

### Carcinoma of Tongue

SIR,—While your leading article on carcinoma of the tongue (6 May, p. 308) draws welcome attention to the epidemiological and diagnostic aspects of this condition, the section on treatment is open to criticism. It neither points to the recent and developing aspects of management—such as reparative surgery or the use of radiotherapy in the control of lymph node metastasis, nor gives a satisfactory “conventional” picture of management. Few surgeons with experience of block dissection bilaterally would recommend its prophylactic use save in exceptional circumstances.

Perhaps the most disappointing feature of the review is the continuing attitude which regards cancer of the tongue as a single entity which may be suitably treated by a single treatment programme. Surely the time has come when every cancer must be treated as an individual problem in an individual patient. While the small or moderate sized tumour is well treated by radiation, the rare exophytic or verrucous type of cancer not only tends to be radioresistant, but radiotherapy may actually be harmful.<sup>1</sup> In this type of tumour the results of surgery alone are excellent. In bulky, advanced tumours both radiotherapy and surgery alone are disappointing, and the only hope of improving results lies in combination therapy.

The programme of management recommended for cervical lymph nodes differs from that used by many workers. Again the selective attitude is the only logical approach. In experience here the incidence of lymph node metastasis from small (less than 2 cm) well differentiated lesions is very low and careful follow up is adequate treatment. Where cervical metastasis is considered likely, there is mounting evidence that radiotherapy in therapeutic doses gives a high incidence of local control. This is particularly important in the many cases where bilateral spread is possible since a good measure of control can be achieved with little or no morbidity.

No review of this subject can neglect the fact that the management of advanced cases and those recurrent after radiotherapy has been revolutionized by recent reparative techniques. The use of forehead, deltopectoral, or cervical flaps to replace soft tissue defects and bring a new blood supply into the area not only aids primary healing but gives vastly improved cosmetic and functional results.

Finally, it should be stressed that many specialties besides surgery and radiotherapy have much to offer in this condition. There is little doubt that oral cancer is best treated initially by a group of people especially interested in this field, each bringing their own specific expertise to bear on this problem.—I am, etc.,

L. E. HUGHES

University Department of Surgery,  
University Hospital of Wales,  
Cardiff

<sup>1</sup> Kraus, F. T., and Perez-Mesa, C., *Cancer*, 1966, 19, 26.

### Age and Anorectal Dilatation

SIR,—Mr. T. Bates describes “Rectal Prolapse after Anorectal Dilatation in the Elderly” (27 May, p. 505).

In our recently published series<sup>1</sup> we did

not mention age as a specific contraindication to dilatation, but our oldest patient was a man aged 66. We recommended that patients with weak perineal floors should not be subjected to forced anorectal dilatation. Such patients included those with previous pelvic floor surgery and women of high parity or over the age of 65. Mr. Bates's two elderly women, aged 85 and 78, would have been excluded by us on these grounds. In addition, both his patients had “fibrous anal stenosis” which we have described as another contraindication to forced dilatation, particularly when the fibrosis is caused by previous submucous injection of a sclerosant.

We would agree that great caution must be exercised in subjecting elderly patients to this extreme (eight finger) anorectal dilatation. Also, we would advise the exclusion of highly parous women or those with previous pelvic floor repairs.—We are, etc.,

T. W. BALFOUR  
I. M. C. MACINTYRE

Western General Hospital,  
Edinburgh

<sup>1</sup> Macintyre, I. M. C., and Balfour, T. W., *Lancet*, 1972, 1, 1094.

### Probenecid in Calcification of the Hands

SIR,—Professor C. E. Dent and Dr. T. C. B. Stamp (22 January, p. 216) successfully used probenecid in the treatment of troublesome superficial calcification of the hands occurring during the course of progressive systemic sclerosis. I should like to report experiences during treatment of a similar case where the patient showed initial slight improvement, marked deterioration after temporary interruption of therapy, and thereafter marked improvement when treatment recommenced. The time sequence of events in this case was striking.

