experience have proved that, with extremely rare exceptions, the local application of creams, lotions, or shampoos, or the use of physical methods, manual or vibro-massage, ultraviolet or infrared light treatment, etc., have no influence whatever on hair growth. These exceptions are solely skin conditions affecting the scalp, which may respond to systemic or local treatment available only to the medical profession.

(4) Many disorders of hair growth—for example, telogen effluvium, mild alopecia areata, etc., do recover spontaneously within a few months and the "hair expert" will claim credit for cure when none is due. Failure of this early recovery in alopecia areata will call for medical treatment not permitted in the repertoire of the hair clinic.

(5) There is no treatment of proved value in any hair growth disorder which is not available under the National Health Service.

(6) In these days when V.D. is common once again in the United Kingdom it is vital to remember that syphilis, often undetectable in the female in the primary stage, may present itself, in the secondary stage, as a patchy alopecia, and failure to know and recognize and treat this may well result once again in the reproduction of children with the stigmata of congenital syphilis, and in serious cardiac or C.N.S. disease later in life for the infected person.

It seems to me that the profession can help here in informing their patients of these facts, where relevant, and in referring to us in our clinics those especially who seem to be over-reacting to their clinical state and who might so readily fall into the very expensive orbit of the charlatan. Local authorities are not empowered to refuse licences to private establishments provided their standards of hygiene seem acceptable and perhaps, as with the tattooing of minors, we may yet have to look to a Private Member's Bill in Parliament to deal with this insidious problem.—I am, etc.,

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Self-medication by Jockeys

SIR,—The description of the death of the jockey in the sauna bath (8 July, p. 71) prompts me to write to describe another feature of the pre-race preparation of the modern jockey which has an effect on health. Many jockeys now take diuretics—