SIR,—After reading the paper by Drs A Deshpande and Sheila A McKenzie (19 July, p 169) on the use of steroids in acute asthma in children we analysed the data from 34 out of 50 proposed patients from our own similar study and reached the same conclusions.

Children aged 5 years and over were entered into a double blind randomised placebo controlled trial of a seven day course of prednisolone. All had acute asthma judged to require hospital admission for treatment with a nebulised bronchodilator but not with parenteral steroids or bronchodilators. Those weighing less than 20 kg were given prednisolone or placebo 20 mg daily and those weighing 20 kg or more 40 mg daily. Peak flow rate and clinical score based on heart rate, respiratory rate, use of accessory muscles of respiration, and wheeze were measured on admission and at 24 hours, 48 hours, 7 days, and 21 days. Seventeen received prednisolone and 17 placebo. One child in the placebo group was excluded from the analysis because of deterioration needing treatment with intravenous steroids and bronchodilators. Nine children, four from the prednisolone treated group, could not use the peak flow meter. On admission there was no difference between the two groups in peak flow rates expressed as a percentage of the value predicted from height, or clinical score (table).

Mean (SD) clinical score and peak expiratory flow rate (as % of expected for height) for prednisolone and placebo treated asthmatic children

Time	Clinical score		Peak flow (l/min)	
	Prednisolone	Placebo	Prednisolone	Placebo
0	6.6 (2.1)	7.0 (1.8)	43.6 (18.4)	56.2 (24.0)
24 h	3.2 (2.3)	3.6 (1.3)	73.0 (27.1)	78.8 (21.6)
48 h	2.9 (1.4)	3.3 (1.1)	101.9 (21.1)	77.0 (17.7)
7 days	2.0 (1.0)	2.0 (0.9)	106.8 (27.0)	98.6 (15.2)
21 days	1.7 (1.3)	1.4 (1.1)	109.8 (18.6)	111.6 (19.8)

At 24 hours there were no significant differences between the groups. By 48 hours there was a significantly greater improvement in peak flow in the prednisolone treated group than in the placebo group ($t=3.14$, $p<0.01$). At the same time nine out of 17 in the prednisolone treated group were able to be discharged home compared with only one out of 16 in the placebo group ($\chi^2=6.44$, $p<0.02$). No significant differences were found at seven and 21 days.

Although our patients were treated in hospital, their peak flows at presentation were similar to those of the authors' patients and they seemed to be experiencing acute asthma of similar severity. We therefore agree with their conclusions that oral corticosteroids, in addition to inhaled bronchodilators, speed recovery in mild and moderately severe acute asthma in childhood. We could not show any additional benefit from a seven day course compared with a three day course.

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AIDS: act now, don't pay later

SIR,—Your leading article (9 August, p 348) emphasises the need for informing and educating the public in the campaign against the acquired immune deficiency syndrome. It also emphasises the fact that health education has to be explicit, but interestingly enough it couples this with the

qualification that AIDS may be caught by any sexual contact. After reading your leading article one would be entitled to deduce that AIDS is not first and foremost related to male homosexuality. This is in keeping with the largescale advertisements from the DHSS that have appeared recently, which succeed in avoiding any mention of homosexual activity.

The revised guidelines issued by the Advisory Committee on Dangerous Pathogens in June of this year show that 296 out of 324 cases of AIDS in the United Kingdom occurred in homosexuals. You state that effort should be concentrated in particular on drug abusers. These comprised three cases out of the total, and presumably for these and all needle transmitted infections the original source would still have been a male homosexual.

Primary prevention must direct itself to the source and explicitly state that "Homosexual activity may damage your health."

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Ingestion of button batteries

SIR,—We agree with Drs B Kiely and D Gill (2 August, p 308) that button batteries in the oesophagus should be removed urgently and that the large majority of batteries that reach the stomach will pass spontaneously. On the basis of a review of 119 cases of ingestion of button batteries Litovitz reported that 85% of batteries pass within 72 hours of ingestion.¹ Complications such as mercury poisoning and visceral perforation also seem to be extremely rare.