A woman aged 64 has been under observation and treatment for systemic sclerosis for the past 15 years. The diagnosis is based on the typical facies with beaking of the nose and radial furrowing around the mouth, tightly bound-down skin on the hands and feet, and a creatinine clearance of 65 ml/minute. For the past six years no deterioration in her general condition has been observed, suggesting that the disease process is either quiescent or burnt out, and her main disability during this period has been pain and immobility of the joints of the hands and feet due to overlying calcium deposits. On occasions these have extruded spontaneously, and in addition some improvement has been obtained by the use of von Ebners decalcifying solution applied topically. When seen as an outpatient in January 1972 these deposits were numerous and painful and the decision was therefore made to treat the patient with systemic probenecid.

Plasma calcium level at this time was 9.2 mg/100 ml, plasma inorganic phosphate 4.0 mg/100 ml. Treatment was started with 0.5 g probenecid daily, and increased to 1 g daily after one week. No side effects were observed, and the patient was maintained on this dose until 1 May when she attended the outpatient department complaining of severe pain in her hands. On questioning it was revealed that she had discontinued probenecid therapy 48 hours previously. and on examination large bullae were observed, some over pre-existing areas of deposited

calcium, and some on previously normal skin. Culture of bulla fluid showed no growth, and microscopic examination of a smear showed amorphous material which was heavily stained by von Kossa's method for calcium. Plasma calcium was 8.3 mg, phosphorus 4.2 mg, serum uric acid 2.6 mg, and radiology of the hands showed no change by comparison with films taken in January 1972. Treatment with probenecid was started in a dose of 1 g daily, and for the following week fresh bullae requiring aspiration appeared daily on the hands. After this period new lesions ceased to appear, and as healing progressed it was observed that many of the previously solid calcium deposits had been extruded, and that mobility of the hands was greatly improved. Urinary calcium level at this time was 336 mg/24 hours.

From the time sequence it would appear that the temporary deterioration in this patient's condition can at least in part be attributed to sudden cessation of probenecid therapy, but the subsequent improvement in function is extremely striking.—I am, etc.,

RONA MACKIE

Department of Dermatology,  
University of Glasgow

### Anticonvulsant Hypocalcaemia

SIR,—Several reports<sup>1,2</sup> have confirmed the findings that calcium metabolism is disturbed by anticonvulsant therapy. It has been suggested that a drug-mediated enzyme induction may be the mechanism responsible by causing a greatly increased inactivation of vitamin D. Phenytoin is a much less potent enzyme inducer than phenobarbitone, yet the mean serum calcium was lower in patients receiving phenytoin than in those on phenobarbitone.<sup>1</sup> Thus, it can be assumed that other factors might also play a part.

It seems likely that phenytoin as well as other anticonvulsants may form an insoluble complex with calcium salts, as is known to happen with phenobarbitone, and the absorption of calcium and drugs from intestine in this form may be incomplete. Indeed, McQueen<sup>3</sup> reported that when a sucrose-lactose diluent was substituted for the calcium sulphate in phenytoin capsules patients developed adverse effects with markedly elevated serum phenytoin levels. Tyrer and others<sup>4</sup> made quite similar observations in Australia. They demonstrated also that patient's blood phenytoin level fell rapidly to a quarter of its former value when the phenytoin with a calcium sulphate excipient was used instead of phenytoin with a lactose excipient. The blood phenytoin concentration rose again when phenytoin-lactose excipient was reintroduced. There was no increase in faecal phenytoin loss at the time of the fall in blood phenytoin concentration. It must be emphasized, however, that this may be erroneous owing to a fault in the extraction method of faecal insoluble calcium-phenytoin complexes.

In our previous study<sup>5</sup> 71% of the 120 serum calcium determinations were subnormal as well as all the mean serum calcium levels of 40 mentally subnormal epileptics on phenytoin in three serial determinations:  $4.38 \pm 0.20$ ;  $4.47 \pm 0.21$  and  $4.27 \pm 0.27$  (S.D.) mEq/l. Nevertheless, only 9% of the 120 cerebrospinal fluid (C.S.F.) calcium determinations were subnormal and