three died.[1] With such a small relapse rate intensive radiological follow up is unnecessary, and four or five chest radiographs within the first two years after treatment are probably ample.

Although we strongly agree that close surveillance is essential in stage 1 teratoma, we question whether computed tomography beyond the initial investigation is required. Although about half of the 15-25% of patients who relapse have para-aortic disease, nearly all of these patients will be detected by virtue of raised tumour markers or associated lung metastases. Repeat abdominal computed tomography is therefore of value only for the few patients who relapse with disease in the abdomen without raised markers. There were no deaths in 45 patients followed up without repeat computed tomography,[2] and this remains true of the current total of 93.

These considerations would not, however, apply if only surveillance were to be considered in stage 1 seminoma since the para-aortic nodes are almost the only site at which relapse is likely to occur and there is currently no adequate marker available for seminoma. These facts together with the expense and the longer clinical course of seminoma support the benefits of a single radiotherapy course with minimal morbidity, after which further follow up is almost unnecessary.

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Trimethoprim resistance in Gram negative urinary pathogens

SIR,—In 1983 I reported a sudden rise in 1982 of trimethoprim resistance in normally susceptible Gram negative urinary pathogens isolated in this laboratory.[1] The rise was sharpest in organisms isolated from general practice patients, and I suggested that this might be due to the increasing use of trimethoprim alone outside hospital. In the two years since then we have persisted in our policy of not reporting the sensitivity of urinary organisms to trimethoprim alone, and I have analysed figures obtained in the same way for October 1983 and 1984 (see table). There has been a gradual decline in resistance, both in hospital and general practice isolates, the overall percentage having now returned to the level of 1979-81.

The original report received considerable publicity locally, and it is tempting to wonder whether the satisfactory improvement might be due to a change in prescribing habits. The ratio of the use of co-trimoxazole to trimethoprim in this hospital in 1982 was 16:1; in 1984 it was 19:1. When I wrote my previous report I had not been able to obtain figures for comparative use in general practice, but I have now compared the figures for the use of proprietary preparations for the year December 1981 to November 1982 with those for the year September 1983 to August 1984. In those areas served exclusively by this laboratory the ratio of the use of co-trimoxazole to trimethoprim was 17:1 in 1981-2 and 18:1 in 1983-4. These figures compare with 8:1 (1981-2) and 3:1 (1983-4) in an area served only by a laboratory which reports trimethoprim sensitivity of urinary pathogens, and 11:1 and 6:1 respectively in two areas which use our laboratory for some of their work. All the 1983-4 figures for co-trimoxazole are likely to be an underestimate because they refer only to proprietary preparations; over the past year there has been increasing use of generic co-trimoxazole and our laboratory figures suggest that generic prescribing now accounts for about 25% of the co-trimoxazole used.

Clearly our laboratory policy has kept the level of trimethoprim prescribing well below that of other areas, and trimethoprim's use in comparison with that of co-trimoxazole has declined over the past two years, in marked contrast to the pattern in areas that have adopted a different policy. The overall level of trimethoprim resistance here has returned to that of 1979-81.

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Percentage of normally susceptible Gram negative urinary pathogens resistant to trimethoprim. Figures in parentheses are number of isolates tested

	1978	1979	1980	1981	1982	1983	1984
Hospital (inpatient and outpatient)	NA	NA	NA	19 (326)	24 (381)	24 (419)	16 (382)
General practice	NA	NA	NA	7 (521)	15 (507)	12 (701)	11 (807)
Total	9 (709)	12 (821)	13 (794)	12 (847)	19 (888)	17 (1120)	12 (1189)

NA = Not available.

Calcium supplementation and postmenopausal bone loss

SIR,—The paper by Dr Nilas and her colleagues (27 October, p 1103) contains data on which it would be helpful to have the authors' further comments, particularly if they have additional data.

(1) Three groups of normal women in the early menopause with low, medium, and high intakes of dietary calcium were given an additional 500 mg of calcium daily. In each group the urinary calcium concentration at first rose over three to six months and then gradually returned to the basal value, or even below in the high intake group. What is happening here? Does the absorption of calcium at first increase and then gradually diminish, and, if so, might this not be important in the management and perhaps the aetiology of postmenopausal demineralisation?

(2) The authors state that all three groups showed a similar fall in bone mineral content over two years. Fig 2 shows quite clearly, however, that in the low intake group the fall occurred over the first 15 months and was then arrested. A similar effect may possibly have occurred in the high intake group after 21 months. Can the authors explain the arrest, perhaps from subsequent follow up of the low intake group?

