Compression of Coeliac Axis

SIR,—The suggestion in your recent leading article (13 November, p. 378) is that the symptoms attributable to coeliac axis stenosis are the result of ischaemia. I would suggest that the mechanism of production of pain is not so mediated, but is the result of abnormal bowel motility.

I have recently had under my care a man aged 26 with coeliac axis obstruction, proved by aortography, who at operation had obstruction of the coeliac axis by voluntary muscular and fibrous tissue. This patient experienced upper abdominal pain immediately after food and, in the case of a large meal, the pain would even come on before he had finished it. When a barium sulphate suspension was given with a meal, the pain was associated with shunting of the barium to and fro in the proximal half of the duodenum. When atropine was administered immediately before the meal no pain developed, and this to and fro movement was abolished.

I suggest that the duodenum is partially obstructed by the superior mesenteric artery where it crosses the third part. In my patient, and in some others reported, the superior mesenteric artery has been abnormally large in calibre, possibly the consequence of the coeliac artery obstruction.—I am, etc.,

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Screening for Spastics

SIR,—Your leading article (13 November, p. 381) makes fair comment on many of the moot points raised by the Spastic Society's report¹ on the implications of universal periodic developmental screening, but is sceptical about the suggested feasibility of universal neonatal assessment. You say that screening tests have not been proved reliable, that they would make excessive demands on the valuable time of expert paediatricians, and that in any case there is as yet no proof that early treatment is advantageous.

I would draw your attention to an assessment test for the newborn developed over the past decade.² A sample of children found to have minor neurological dysfunction according to this test were later examined together with matched controls and a significant level of specific learning difficulty demonstrated.³ This correlation suggests that the test may help in the early ascertainment of forms of handicap susceptible of amelioration. There are 17 items incorporated in the assessment, so it is rather more than the short screening test proposed by Prechtl and Beintema.⁴ A doctor trained in this method can examine the normal neonate in five (not 15) minutes. Willing juniors and some local authority doctors have been trained in an intensive part-time, one-week course, after which they could diagnose the great majority of normal babies with confidence.

This assessment is currently used in our area for 4,000 babies per annum born in hospital. The basic service for routine examinations is given by one full-time senior house officer with about 1/5 middle-grade registrar and 1/10 consultant paediatrician time. We believe it has not unbalanced or

diluted the routine service but rather enriched it. We are guardedly optimistic so far about the outcome for children with cerebral palsy brought early to therapy, but, as you say, it is best to await proof one way or the other before general implementation can be considered.—I am, etc.,

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- 1 *The Children Who Cannot Wait*, ed. J. Loring. London, The Spastics Society, 1971.
- 2 O'Doherty, N., and Zinkin, P., *Proceedings of the Royal Society of Medicine*, 1971, **64**, 476.
- 3 Francis-Williams, J., *Children with Specific Learning Difficulties*. London, Pergamon Press, 1970.
- 4 Prechtl, H. F. R., and Beintema, D. J., *The Neurological Examination of the Full-term Newborn Infant. Clinics in Developmental Medicine*, No. 12. London, Spastics Society with Heinemann, 1964.

Taste and Smell

SIR,—Clinical states of persisting perversion of taste and smell, other than those attributable to a psychiatric disorder, merit a degree of appreciation greater than would appear to prevail, as judged by the scanty literature on the subject. Your leading article (27 November, p. 508) refers to a new syndrome designated as "idiopathic hypoageusia, hyposmia, and dysosmia." In a communication under the title of "dysageusia in the elderly" I reported a similar condition, the disorder being confined to the sense of taste. The patients experience a continuous taste of an unpleasant nature which transmits itself to all food and drink. Often its changed nature is foreign to ordinary experience so that a satisfactory description could prove difficult. Loss of weight reflected the consequent impairment of appetite, and the attending emotional disturbance was due to the gustatory abnormality and was not in itself the precipitating factor.

