sites must be affected by some secondary mechanism (for example, allergy) it is likely that the intestinal lesions are of like origin rather than simple irritation.

It is not easy to date onset. Cases observed in depth show symptoms in childhood which in retrospect can be ascribed to Crohn's disease. Thus it could well be that patients with "preclinical" Crohn's disease find cornflakes more acceptable than other cereals. Likewise, the cornflake hypothesis does not account for cases in my knowledge with florid features before the introduction (about 1920) of cornflakes. I would rather put my money on refined sugar—though in my observation alcohol can provoke acute exacerbations.

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SIR,—I must respectfully criticise Dr A H James's article (9 April, p 943). His retrospective survey was unfortunately too small to be significant (a point to keep in mind when planning a proper prospective survey). Also there are many factors which should be taken into consideration.

Firstly, how many of the cornflakes users brushed their teeth before breakfast, after, or before going to bed? Secondly, how many took their breakfast standing, sitting, or leisurely in bed? Thirdly, as 19 patients had had surgery (that is, more than 50%) how long had they eaten cornflakes before operation, and how many of us remember how regularly we had cornflakes five years ago? Fourthly, are we not manipulating excessively the results when we add with regular eaters those taking cornflakes once a week? And lastly, how many in Dr James's survey resorted to cornflakes to make up for their creeping lack of energy due to Crohn's disease as yet undiagnosed but nevertheless existing?

Perhaps the answer would be in a prospective study. It would be wrong to give much significance to a very small retrospective study.

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Practice in Canada

SIR,—I have read with interest and appreciation the article "Where Shall John Go?" prepared by Dr M J Chamberlain with respect to Canada (12 March, p 693). I think he has done an excellent job and I found it most interesting. I would like, however, to stress a couple of points, as I occupy a position where I tend to see the things that go wrong.

I would first re-emphasise Dr Chamberlain's advice to check with the pertinent provincial licensing body and to allow time. No visa can be issued until one has both an offer of employment and proof of registrability. The offer of employment is always dependent on registrability, and therefore the first action after making a contact should be to write to the licensing body in the province of the intended appointment. There are 10 separate provincial licensing authorities and each has slightly different rules. Dr Chamberlain indicates that a full licence is normally granted only to physicians who have the LMCC. This is true in the four most populous provinces but is not so in the others, and even where it is required there may be exceptions.

Secondly, one aspect of practice in Canada to which British graduates seem to need adjustment is the increasing degree of medical audit and peer review. I realise that this is developing in Britain but I do not believe to the same extent. We sometimes see resentment from new immigrants when they are first exposed to audit and tend to regard it as either an unwarranted intrusion on their privacy or as a disciplinary procedure.

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Impeccable syllable

SIR,—Miss Madeline A Stringer of Dublin (2 April, p 908) is wrong to stigmatise "dilatation" as a pretentious Americanism. Not only is the "superfluous" syllable etymologically impeccable but the word has time-honoured medical usage (in Lanfranc's *Science of Chirurgie*, circa 1400: "And if that the blood go out of arterie thou schalt knowe it by construccion and dilatacion of the same arterie"). It is the word "dilation" that, except in an obsolete form for deferment or delay, is regarded as improper by the *Oxford English Dictionary*. Slightly to adapt Miss Stringer's distinguished fellow-Dubliner, these delightful records should have been her constant study.¹

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¹ Wilde, O F O'F W, *The Importance of Being Earnest*, Act III.

Bromocriptine for severe mastalgia

SIR,—Your leading article on the subject of bromocriptine (2 April, p 863) gives an excellent review of the use of this drug by the endocrinologist. However, it does not cover its other roles, such as the control of the symptoms of severe mastalgia and premenstrual tension.

