12 Feinleib M, Garrison RJ, Fabsitz R, *et al.* The NHLBI twin study of cardiovascular disease risk factors: methodology and summary of results. *Am J Epidemiol* 1977;**106**:284–95.
13 Abraham S, Collins G, Nordsieck M. Relationship of childhood weight status to morbidity in adults. *Health Services and Mental Health Administration Health Reports* 1971;**86**:273–84.
14 Wilcox AJ, Russell IT. Birthweight and perinatal mortality:III. Towards a new method of analysis. *Int J Epidemiol* 1986;**15**:188–96.
15 Khoury MJ, Cohen BH. Genetic heterogeneity of prematurity and intrauterine growth retardation: clues from the old order Amish. *Am J Obstet Gynecol* 1987;**157**:400–10.
16 Garn SM, Shaw HA, McCabe KD. Birth size and growth appraisal. *J Pediatr* 1977;**90**:1049–51.
17 Bradley PJ. Is obesity an advantageous adaptation? *Int J Obes* 1982;**6**:43–52.
18 Bradley PJ. Weight in infancy and death from ischaemic heart disease. *Lancet* 1989;**ii**:985.
19 Bradley PJ. Fetal and placental size and risk of hypertension in adult life. *Br Med J* 1990;**301**:551.
20 Bradley PJ. Obesity and risk of coronary heart disease in women. *New Eng J Med* 1990;**323**:1144.
21 Bradley PJ. Fetal and infant origins of adult disease. *Br Med J* 1991;**302**:113.
22 Bradley PJ. Deprivation in infancy or in adult life. *Lancet* 1991;**337**;1043–4.
23 Court JM, Dunlop M. Obese from infancy: a clinical entity. In: Howard A, editor. *Recent advances in obesity research. Vol. I.* London: Newman Publishing, 1975:34–6.
24 Garn SM, McCabe KD. Maternal fatness and placental size. *Am J Clin Nutr* 1979;**32**:277–9.
25 Garn SM, Cole PE, Bailey SM. Effect of parental fatness levels on the fatness of biological and adoptive children. *Ecology of Food and Nutrition* 1977;**7**:91–3.
26 Williams PT. Weight set-point theory predicts HDL-cholesterol levels in previously obese long-distance runners. *Int J Obes* 1990;**14**:421–7.

Czechoslovakia revisited

Sir—We were delighted by Dr Suri's article published in the April issue of the *Journal.* Dr Suri gave an excellent lecture on The organisation and funding of the geriatric services in the UK at the course on geriatric medicine during his stay in our country. It created such interest that he has been asked to publish it in a Czechoslovak medical journal.

Our Gerontological Society also has very good contacts with the University Department of Geriatric Medicine at the University of Wales in Cardiff. These contacts started five years ago with a visit to Cardiff by Dr Reban who is vice-president of the Czech Gerontological Society.

Eight Czechoslovak geriatricians took part in the first British–Czechoslovak Geriatric Symposium in Cardiff in 1987. The second symposium was held the next year in Prague and the third one in Cardiff in 1990. Five British colleagues participated in the last symposium which was held on 16 May 1991 in Bratislava. The papers will be printed in the fifth volume of *Current Problems of Geriatrics.*

Czechoslovak geriatricians have appreciated the high quality of care for the elderly in the UK for a long time. The British system of care for the elderly is the model for the currently prepared reform of geriatric services in Czechoslovakia.

The Czecho-Slovak Gerontological Society was founded in 1969 and has currently about 1500 members. Its main purpose is to spread knowledge of advances in the field of gerontology and geriatrics. This aim is achieved by holding seminars, conferences and symposia in various towns of our country. The geriatric section is a very active, integral part of the Society.

The structure of Czecho-Slovak Gerontological Society reflects the federal structure of our state. It is composed of the Czech Gerontological Society and the Slovak Gerontological Society. Officers from each society alternate for a period of two years in the administrative charge of the combined society.

S. KRAJCIK,
Secretary of Czecho-Slovak Gerontological Society
S. LITOMERICKY,
President of Czecho-Slovak Gerontological Society

Lumbar Puncture at The National Hospital, Queen Square

Sir—Dr Alex Sakula's article (April 1991) on a hundred years of lumbar puncture reminded me of my past interest in cerebrospinal fluid.

At the 1964 meeting of the Association of British Neurologists in Winchester, Dr William Gooddy, that year's President of the Association, introduced me to Sir Gordon Holmes. I wanted to know who introduced lumbar puncture to Queen Square and when. Sir Gordon, then aged 88, tall and upright, was clearly mentally alert and spoke with a deep, commanding voice. The reply to my question was:

"Let me see, was I on the house in One or in Two?' He peered into the distance and after a seemingly long pause turned to me, slightly bent forward, and then came the explosive: 'Never mind, it doesn't matter. Gowers was against it! So we did not do it.'

Such was the power of the senior physician of former days that no-one was permitted to do a lumbar puncture until Sir William Gowers retired in 1910. Hence, no British book on cerebral spinal fluid appeared until 1925 [1].

Now that I am senior physician, I occasionally regret not to have the power and influence exerted by my predecessors. But not frequently—democracy is preferable.