Subsequent experience has revealed a wider age incidence. When spontaneous disappearance of the symptoms did not ensue, phenytoin proved a valuable form of treatment, but it is necessary to persevere since the beneficial effects may be delayed. Treatment with oral copper, zinc, or nickel as suggested by Henkin and his colleagues² for the combined anomalies of taste and smell would seem worthy of trial in this more limited form.—I am, etc.,

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- 1 Shafar, J., *Lancet*, 1965, **1**, 83.
- 2 Henkin, R. I., Graziadei, P. P., and Bradley, B. F., *Annals of Internal Medicine*, 1969, **71**, 791.

Myxoedema during Cyclophosphamide Therapy

SIR,—Autoimmunity has been implicated in the aetiology of both thyrotoxicosis and myxoedema, and the report (30 October, p. 275) by Dr. I. R. McDougall and others of a case of thyrotoxicosis developing during cyclophosphamide therapy suggests that the relationship between thyroid dysfunction and immunity is even more complex than has been thought. To complement their case I here describe a case of myxoedema developing during cyclophosphamide therapy.

In March 1967 a 44-year-old woman had a simple mastectomy and postoperative radiotherapy for carcinoma of the breast. In February 1969 local recurrences were excised from the axilla and chest wall. A bilateral oophorectomy was performed, but a further skin nodule had appeared by October 1969. Cyclophosphamide 100 mg daily was started, and the dose was increased to 150 mg daily in June 1970. There was a satisfactory response.

Durabolin (nandrolone phenylpropionate) 50 mg weekly was started in July 1970 when the cyclophosphamide was stopped owing to a severe leucopenia. A month later cyclophosphamide 100 mg daily was again given. A slight increase in weight, a rough voice, and some falling out of hair were attributed to the combined effects of Durabolin and cyclophosphamide and the Durabolin was stopped in December. In May 1971 the patient weighed 68.9 kg and was taking only 50 mg of cyclophosphamide daily. This was increased to 100 mg daily in September. By mid-November she weighed 74.4, was lethargic, disliked the cold, looked pale, she had lost hair from the eyebrows, and her skin was dry. The ankle jerks were slow to relax. Serum cholesterol was 495 mg/100 ml, and four-hour iodine uptake of the thyroid was only 4%. Cyclophosphamide was discontinued but the patient was so ill that thyroxine could not be withheld. Already she feels better.

In our case and Dr. McDougall's the association of thyroid dysfunction with immunosuppressive therapy could be coincidental. However, it is not too surprising that both hypofunction and hyperfunction might be produced since the progression of hyperthyroidism to hypothyroidism is well known and the sequence of hypothyroidism followed by hyperthyroidism has recently been described.¹—I am, etc.,

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- 1 James, K. W., *Lancet*, 1971, **2**, 156.

Coronary Deaths—How Unexpected?

SIR,—Drs. P. G. F. Nixon and H. J. N. Bethell (20 November, p. 486) draw attention again to the long-standing coronary arterial disease process leading up to a pre-infarction state with clearly recognizable features. However, the decisive steps to protect the individual at this stage are not mentioned. If they have in mind what is generally known today to be beneficial—namely, reducing activities and responsibilities, dieting, cholesterol-lowering regimens, reducing hypertension, giving up smoking, and possibly anticoagulant or fibrinolytic therapy, or operative treatment to improve the coronary circulation—these methods are probably applied too late and may be of doubtful immediate benefit, as the number of patients who have coronary thrombosis does not appear to have been remarkably reduced.

There are a number of observations, however, which seem to have some bearing on a solution of this problem. We know of the "coronary personality" with the aggressive type of behaviour pattern. We are aware of certain physical events usually of a strenuous nature, or of a severe emotional disturbance, preceding the coronary thrombosis by a few hours or days. We are informed that people subjected to prolonged grief, such as widowers, have a greater incidence of cardiac infarctions. The link between all these examples, through the mediation of catecho-