We have recently used bromocriptine in treating 16 women suffering from severe premenstrual mastalgia which had not been adequately controlled with the orthodox treatment of diuretics and analgesics. Each patient had her plasma prolactin concentration measured on several occasions before starting to take bromocriptine. It is interesting that only a minority of this group had prolactin levels above the upper limit of normal. The bromocriptine was started at a low dose, quickly increasing to 2.5 mg twice daily. The patients were then seen at monthly intervals to assess the results and measure the plasma prolactin levels. The breasts became smaller and softer and the premenstrual tenderness disappeared or was much reduced. A few patients required 2.5 mg three times a day to control their symptoms adequately. The transient nausea and general malaise that most of the patients complained of in the first few days of treatment were acceptable when severe mastalgia was relieved. We used antiemetics routinely in the early days of treatment, but metoclopramide should not be used on the theoretical basis that it can increase the prolactin level.¹

In our small series 13 out of 16 patients

were well satisfied with the results. These results are similar to those in other series.²⁻⁴ If the plasma prolactin level is reduced to below 5 $\mu\text{g/l}$ there is usually good symptomatic relief. We have not seen any relationship between the efficacy of bromocriptine treatment and the pretreatment prolactin level.

We have found this to be a very effective treatment in what can be an extremely distressing condition. Our problem now will be in persuading some of the patients to stop the only treatment that has given them real relief.

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¹ Besser, G M, and Thorner, M O, in *Advanced Medicine Symposium 11*, ed A F Lant, p 255. London, Pitman Medical, 1975.

² Benedek-Jaszman, L J, and Hearn-Sturtevant, M D, *Lancet*, 1976, **1**, 1095.

³ Martin-Comin, J, et al, *Obstetrics and Gynecology*, 1976, **48**, 703.

⁴ Schulz, K-D, et al, *Archiv für Gynäkologie*, 1975, **220**, 83.

Bromocriptine for Cushing's disease

SIR,—In your leading article "Bromocriptine—fact or expensive fancy?" (2 April, p 863) you state that, at present, bromocriptine's endocrinological uses "are limited to suppressing puerperal lactation and pathological hyperprolactinaemia and to treating acromegaly." We feel that, as others have shown^{1 2} and as the following case illustrates, bromocriptine may have a role in the management of Cushing's disease.

A 35-year-old man presented with lack of energy, muscle weakness, impotence, depression, and polydipsia. He had classical features of Cushing's syndrome, with profound proximal muscle weakness. Investigation revealed a hypokalaemic alkalosis, a diabetic glucose tolerance curve, and widespread changes in the electrocardiogram (ECG)—with S-T depression and T-wave inversion. Morning and late night plasma cortisol concentrations were 1446 nmol/l (57 $\mu\text{g}/100\text{ ml}$) and 1690 nmol/l (61 $\mu\text{g}/100\text{ ml}$) respectively with corresponding ACTH (corticotrophin) levels of 187 and 116 ng/l. Dexamethasone (8 mg/24 h) for four days did not cause adrenal suppression. Pituitary tomography showed a sella at the upper limit of normal. Adrenal venography revealed bilateral adrenal enlargement, and plasma cortisol values of 3153 nmol/l (114 $\mu\text{g}/100\text{ ml}$) and 3833 nmol/l (139 $\mu\text{g}/100\text{ ml}$) were found in the right and left adrenal veins respectively.

In view of the severity of his condition urgent measures to reduce his plasma cortisol levels were felt to be necessary. Treatment with aminoglutethimide 2 g and metyrapone 3 g daily was begun. After a week morning and late night plasma cortisol concentrations were 215 and 174 nmol/l (7.8 and 6.3 $\mu\text{g}/100\text{ ml}$) respectively and the patient felt stronger. Bromocriptine 1.25 mg twice daily and dexamethasone 1 mg daily were added and the aminoglutethimide and metyrapone withdrawn after a further week. The accompanying table shows his plasma cortisol and ACTH levels (taken between

Plasma cortisol and ACTH levels during bromocriptine and dexamethasone treatment

	Time (weeks) from start of initial medical treatment					
	4	6	16	20	24	28
Cortisol (nmol/l) . .	653	274	562	342	417	482
ACTH (ng/l) . .	66	25	50	98	59.6	50

Conversion: SI to traditional units—Plasma cortisol: 1 nmol/l $\approx$ 0.036 $\mu\text{g}/100\text{ ml}$.