Some years earlier I had written to Dr Parkes Weber asking the same question. The reply, on a post card signed but not written by him (he was blind), stated that he thought he was the first to perform a lumbar puncture in London at The German Hospital, Hackney in 1896. His invitation to a surgeon on the staff of the hospital to perform the procedure was declined so he did it himself. But clearly Dr Essex Wynter at the Middlesex Hospital preceded Dr Parkes Weber's lum-

bar puncture by five years. Nevertheless Wynter must have been quick off the mark, Quincke's report at the international congress being made only a month earlier.

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Reference

1 Greenfield J G, Carmichael E A. *The cerebral spinal fluid in clinical diagnosis*, McMilland, London. 1925.

Atopic Eczema

Sir—I was interested to read the recent overview article concerning new developments in the treatment of atopic eczema [1]. The author discusses the use of evening primrose oil (EPO) in just one paragraph in the article. This in itself is surprising, since EPO is the only new development in the management of atopic eczema to have received a licence for use in the condition. But there are several serious errors of fact which should be corrected.

First, the study conducted by myself and Dr Burton did indeed show a significant benefit in children with atopic eczema from the use of EPO. This is very clearly stated in our paper [2]. It is true that the results in children were not as good as in adults, and we discussed the possible reasons for this. Your overview fails to mention that the other published controlled study of EPO in children, using a larger dose, was very significantly positive in favour of EPO [3]. One further completed study confirms the clinical benefit of EPO over placebo [4], and further controlled studies of EPO in young children are in progress.

The meta-analysis of the controlled studies referred to by Dr David does not include *selected* studies. It includes *all* studies, as a meta-analysis must. He is quite mistaken to state that the meta-analysis did not include the single negative study, and the fact that the Bamford *et al* study was included in the meta-analysis is very clear in the published article [5].

While Dr David is correct to state that the negative study of Bamford *et al* [6] was the single largest trial of EPO, there were several serious drawbacks to this study. First, compliance was extremely poor—65% of the patients achieved 50% compliance. Second, analyses of blood fatty acid patterns strongly suggested that there had been mixing of placebo and active capsules. Third, patients were assessed at three months intervals only, making reliable assessment of the disease severity extremely difficult, and finally, the age group of patients used was particularly old. There is ample justification therefore for doubting the validity of this study. Because of the questionable validity of the Bamford study, the meta-analysis was carried out both with and without these results, and in both cases shows a significant benefit of EPO over placebo. It may be that this has confused Dr David.

In the context of a discussion of whether EPO has a place in the management of atopic eczema, it seems a shame that Dr David has failed to point out the following:

i. No other single treatment approved for use in atopic eczema has been subjected to controlled clinical trials in so many patients.
ii. The use of EPO is based on the recognition of an abnormality of essential fatty acid composition in plasma and mononuclear cells in both adults and children [3,7–9], and in the umbilical cord blood of infants at risk of eczema [10,11]; in adipose tissue, [12] and in breast milk [13] of affected mothers. The skin of patients with atopic eczema similarly has abnormalities of fatty acid composition [14,15]. It is therefore the only treatment licensed for use in atopic eczema with a sound rationale for its use.
iii. EPO is extremely safe.

I hope that correction of these factual inaccuracies may prove helpful to your readers.

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References

1 David TJ. Recent developments in the treatment of childhood atopic eczema. *J R Coll Physicians Lond* 1991;**25:**95–101.
2 Wright S and Burton JL. Evening primrose seed oil improves atopic eczema. *Lancet* 1982; **ii:**1120–1122.
3 Bordoni A, Biagi PL, Masi M *et al.* Evening Primrose oil in the treatment of children with atopic eczema. *Drugs Exp Clin Res* 1988;**14:**291–97.
4 Heddle RJ, Forsyth K, Gordon LA *et al.* Oral evening primrose oil (EPO) in atopic eczema. *Med J Aust* 1991; (submitted for publication).
5 Morse PF, Horrobin DF, Manku MS *et al.* Meta analysis of placebo controlled studies of the efficacy of gammalinolenic acid in the treatment of atopic eczema. Relationships between plasma essential fatty acid changes and clinical response. *Br J Dermatol* 1989;**121:**75–90.
6 Bamford JTM, Gibson RW, Renier CM. Atopic eczema unresponsive to evening primrose oil. *J Am Acad Dermatol* 1985;**13:**959–63.
7 Manku MS, Horrobin DF, Morse NL *et al.* Reduced levels of prostaglandin precursors in the blood of atopic patients: defective delta-6-desaturase function as a biochemical basis for atopy. *Prostaglandins, leukot Med* 1982;**2:**615–628.
8 Manku MS, Horrobin DF, Morse NL *et al.* Essential fatty acids in the plasma phospholipids of patients with atopic eczema. *B J Dermatol* 1984;**110:**643–48.
9 Muhlemann M, Manku MS, Leonard TJ *et al.* Essential fatty acids in the plasma phospholipids of patients with atopic cataracts. *Br J Dermatol* 1987;**116:**179–82.
10 Strannegard I-L, Svennerholm L, Strannegard O. Essential fatty acids in serum lecithin of children with atopic dermatitis and in umbilical cord serum of infants with high or low IgE levels. *Int Arch Allergy Appl Immunol* 1987;**82:**422–23.
11 Picardo GE, De Luca C, Businco L *et al.* Essential fatty acids in cord blood lymphocytes of infants "at risk" of atopy. *Pediatr Research* 1989;**26:**519.
12 Wright S and Sanders TAB. Adipose tissue essential fatty acid composition in patients with atopic eczema. *Eur J Clin Nutr* 1991; (in press).