However, should a battery not pass it may still be unnecessary to submit a child to laparotomy to remove it. We recently treated an 11 month old girl who had swallowed a 16 mm diameter, mercuric oxide button battery. Within 24 hours she was passing grey-black motions. Aperients and metaclopramide were administered but by three days after ingestion radiographs showed that the battery was still in the stomach and that the encircling metal flange was separating. In view of this delay in transit the battery was removed using a 10×30 mm bar magnet (E 807, Eclipse magnet, Neill Tools Ltd, Sheffield). This was attached to a polyethylene nasogastric tube by stainless steel wire, which was threaded through the tube lumen. The magnet was passed through the mouth with the infant head down on an inclined x ray table. X ray screening showed that the magnet rapidly attracted the battery, and the two were removed easily together. Although an anaesthetist was present throughout the procedure, the patient was not anaesthetised. Subsequently there were no clinical sequelae.

We attributed the delay in transit of the battery to its size (most batteries ingested are less than 12 mm in diameter¹) and the young age of the patient. Removal was undertaken because of this and the complications mentioned. In the absence of mercury poisoning we might have persisted longer with aperients. However, if it becomes necessary to remove a button battery this method is preferable to laparotomy.

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1 Litovitz TL. Battery ingestions: product accessibility and clinical course. *Pediatrics* 1985;75:469-76.

Whatever happened to the Black report?

SIR,—I shall reply to only two of the points raised by Drs Peter Goldblatt and Alison McFarlane (22 August, p 503) since Sunderland and his colleagues have an article in press on the same subject.¹ In Dr Goldblatt's table the outstanding decline in post-neonatal mortality was from 1970-2 to 1975-6 in social class V—from 13.1/1000 live births to 8.5.

These years are, of course, exactly those of the early 1970s, when a marked decline took place all over England and Wales.² In Sheffield at one time this decline was thought to be due to "intervention" but we now know that it was widespread, at least in northern England.^{1,3} We believe that this early 1970s fall was due to the disappearance of deaths from acute (viral) bronchiolitis—and that therefore the plateau in the 1960s was due to their persistence. The fact that this major fall took place in social class V at the same time supports this concept fully since deaths from acute bronchiolitis occurred almost exclusively in babies from that class.

As Dr McFarlane says, there has been a further plateau since 1975 lasting up to now. Since many of the babies are certified as dying from respiratory infections these should be "possibly preventable." The fact that they are not being prevented may, however, be due to confusion in the minds of certifying doctors. In 1969, of a postneonatal death rate of 6 per 1000 live births in England and Wales, 3 per 1000 were attributed to respiratory diseases and none to sudden infant death syndrome,⁴ which was only just becoming respectable as a cause of death. By 1984 the figures had become 0.5 for respiratory diseases and 1.8 for sudden infant death as the latter diagnosis became more respectable (the missing 0.7 represented the fall in mortality in the early 1970s).

But we have now reached the stage where we do not quite know what we mean by deaths from respiratory infection. The term bronchopneumonia is often used to explain the death of a baby with a severe congenital anomaly when it is really just a terminal event due to the baby being unable to clear the secretions. But even more worrying are the pathologists who certify some form of respiratory infection after a necropsy at the request of the coroner on a baby who has suffered a cot death.

