undoubted, and we should applaud the white knight's progress, especially over the concept of an essential drug list (24 May, p 1347). Nevertheless, the WHO's advice should not be above criticism, and its suggestions for primary care treatment in epilepsy are highly questionable, specifically its recommendation that phenobarbitone should be the first line anticonvulsant. Phenobarbitone is cheap and efficacious but has several drawbacks which render it peculiarly undesirable in poorly supervised settings in the developing world.

Firstly, it is a drug of abuse, and in some countries is subject to special controls. I would not relish encouraging the prospect of a black market in government supplied barbiturate among addicts in the slums of the great Third World cities.

Secondly, the safe use of phenobarbitone in epilepsy requires a consistent and reliable supply. The sudden withdrawal of established phenobarbitone therapy in an epileptic is dangerous, with the frequent precipitation of withdrawal seizures and other withdrawal phenomena. The fact that abrupt discontinuation may result in status epilepticus was recognised 75 years ago, within a short time of the drug's introduction, and all experienced physicians will treat even its gradual withdrawal with considerable apprehension. Our advisers in Geneva—many of whom incidentally are not practising clinicians—do not seem to have heeded this lesson. In my experience in developing countries in three continents the availability of phenobarbitone (and indeed other antiepileptic drugs) is notoriously unreliable. In rural hospitals drug supplies are often erratic and intermittent, and in the case of phenobarbitone this may have serious and sometimes fatal consequences. Ironically, the very cheapness of phenobarbitone in some countries exacerbates this problem of availability; because of its low profit margins the manufacturers seldom seem bothered to produce the drug in adequate quantities. These problems of supply are widespread in Africa, Asia, and South America.

Thirdly, there are the problems of the interaction of phenobarbitone with alcohol, a potentially serious public health issue. Fourthly, the risks of death in overdose are greater than with other anticonvulsants. Finally, and very important, are the mental side effects of the drug. In children drug induced hyperactivity is sometimes profound, and at all ages depression, sedation, and intellectual slowing may occur. These toxic effects require careful monitoring, and many clinicians in the West no longer recommend this drug for first line treatment, and certainly not in children.

I sense double standards here. If a drug is not recommended in the West why should it be acceptable in developing countries? This is to place a hierarchy on mental functioning in the developed and developing world, and it is cynical to pretend that this is anything but an expediency on the grounds of cost. It is for individual governments to decide on the funding of health, but the WHO should not be party to such medical compromise.

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Short term high doses of etidronate in Paget's disease of bone

SIR,—The findings of Dr Charles J Gibbs and colleagues (10 May, p 1227) of the effects of short courses of high doses of etidronate (20 mg/kg/day for one month) in Paget's disease are similar in many respects to our own observations,¹ but their conclusions about the value of this regimen differ from ours. The reasons for this seem to relate

to two interconnected issues: the importance of a delay in mineralisation of bone and the apparent association of this effect with the effect of etidronate on bone resorption.

Few would disagree that there are concerns about giving high doses of etidronate (20 mg/kg/day) for more than six months, despite the satisfactory biochemical responses. These concerns arise from the possibility that such doses might increase the risk of fracture. No studies have examined directly the risk of fracture with more modest doses of etidronate, but the available information suggests either a reduced or an increased risk.^{2,3} The risks, if any, of fracture from short and long term treatment have yet to be determined with accuracy.

The fact that high doses of etidronate impair the mineralisation of bone is commonly thought to confer a risk of fracture. This view is supported by experimental observations that long term continuous treatment with etidronate may induce spontaneous fractures in dogs.⁴ Spontaneous fractures are also observed, however, with diphosphonates which do not induce osteomalacia.⁵ Any risk from fracture is therefore probably due to the prolonged and marked suppression of bone formation and the delayed repair of fatigue damage.⁶ If so the clinical importance of a transient delay in mineralisation may be negligible. In Paget's disease the rate of offset of any inhibition of mineralisation is relatively rapid, occurring within weeks of stopping treatment,^{1,6} in contrast with the sustained biochemical control: it was this effect we sought to exploit in evaluating the short high dose regimen. Thus we neither expected nor found that short term treatments prevented transient decreases in mineralisation, particularly since we had earlier shown that the onset of delayed mineralisation occurred abruptly within days of starting etidronate.⁸ Nevertheless, the generalisation of Dr Gibbs and colleagues that the effects of etidronate on resorption cannot be dissociated from its effects on mineralisation is less than accurate since this has been achieved when lower doses are used (5–10 mg/kg/day).⁹

Nevertheless, we would agree that the short term high dose treatment has effects on both mineralisation and bone turnover. The delay in mineralisation is transient and unlikely to be clinically important. Such a treatment strategy would seem to have advantages if the duration of effect of etidronate on the biochemical control of Paget's disease were prolonged and significantly outlasted the effects on mineralisation. Using actuarial analysis to characterise the duration of remission in patients given high doses for one month, we found that the rate of relapse of alkaline phosphatase was not significantly different in patients given high doses for one or for six months (figure). This suggests that extending treatment beyond one month confers no advantage for either the degree of biochemical suppression attained or the duration of the response.

