doctors to prescribe oral contraceptives without the knowledge or consent of those parents.

The present so called "guidelines" are a misdirected loophole in the law, which confers on parents the legal guardianship of their children up to the age of 16. The loophole was devised in haste among the Department of Health and Social Security, the General Medical Council, and the BMA to deal with the problem of increased sexual activity in children under 16 years of age. There is no proper mandate from the medical profession or from responsible parents for these "guidelines."

You say "the attitude of doctors would have been very different if the call for legislation had come from the families directly affected—namely, those in which 14 and 15 year olds have been prescribed the pill—or from doctors working with teenagers." In the first place those parents who had not been consulted would not be in a position to call for legislation as they would not know that the pill had been prescribed for their daughters. Those parents who had been consulted and who had consented would not have any reason to ask for legislation. If by "doctors working with teenagers" you mean doctors working in so called "youth advisory clinics" is it not conceivable that they might jeopardise their livelihood by asking for legislation?

When you speak of "a sexual relationship, often stable" at the age of 14 or 15, I am afraid you are very divorced from reality.

I would like to know the scientific basis for your formula of "thirds" when you say: "Most have no wish to keep their mothers in the dark; of those few who do ask for confidentiality, one third can be persuaded at the first interview to tell their parents and another third agree later. The remaining third of girls must believe they have very strong reasons for rejecting the doctor's advice." What criteria were used to establish that the girl had told her parents? Was it just a question of the girl's say so or was there direct involvement of the parents as there is in prescribing other potentially dangerous drugs or performing operations?

After the wide publicity the British Medical Association has given to its "guidelines" every 13 year old girl now knows that if she wants the pill all she has to do is walk into a youth advisory clinic or family planning clinic, tell the doctor she is having a sexual relationship, demand the pill, and say that she does not want her parents to be told about it.

We therefore have oral contraception on demand for children under 16 without the knowledge or consent of their parents. This can only increase promiscuity in young people with the inevitable risks of venereal disease, early carcinoma of the cervix, illegitimate births, and further undermining of the status of marriage and the family.

I would have expected the BMA to ask for legislation to support the role of parents as legal guardians of their children and not to oppose the proposed legislation as they are now actively doing.

Dr Gerard Vaughan, former Minister of Health, writing in *Pulse* (10 December, p 18) and advocating review of the present guidelines states: "It would not only be sensible, but in the overall interest of our society to strengthen the position of parents by saying we will not regulate for the exceptions, but rather for the majority." I am sadly disappointed that such a reasonable statement could not have emanated from the British Medical Association.

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SIR,—The BMA's press statement on 1 December on its opposition to Mrs Victoria Gillick's appeal referred to work by Dr Judith Bury of the Edinburgh Brook Advisory Centre. This was not definitive research and was based on a small number (51) of girls apparently under 16.[1] *The Times* report of 2 December, to which your leading article (17 December, p 1826) referred, stated: "A third agreed to tell their parents at the first consultation."[2] This figure was derived from Dr Bury's impression of the situation in 1983 and does not accord with the findings of the article of January 1980.[3] In 1981 Dr Bury stated: "There is overwhelming evidence that, contrary to what you might expect, the availability of contraception leads to an increase in the abortion rate." This would seem to contradict the claim made in the BMA's statement.

The BMA also failed to point out that no check is made on clients' ages or parental involvement either before or after a visit to a Brook clinic, a fact admitted by Caroline Woodroffe, chairman of the Brook Advisory Centres.

The statistics provided by the Brook Advisory Centres related to the 15-19 year old age group.[3] They did not provide the information that in this age group the illegitimate pregnancy rate had increased from 23·6 of every 1000 girls in this age group in 1970 to 31·9 in 1980 or that the abortion rate had increased from 9·1 to 17·6 in the same years. The highly relevant figures for the under 16s were not given. These again show an increase, the pregnancy rate in 1970 being 1·91 of every 1000 girls in this age group while in 1980 it was 2·55 (in 1982 it was 2·66). The abortion rates in the under 16s increased from 1·04 in 1970 to 1·88 in 1980 (in 1982 it was 2·04) (Office of Population Censuses and Surveys, personal communication).

The birth control campaign circular to MPs (November 1983) restricted its statistics to 15 year olds from 1975 to 1982. It failed to mention that the abortion rate in all under 16s had increased from 1·87 in 1975 to 2·04 in 1982, while the pregnancy rate had remained at 2·7 for both years.

Everyone is entitled to his or her opinion in this debate, but please let us have relevant figures and evidence that is scientific and corroborated rather than speculative and anecdotal.

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[1] Bury JW. Some social aspects of providing contraception for under-16 year olds. *Fertility and Contraception* 1980;**4**:1-6.
[2] Timmins N. All children's treatment threatened by pill challenge, doctors say. *The Times* 1983 Dec 2: 3 (cols 1-3).
[3] Witherow J. Fewer teenagers pregnant. *The Times* 1983 Aug 16:2 (cols 3-4).

The disappearing stammer?