Not since 1975, in Sheffield at least, has a clinician certified a death in an otherwise normal child as being due to any form of respiratory infection—except acute epiglottitis. But in the past many were so described by the then pathologist. This no longer happens in Sheffield but it does elsewhere. In Nottingham a change in pathologist resulted in a decrease in deaths attributable to sudden infant death syndrome from 40.2% to 24.6% but at the same time the percentage attributable to respiratory infections rose from 28.3% to 43.9%.⁵ As the authors pointed out, the two combined in each case came to 68.5%. So it seems that in cot death one pathologist may certify sudden infant death syndrome and another (mainly with the laudable wish to help the parents) will certify respiratory infection. Since the victims of such deaths are no longer seen by clinicians—as they have been in the past—it seems unlikely that the infections cause cot death only. So now a victim of cot death who at necropsy is found to have quite a mild respiratory infection is really an example of sudden infant death syndrome. On this basis if the 1984 figures for respiratory infection deaths in England and Wales (0.5/1000)⁴ are added to those for sudden infant death syndrome (1.8/1000) we get the figure of 2.3, which is exactly the same as the Scottish figure for unexplained cot deaths.⁶ So if deaths from respiratory diseases are added to those for sudden infant death syndrome and for congenital anomalies (as they will when certifica-

tion improves) this will bring the percentage due to these two conditions up to 90%. And this, of course, explains the second plateau, which will continue until we get some answer to these two conditions—the only improvement so far is with congenital abnormalities.

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- 2 Pharoah POD, Morris JN. *Postneonatal Mortality and Epidemiological Reviews* 1979;1:170-83.
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- 5 Madley RJ, Hull D, Holland T. Prevention of postneonatal mortality. *Arch Dis Child* 1986;61:459-63.
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Dietary supplementation in pregnancy

SIR,—Mr A Malhotra and Mr R S Sawers (23 August, p 465) referred to the first paper from the Sorrento Maternity Hospital concerning dietary supplementation¹ but did not refer to the second,² which immediately followed it in the same edition of the *BMJ*. The papers have similar titles and were obviously intended to be read in tandem.

The Sorrento trials suggested three points. Firstly, unselective supplementation for all Asian mothers during pregnancy is of no clear value,¹ as they quote. Secondly, however, a protein energy supplement when given selectively to Asian mothers (mainly Pakistanis) who had evidence during the second trimester of nutritional stress (inadequate increase in triceps skinfold during the second trimester—that is, some evidence of a compromised energy balance) did enhance birth weight.² Thirdly, a protein energy supplement given to mothers who did not have evidence of nutritional stress did not enhance birth weight and may have had an adverse effect.^{2,3} Selection is essential.

We accept that the numbers were small (but statistically significant; further analysis by another group confirmed this³) and that what applies to mainly Pakistani patients in central Birmingham does not necessarily apply elsewhere. However, a larger study at Sorrento has subsequently confirmed the relation between triceps fat deposition in the middle trimester and fetal growth.⁴ Furthermore, these observations are paralleled by those published from The Gambia shortly afterwards.⁵ A protein energy supplement given to mothers during the wet season, when food supplies are less, work is harder, and energy balance may therefore be compromised, enhanced birth weight. When given during the dry season the supplement did not enhance birth weight; indeed, there was a suggestion of an adverse effect.

The authors are surely correct to emphasise dietary education; the snag is what does one teach? "Eat to appetite," "An extra pint of milk a day," "More protein foods," "More fibre" all appear in various leaflets. Apart from urging a vitamin D supplement to prevent maternal osteomalacia, neonatal hypocalcaemia, enamel hypoplasia, etc (for which the evidence is very strong from two other trials^{6,7} apart from the one they quote⁸), we are not sure what to say specifically about pregnancy. Until we do know what to say perhaps we should concentrate on the vitamin D. It is stated DHSS policy,^{9,10} but few hospitals follow it.

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- 8 Brooke OG, Brown IRF, Bond CDM, et al. Vitamin D supplements in pregnant Asian women: effects on calcium status and fetal growth. *Br Med J* 1980;280:751-4.
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- 10 Department of Health and Social Security. *Present day practice in infant feeding*. London: HMSO, 1980:38.

SIR,—We read with interest the leading article on dietary supplementation in pregnancy (23 August, p 465). We performed a dietary recall survey of 43 women, all between the 6th and 22nd weeks of pregnancy. Thirty two had their plasma concentrations of vitamin D and folate estimated in addition to giving a recall of dietary intake over 24 hours. The racial distribution of the women was representative of the mixed population in west London (81% white, 12% black, 7% Asian). The mean age of the patients was 26 years (range 17 to 41 years).