(3) The medium intake subjects appear to fall into two clusters with respect to loss of bone mineral over two years (fig 1). One cluster suffers a bone mineral loss of 1 to 2% and the other 6 to 7%. The two other groups do not show this effect, but when all three groups are combined the clustering is still present. It is not unusual in osteoporosis to find that data which might be expected to be distributed in a Gaussian fashion show two overlapping populations.[1 2] It was first reported eight years ago that the various clinical types of osteoporosis (postmenopausal, senile, male, steroid, and postgastrectomy) are not metabolically homogenous and that osteoporosis in any individual results from one or both of two general processes, poor absorption of calcium and increased loss of bone mineral.[1] Would Dr Nilas and her colleagues consider that their data might indicate two populations, and perhaps two processes, in the early menopause of normal subjects?

(4) The reader is rather left to conclude that loss of bone mineral will occur despite increasing the intake of calcium. Fourteen of the 103 subjects, however, increased their bone mineral content. In the context of preventing osteoporosis, such individual results must be considered satisfactory; the problem is to identify these subjects beforehand. Can the authors do so? Interestingly, the number of subjects whose change of bone mineral was between −2% and +7% was 37 (35·9%)—roughly the same proportion (44%) as that of postmenopausal women with osteoporosis who were found to have a poor absorption of calcium[3] and in whom extra calcium might help.

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**The authors reply below.—ED, *BMJ*.

SIR,—We offer the following answers to Dr Taylor's questions.

(1) There may be two explanations for the pattern of urinary calcium excretion: either an adaptation in intestinal absorption occurs or the women—although well instructed—lose interest in taking calcium. Neither explanation would support calcium supplementation. As can be seen from our paper we have greater faith in bone mineral content measurements than in measurements of calcium excretion; therefore we certainly do not think that the fall in calcium excretion indicates retention of calcium.

(2) As stated in our paper, there were no significant differences between the groups: when the mean values between groups at each of the eight measurements were tested by analysis of variance only one set of values differed at the 5% level. Dr Taylor should observe that the ordinates in fig 2 are interrupted and mostly show what happens from 100 to 96%. The vertical bars are 1 SEM: they are smallest in the medium group because this group contained more than half the women. According to our statistical analysis the curves are similar—that is, not significantly different. Likewise the regression lines on bone mineral content versus time in the three populations had r values of 0·92, 0·99, and 0·99, indicating linear falls. The vital point in the evaluation of

our data is that the three groups were alike from the start.

(3) We believe that Dr Taylor overrates the importance of a few data points, say the eight points around −6·5% in the medium group. Were four out of these points instead scattered from −7 to −9% then no one would think of bimodality. In fact we cannot determine from the present study whether this population is bimodal. A large number of values are necessary to disprove a Gaussian distribution. Rather arbitrarily we divided "fast" from "slow" losers in early menopause at −3% per year. With the three groups thus divided a χ^2 test showed no difference between the groups.

(4) We compared the 14 women with an increased bone mineral content with other women, and our point was that the "positive difference" was not related to calcium intake. Furthermore, when we compared these 14 women with the 14 with the greatest losses we found that the latter weighed more and had higher concentrations of oestrogens. This is described elsewhere and may show how "fast" losers can be picked up by biochemical means. Again, however, this does not seem to be connected to calcium intake. We know of one way, and one only, of preventing postmenopausal bone loss, and that is hormone replacement therapy.

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Instructions for inhalers

SIR,—For economic reasons there appears to be an increase in the parallel imports of beclamethasone and salbutamol inhalers into the United Kingdom, and these are now being dispensed by high street pharmacists. These imports contain instructions in the language of the country of origin as well as English and other common languages. We have observed that the instructions accompanying these imports are different from those with their home produced counterparts, despite all the inhalers being made for the same parent company. Listed below are the main differences in the instructions on two products.

Italian Becotide	*United Kingdom Becotide*
(1) Hold breath as long as you can.	(1) Hold breath. [No time specified in instructions.]
(2) Wait for one minute if further puff is required.	(2) Wait approximately 10 seconds if further puff is required.
Special instructions	
No information given.	Given to older children and those with weak hands.
French Ventolin	*United Kingdom Ventolin*
(1) Place inhaler in mouth, close mouth around it, and breath out completely.	(1) Before placing inhaler in mouth breath out normally.
(2) At the same time as taking a deep breath press down the canister.	(2) Just after starting a slow deep inhalation press inhaler.
(3) Hold breath as long as possible.	(3) Hold breath for 10 seconds or for as long as is comfortable.

It is obviously important to inform the patient as fully as possible how to use the inhalers as poor technique has been implicated in treatment failure. Some patients who use the United Kingdom inhaler have been confused by the foreign imports and have brought the differences in inhaler technique to our attention. Can the company not decide on the best way to use its inhalers, or are the French and Italians being encouraged to behave differently from the British? Vive la différence!

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Acute diarrhoea in children

SIR,—The primary treatment of children with acute diarrhoea is neither so contentious nor so confusing as Dr H G Easton and Dr T H Hughes-Davies make out (1 December, p 1541). We believe it is essential to clarify several points.