SIR,—We would like to reply to Dr Irvine Loudon's questions on the "disappearing stammer" (3 December, p 1715).

From as early as 1900 studies have shown that the prevalence of stammering is around 1% and it has been reported as high as 4% in the preschool population.[1 2] Recent studies do not show a decreasing trend. The last epidemiological study confirmed the accepted prevalence rate of this disorder among 10-14 year olds.[3] There is no evidence that stammering is caused by "nervousness," although this remains a popular misconception. There is, however, support for the suggestion that both genetic and environmental factors play a part in its aetiology. As rate of recovery is highest in children and diminishes as the problem persists we are concerned with early intervention procedures.

The tendency for specialisation in speech therapy has given us the opportunity to understand the complexities of the management of the dysfluent or stammering child. We can assure Dr Irvine Loudon that according to the high number of referrals we receive stammering is not out of fashion. We hope that our treatment strategies will produce the desired trend towards a more fluent population.

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[1] Andrews G, Harris M. *The syndrome of stuttering.* Clinics in developmental medicine, No 17. London: Spastics Society Medical Education and Information Unit in association with Heinemann Medical Books, 1964.
[2] Bloodstein O. *A handbook on stuttering.* Chicago: National Easter Seal Society, 1981.
[3] Rutter M, Tizard J, Whitmore K, eds. *Education and behaviour.* London: Longman Press, 1970, 10-4.

Who should be an author

SIR,—We should like to comment on the controversy on who should be an author (26 November, p 1569), which has previously raged through the British medical press and is currently inflaming correspondents of their transatlantic counterparts.[1] We are agin multiauthorship; but for the sake of brevity have added, for the last time, our combined weight behind a single communication.

We are wholeheartedly behind the use of the Vancouver style of referencing[2] and hope that this will help to discourage such multiauthorship, particularly as only the first three authors (and the ubiquitous *et al*) are cited in any reference boasting more than six names.

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[1] Burman KD. Hanging from the masted: reflections on authorship. *Ann Intern Med* 1982;**97**:602-5.
[2] International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1982;**284**:1766-70.

Respiratory syncytial virus infection in the elderly

SIR,—On the topic of infection in the elderly with respiratory syncytial virus (26 November, p 1618) the forthcoming edition of the *Journal of Infection* will carry a report of a prospective study of respiratory infections that affected 125 patients in the geriatric wards of the City and Royal Victoria Hospitals, Edinburgh, from December 1981 to March 1982. During this study we detected 12 cases of respiratory syncytial virus infection as proved by a fourfold rise in antibody titre. All 12 cases occurred in the four week period from the middle of

December and all were in the City Hospital alone. Five of these patients died within the next two months. During the same period there were three patients in whom the initial antibody titre was high (256 or greater) and who died within a few days of the onset of symptoms. An additional four patients (one asymptomatic) were subsequently found to have high initial titres that remained raised, and of these only one survived a further nine months, by which time his respiratory syncytial virus titre had fallen to a quarter of its original level. Isolation of the virus was achieved in only one case.

During the same study we detected small epidemics of influenza A and B and in comparison the cases of respiratory syncytial virus tended to have a more prolonged illness; wheezing was a frequent feature, while sore throat was not. None of the cases with only viral infection had either clinical or radiological evidence of pneumonia (this contrasts with 87 out of 192 reported to the Public Health Laboratory Service (26 November, p 1618), though two patients who had proved additional bacterial infection did have signs and radiological evidence of consolidation.

This outbreak of respiratory syncytial virus infection was confined to one hospital and all the proved cases occurred within a four week period. A repeat prospective study carried out over the winter of 1982-3 showed only one case of respiratory syncytial virus infection (this patient died) and 10 cases of proved influenza A.

We conclude that respiratory syncytial virus infections in the elderly are associated with appreciable morbidity and mortality.

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Range of mycobacterial disease

SIR,—Although we do not doubt the interpretation of the case reported by Dr J H Shribman and others (26 November, p 1593), we question the increasing acceptance of the view to which they subscribe—namely, that there is in tuberculosis a clinical and immunological range comparable with that of leprosy. The evidence of such a range in tuberculosis is so far mainly immunological, and though this may be of some importance one doubts that it is fundamental, or that it constitutes a sound basis for classification, unless clinical and histological counterparts can be shown. The range of leprosy was worked out clinically and histologically.[1] The only attempt so far to show a comparable histological range in tuberculosis has shown instead that the vast majority of clinically overt cases are characterised by a fairly consistent histological response[2] (further work is in progress). Only in a few are there signs corresponding to the high or low immune responses of leprosy. As regards clinical manifestations many reports are silent, and anergic cases cannot be recognised except by skin testing.[3]

One apparent similarity between the two diseases, the haematogenous dissemination of miliary tuberculosis and lepromatous leprosy, needs to be treated with caution. Lepromatous leprosy is disseminated often almost from the inception of the infection. Miliary tuberculosis arises from the discharge of a large focus into a blood vessel, and though a low immune state is a predisposing factor the event is partly a matter of chance. Miliary tubercles, therefore, do not present a uniformly low immune histological response. Rich describes "hard" epithelioid cell tubercles with few bacilli and "soft" necrotic ones with many bacilli.[4]