Average intakes of the major nutrients, with the exception of folate, vitamin B₆, and vitamin D, were all above DHSS (1979) recommended daily intakes for pregnancy. On the other hand, the proportions of patients not having the recommended intakes were: energy 46%; protein 14%; vitamin B₁ 19%; riboflavin 23%; nicotinic acid 40%; vitamin C 28%; fibre 46%; iron 48%; calcium 48%. Of the 43 women, only two were taking the DHSS recommended doses of folate (500 µg/day), only two were taking the recommended vitamin B₆ (2.5 mg/day), and none were taking the recommended vitamin D (10 µg/day). None the less, 27 patients had serum folate values of above 6 µg/l, and 23 had 25-hydroxycholecalciferol levels above 7 ng/ml.

There was no correlation between any of the dietary variables and maturity at delivery, birth weight, or birthweight centile. The proportion of low birthweight babies (<10th centile) was 5%, which is below the national average. No fetal abnormalities were detected.

The DHSS recommendations may therefore be set at too high a level. We have attempted to ascertain their scientific basis without success—apparently they are based on the opinions of a "group of experts."

It is unwise to review the recommendations without more scientific fact. Reduction of dietary intakes to ascertain if any harm resulted would be unreasonable. The only sensible approach is a series of large scale controlled prospective studies in which patients are given modest supplements of an individual dietary constituent to ascertain any possible benefit. Let us get our facts correct on the dietary factors of known importance before looking into plasma molybdenum concentrations.

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Graduated elastic stockings

SIR,—The leading article by Messrs K G Burnand and G T Layer (26 July, p 224) has provoked an interesting correspondence. It is odd that there is great enthusiasm for accurate measurement of the pressures produced by an elastic stocking on devices such as the medical stocking tester but much less for relating what actual effects these pressures have on venous physiology in a limb fitted with a stocking.

Mr S D Blair and others (16 August, p 447) would have been in a position to do this, but their use of ulcer healing alone as an end point is unsatisfactory, especially as they had no control group. Many studies of bandaging with varying types of new dressing produce apparent improvements in ulcer healing. Unfortunately, once these studies are completed the enthusiasm for assiduous care wanes and the ulcers recur.

We still need to know what bandages really do. They certainly promote healing, but is graduated compression really superior to ordinary compression? We often seem to be optimistic about the degree of graduation obtained. We think that it will reduce reflux but have little evidence that this is so; in the papers quoted in support of this by Messrs Burnand and Layer, and in others, the actual changes in ambulatory venous pressures and reflux produced by stockings were minor and disappointing^{1,2}; in my own study I was greatly disappointed by the lack of reduction of reflux.³ Only in the study by Partsch were some values returned to normal, and then significant changes were obtained only by the use of the tightest grades of Sigvaris stockings, 504 and 505.⁴ However, the results are confused because most of these patients had long saphenous vein incompetence, rather than deep venous incompetence. This is shown by the fact that the better results were obtained by digital pressure over the saphenous opening than by the use of stockings. These grades of stocking are rarely if ever prescribed in Britain and are extraordinarily difficult to get on unaided, even by the young and able bodied.

So until a large study relates three factors—changes in venous pressures and reflux, accurate measurements of compression provided by the stocking, and ulcer healing rate—in legs with proved deep venous incompetence, and not just long saphenous vein incompetence, one must be circumspect in believing that graduated compression is the answer. This matters economically, as getting effective graduated support is expensive and venous ulceration is a common disease.

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Obstetrics at the London Hospital Medical College

SIR,—Both before and after the report of the Beaumont inquiry and the welcome restoration of Mrs Savage's honorary consultant contract by Tower Hamlets District Health Authority there has been much criticism in the press of Professor Grudzinskas, professor of obstetrics and gynaecology of this college.

Among other allegations, it is said that Professor