In 1979-80 Morrison and Little found that none of the 181 children admitted with acute diarrhoea to the Chatham Hospital had been offered glucose-electrolyte solution before admission.[1] Three years later, over a similar period, this proportion had risen to 10% of 186 admissions (T M Little, personal communication). In a recent unpublished series in Edinburgh 43% of 242 children admitted with acute diarrhoea had already received a glucose-electrolyte solution from their general practitioners. The latest study from Manchester indicated that most children who had been given replacement fluids at home had received "inadequate volumes."[2] This evidence suggests an increasing use of oral rehydration fluids, but generally in insufficient amounts. This hardly constitutes the "widespread misuse" suggested by Dr Easton.

With regard to food withdrawal, in the Manchester study only 11% of 406 children had had milk feeds or solids completely stopped because of diarrhoea,[2] so it seems that Dr Easton's allegation of starvation by oral rehydration is probably uncommon in Britain. Stopping feeding during diarrhoea is more hazardous for undernourished children in developing countries.

Dr Hughes-Davies suggests that the World Health Organisation should consider a trial of plain water for early diarrhoea. The WHO already recognises a role for water in treatment. Its recommendations for managing acute diarrhoea can be summarised:

Prevention of dehydration—As soon as diarrhoea starts "increase the normal intake of fluids—for example, water, soup, rice water, weak tea, fruit juice, or home prepared sugar and salt solutions."[3]

Rehydration by mouth—If there are signs of dehydration use "a balanced glucose-salt solution,"[4] of which the most widely tested is oral rehydration salts (ORS) solution, which has proved to be both effective and safe when used correctly. This is given in volumes to replace existing water and electrolyte deficits and to maintain any continuing losses. Water or ordinary drinks should also be given to provide for normal daily fluid requirements. (It is accepted that a small proportion of patients, perhaps 5%, require intravenous infusions at the beginning of treatment, and that when oral rehydration salts are unavailable sugar-salt solutions are a valuable alternative.)

Maintenance of nutrition—In addition to the rehydration fluid breast feeding should continue during diarrhoea, other milk feeds can be given diluted with an equal volume of clean water, and soft digestible foods should be given ("there is no physiological basis for resting the bowel"[3]). Extra food in the convalescent period is important to repay the nutritional cost of diarrhoea.[5]

These are the principles of management for acute diarrhoea. They should be promoted widely and applied sensibly, and then the serious tragic consequences of diarrhoea will decline in all countries.

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Can we have safer cigarettes?

SIR,—The suggestion arising from a conference (17 November, p 1374) that advertising should be permitted only for cigarettes yielding less than 10 mg of tar is potentially hazardous. Though weaker than the tobacco industry's continual claim that advertising is needed to promote the switch to lower tar brands, the proposal shares the same untenable premises: that there is clear evidence that low tar cigarettes confer less risk, that such advertising can do no harm, and that there are not available more effective and less dangerous methods of effecting the switch to low tar brands.

There are two proved methods of promoting the switch to low tar: differential pricing through taxation and product modification. In 1978 in the UK a supplementary tax was placed on all cigarettes yielding 20 mg of tar or more. Within three months the market share of these brands fell from 15% to 3%,[1] a change of such speed and magnitude which no advertising campaign could hope to emulate. Accompanied by a total ban on tobacco promotion, the mechanism of product modification has been found effective in Finland. Since the implementation of the Finnish Tobacco Act of 1977, which empowered the health authorities to set progressively lower limits on tar, nicotine, and carbon monoxide yields of cigarettes, the market share of cigarettes of below 11 mg of tar has increased dramatically—from under 5% in 1978 to 31% in 1984. By contrast, in the United Kingdom the market share of such cigarettes has remained static at around 15-17%, as reported by Jarvis at the conference. It is indefensible for the government to agree with the tobacco industry that "the new tar groupings structure . . . should remain suitable (not) less than 10 years" (DHSS press release, 22 March 1984) when a progressive reduction in yields allowed has proved so successful in Finland.

There is no sound scientific basis available on which to recommend low tar cigarettes as a second best alternative to stopping smoking. The comparison of risk ratios for high tar (>30 mg) and medium tar (15-18 mg) is not relevant in considering the possible benefits of cigarettes below 11 mg since the technology of producing such cigarettes requires the use of ventilated filters and more porous papers, which produce lower yields on smoking machines but not necessarily in smokers, who can substantially increase yields by their method of smoking. Smokers of low tar cigarettes do not consume less nicotine than smokers of medium tar cigarettes[3] nor are they less exposed to mutagenic compounds.[4]

There are potentially dangerous side effects of allowing cigarette advertising at all or only for low tar cigarettes. It is simplistic to suggest that advertising merely informs, since it also promotes the cultural acceptability of the product. The