All infectious diseases must be subject to some modification dependent on host responses. For a disease caused by a single species of organism the range of leprosy is perhaps uniquely broad, being the consequence of remarkably low immunogenicity in an organism with an immunologically privileged accommodation with nerve Schwann cells.[2]

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[1] Ridley DS, Jopling WH. The classification of leprosy according to immunity. *Int J Lepr* 1966;**34**:255-73.
[2] Ridley DS. The histopathological spectrum of the mycobacterioses. In: Ratledge C, Stanford J, eds. *Biology of the mycobacteria*. Vol 2. London: Academic Press, 1983:129-71.
[3] Daniel TM, Oxtoby MJ, Pinto E, Moreno E. The immunological spectrum in patients with pulmonary disease. *Am Rev Respir Dis* 1981;**123**:556-8.
[4] Rich AR. The pathogenesis of tuberculosis. 2nd ed. Oxford: Blackwell Scientific Publications, 1951: 828-36.

Allergy to cows' milk presenting as chronic constipation

SIR,—As the parent of a child with a quite severe allergy to cows' milk, I read with considerable interest the report by Dr K C Chin and others (26 November, p 1593).

My third daughter, unlike her siblings, had feeding problems from birth with the usual label of "colic" applied to these difficulties. At about the 18th month of her life she developed constipation, which subsequently progressed to faecal incontinence. After numerous medical consultations (including a barium enema examination) we were gently upbraided for allowing our child to become so constipated, and various powerful purgatives and enemas were prescribed, with no effect.

At that time she was lethargic, appeared to have hearing difficulties, had a watery discharge from her nose, cervical lymphadenopathy, and her abdomen was distended and tympanitic. Her stools were liquid and offensive.

We stumbled on the diagnosis purely by chance and thereafter curtailed not only milk but any food product which contained cows' milk. Her symptoms soon resolved and within a week she passed a normal stool. Since then she has remained well, except when she succumbs to the temptation of ice cream or chocolate.

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Calcium antagonists in hypertension

SIR,—Dr A M Heagerty and others (12 November, p 1405) report the lack of effect of treatment with nifedipine on abnormal leucocyte sodium transport in hypertension. From this they have concluded that a link between calcium and sodium transport is improbable and that sodium transport is therefore irrelevant to the aetiology of the condition. They suggest that the abnormality of sodium transport characteristic of the leucocytes of hypertensive patients may be "an innocent bystander in the cellular changes underlying hypertension." We cannot accept that the failure to show an effect of nifedipine on sodium transport in leucocytes justifies such a conclusion.

In a similar study using the calcium antagonist verapamil we have reported an improvement in leucocyte sodium transport accompanying a reduction in blood pressure.[1] Comparable effects on sodium transport were also obtained by exposing normal cells to therapeutic concentrations of verapamil in vitro. This suggests either that a link between sodium and calcium transport does exist or that verapamil has an effect on sodium transport independent of its effects on intracellular calcium. The failure of Dr Heagerty and others to show a similar effect using nifedipine may be due to two substantial differences between these drugs. Firstly, there is evidence that nifedipine (unlike verapamil) penetrates poorly into cells,[2] and its effects may therefore be dependent on a continued presence in the extracellular fluid, a condition that would not have been satisfied in the studies of Dr Heagerty and others.

Secondly, nifedipine is intensely sensitive to photodegradation, and Dr Heagerty and his colleagues do not mention any precautions taken to shield the cell suspension from light. If the cells were exposed to daylight or ordinary artificial light during the experiment then a spurious negative result could have been obtained as a result of destruction of the drug during preparation of the leucocyte. For these reasons it appears unjustified to conclude that nifedipine has no effect on leucocyte sodium transport.

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[1] Gray HH, Poston L, Hilton PJ, Smith SJ, Markandu ND, MacGregor GA. Reversal by verapamil of the sodium transport defect in leucocytes in essential hypertension. *Clin Sci* 1983;**65**:12.
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****The authors reply below.—ED, *BMJ*.

SIR,—We are grateful for the comments of Dr Gray and his colleagues. To reiterate the conclusions of our paper: we observed no relation between initial changes in leucocyte handling in hypertensive patients and the fall in blood pressure produced by the calcium channel blocker nifedipine. Thus there is a dissociation between calcium-mediated blood pressure changes on the one hand and abnormalities of leucocyte handling on the other. The failure of nifedipine to influence sodium handling was a secondary observation. We would not wish to deny that changes in intracellular calcium can influence, for instance, membrane sodium permeability in some situations.[1]

We are particularly interested to note that in this context verapamil in vitro influences sodium transport in leucocytes. This might suggest that changes in sodium transport induced by verapamil are secondary to a primary abnormality in calcium handling and not, as Dr Gray and his colleagues suggested in the abstract they cite, a consequence of a reduction in circulating concentrations of a