in the news columns of the *Toronto Journal* (1 May 1982, p 1).

We now have a copy of the still unpublished manuscript. We are surprised to discover that Dr Fornazzari's study, while it confirms an association between chronic heavy toluene abuse and persistent cerebral impairment, does not support as clearly as Dr King and others suggest the contention that cerebral damage is a direct pharmacological effect of toluene (Fornazzari L, personal communication). Not all of Fornazzari *et al*'s sample of chronic heavy toluene abusers showed evidence of cerebral impairment, nor was there a clear relation between dosage and damage. These facts led them to suggest that mediating factors such as anoxia, malnutrition, or genetic differences might have caused the cerebral impairment they observed.

Since your readers are unlikely to have access to Dr Fornazzari *et al*'s unpublished manuscript we feel it is important to draw attention to the misleading interpretation that Dr King and others seem to have placed on their work.

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Maternal nutrition, breast-feeding, and contraception

SIR,—In an otherwise admirable and concise summary of the theoretical effect of breast-feeding on conception spacing, Professor and Mrs E F P Jelliffe (18 September, p 806) have unfortunately oversimplified the issue.

Even if good sustained lactation of the type practised by ecologically minded people did manage to produce a four-year spacing, all such women would end up with eight children. No such formidable population explosions occurred in any known society. Breast-feeding was therefore not the only ancestral means of birth spacing although it might have been the only endocrinological one.

In many Third-World countries today in which breast-feeding is the rule child spacing is just over two years and many women become pregnant even while lactating, frequently and effectively. Indeed, pregnancy is often given as the sole reason for cessation of lactation, and it is not unusual to see two siblings being breast-fed at the same time. While breast-feeding should be encouraged in all societies, and encouraged for as long as possible, to await Professor and Mrs Jelliffe's suggestion of "usual length of lactation ... minus two months" before the introduction of modern contraceptives would not work because many women would already be pregnant.

Hunter-gatherers had other forms of population control which have never since been equalled in their effectiveness. Infanticide was one method, and while we may abhor such a practice to their way of reckoning it was no more abhorrent than the thousands of abortions undertaken by us in an attempt to keep our numbers down. But there were other much more subtle ways in which people managed to stabilise their numbers,[1] and some of these might prove effective today in societies in which modern technological contraception has failed and will continue to do so.

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Thyrotoxic atrial fibrillation

SIR,—I found the leader on thyrotoxic atrial fibrillation (2 October, p 909) most interesting, in particular the increased likelihood of atrial fibrillation in those with hyperthyroidism aged 60 and over.

Atrial fibrillation is not rare in the elderly, and thyroid disease has been found in up to 2% of elderly patients admitted to hospital. Results of thyroid function tests are often raised on admission, but they return to normal over a period of two to three weeks in many patients.

Not only must one be careful to exclude hyperthyroidism in patients with atrial fibrillation but one must also be careful to confirm the diagnosis so that a number of normal elderly patients are not submitted to antithyroid treatment.

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SIR,—The leading article by Dr J C Forfar and Dr A D Toft (2 October, p 909) is timely and should lead to a greater awareness of the possibility of hyperthyroidism in patients with unexplained atrial fibrillation. As emphasised by these authors, symptoms and signs of thyroid overactivity may be subtle and the serum total triiodothyronine and thyroxine concentrations may deceptively be within the normal range in hyperthyroid patients with a coexisting non-thyroidal illness or who are receiving drugs that lower circulating concentrations of thyroid hormones. Under these circumstances the thyrotrophin-releasing hormone test is a very useful investigation, but unfortunately the interpretation of this test is not as straightforward as depicted in the leading article.

Dr Forfar and Dr Toft state that in the absence of hypopituitarism a lack of response of serum thyroid-stimulating hormone after intravenous administration of thyrotrophin-releasing hormone is an indication of hyperthyroidism irrespective of the concentrations of serum total thyroid hormones. We are rather concerned this may be misleading since the thyrotrophin-releasing hormone test is not as specific as the authors have implied. A lack of response of thyroid-stimulating hormone to exogenous thyrotrophin-releasing hormone is merely consistent with, and not necessarily indicative of, hyperthyroidism. Thus, such a response has been noted frequently in healthy relatives of patients with Graves's disease and in euthyroid patients suffering from multinodular goitre, non-toxic diffuse goitre, ophthalmic Graves's disease, acromegaly, Cushing's syndrome, chronic renal failure, or unipolar depression.[1 2] Moreover, while Dr Forfar and Dr Toft referred to the lowering effects of certain drugs on circulating concentrations of thyroid hormones, they failed to point out that secretion of thyroid-stimulating hormone in response to thyrotrophin-releasing hormone may be inhibited to a greater or lesser extent by agents such as levodopa, bromocriptine, cyproheptadine, and pharmacological doses of corticosteroids.[3-6] Accordingly, in a proportion of euthyroid patients being treated with such drugs the response of thyroid-stimulating hormone to thyrotrophin-releasing hormone may be non-existent or impaired. Finally, contrary to what is stated in the leading article and in standard endocrinology textbooks, a normal response of thyroid-stimulating hormone in the thyrotrophin-releasing hormone test does not entirely exclude a diagnosis of hyperthyroidism. In the rare but increasingly recognised condition of non-neoplastic pituitary insensitivity to thyroid hormones, clinical and biochemical hyperthyroidism may be associated with a normal thyrotrophin-releasing hormone test.[7-9]

The thyrotrophin-releasing hormone test is undoubtedly useful, but to diagnose hyperthyroidism solely on the basis of a lack of response of thyroid-stimulating hormone to thyrotrophin-releasing hormone would seem unwise and is not without risk since this will lead to inappropriate treatment in some cases.

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SIR,—It was refreshing to read the leading article by Dr J C Forfar and Dr A D Toft (2 October, p 909) on thyrotoxic atrial fibrillation which squarely places the responsibility on the physician and cardiologist to request the correct thyroid function tests on all patients with "lone" atrial fibrillation. Sadly, this is the lesser part of the problem because our biochemist colleagues, ever conscious of costs, always need persuasion to perform estimations of serum thyroid-stimulating hormone after administration of thyrotrophin-releasing hormone in the presence of a normal total serum thyroxine. It is not sufficiently recognised in the endocrine laboratory that the total thyroxine often cannot pick out the hyperthyroid patient who has cardiac disease—by virtue of the latter the heart is more sensitive to changes in hormone level. It is surely time for serum free-thyroxine estimation to replace the more widely used and inadequate total thyroxine estimation.

Perhaps because of the logistics involved Dr Forfar and Dr Toft stop short of advising the screening of all patients with paroxysmal or persistent atrial arrhythmias (even including sinoatrial disease) for thyroid disease, with