Kaplan-Meier analysis of relapse of hyperphosphataemia after treatment with etidronate 20 mg/kg/day for six months (solid line) and for one month (broken line). In 19 patients given one month's treatment 62 (SE 11)% remained in remission 12 months after the start of treatment compared with 51 (8)% (NS) of 41 patients given the longer treatment. A similar proportion of patients given each treatment remained in remission at 2 years (29 (13) v 20 (7)%). Relapse was defined as a consistent increase in serum alkaline phosphate >25% of minimum values.

Unlike Dr Gibbs and his colleagues, we conclude that the effects of etidronate on mineralisation can be dissociated from its beneficial effects on resorption in that the latter significantly outlast any effect on mineralisation. We therefore defend our original claim that short treatments with high doses of etidronate offer a promising new strategy for the biochemical control of Paget's disease.

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Oral contraceptives and hepatocellular carcinoma

SIR,—Because hepatocellular carcinoma is so rare in Western Europe and North America the investigators of any possible relation with oral contraceptive use face enormous difficulties. Both Dr J Neuberger (24 May, p 1355) and others and Dr D Forman and others (p 1357) recognise many of the limitations of their studies.

The major problem with the first is the estimate of the prevalence of past hepatitis in the two groups; also, current testing for hepatitis B virus fails to identify a significant proportion of exposed individuals. The second study also fails to take account of the incidence of hepatitis and other social and environmental factors or the fact that oral contraceptives may be provided by sources other than GPs, thus leading to an underestimate of oral contraceptive use.

The problem of hepatocellular carcinoma and oral contraceptive use has been investigated for over a decade. A review of 128 cases of hepatocellular carcinoma from 1953 to 1980 concluded that the apparent increase in the incidence of hepatocellular carcinoma in young women could be explained by cases of fibrolamellar carcinoma and that the association with oral contraceptive use was probably coincidental.¹ Forman *et al* reported a small but consistent increase in mortality from liver cancer in young women from the end of the last decade,² but no such trend occurred in other countries with comparable pill use (USA, Australia, West Germany, the Netherlands).

The International Committee for Research in Reproduction therefore believes that a causal relation between hepatocellular carcinoma and oral contraceptive use has not been established. Therefore it recommends further national and

international investigations of the association, including timing of the diagnosis in relation to oral contraceptive use and previous exposure to hepatitis. Even if proved, the risk of hepatocellular carcinoma will not substantially alter the overall risk-benefit balance of oral contraceptives. In particular, deaths due to ovarian cancer averted by oral contraceptives (about 1000/year in USA) more than outweigh possible deaths from hepatocellular carcinoma (maximum 120/year).

Great caution is needed in extrapolating the findings of UK studies to developing countries, where the incidence of hepatocellular carcinoma is 10-20 times higher. There is a strong relation between hepatocellular carcinoma and hepatitis B virus, with up to 80% of such cancers attributable to the virus.² If the higher rates of hepatocellular carcinoma in developing countries are due to prior infection with hepatitis B then the proportion of cases of hepatocellular carcinoma attributable to oral contraceptive use would be lower than in developed countries.

After previous scares in Britain about the pill—about thromboembolism in 1964 and breast cancer in 1983-5—many women stopped taking the pill, and after the 1983 scare the number of abortions in British women rose by 14%. The interpretation of controversial scientific data by the scientists themselves and by the media can lead to serious social consequences in terms of unwanted pregnancies, abortions, and discontinuation of the most effective reversible methods of contraception available.

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SIR,—Until the studies of Dr J Neuberger and colleagues (24 May, p 1355) and Dr D Forman and colleagues (p 1357) reports of malignant hepatic tumours associated with oral contraceptive use had been largely anecdotal. High steroid doses and oral contraceptives containing mestranol as the oestrogen component have been linked to these tumours.¹ An increased risk related to mestranol might be due to a genetic inability to demethylate mestranol in the liver.¹

The use of oral contraceptives has steadily increased in Norway since their use for contraception was sanctioned in 1967. About 15-20% of women of fertile age have used them during the past decade. All presently available oral contracep-

tives in Norway contain ethinyloestradiol in low doses ($\leq 50 \mu\text{g}$) as the oestrogen component. Eight out of nine pills containing mestranol were withdrawn from the market in 1972 and the last, which had a low share of the market, in 1981.

Data from the Cancer Registry of Norway, covering all malignant tumours in the country, were reviewed for 1963-83. The incidence of primary hepatic malignant tumours increased steadily. The incidence nearly tripled for women of all age groups (figure 1). For women aged 15-45, on the other hand, the incidence remained constant at an average of slightly more than one new case every year throughout the period. One would expect that any notable increase in the number of these tumours due to the introduction of oral contraceptives should influence the statistics. Benign hepatomas are reported to occur more often, but not a single case has been reported as a complication of the use of oral contraceptives to the Norwegian Committee of Adverse Drug Reactions, although such reporting may not be reliable.

Number of new cases of primary hepatic malignant tumours in Norway 1963-83 (three year periods).

In view of the well documented beneficial effect on the incidence of endometrial and ovarian cancer, a possible effect on liver tumours remains a minor problem. Furthermore, a possible relation with the mestranol containing pills of the 1960s may have disappeared from the oral contraceptives in current use, which contain ethinyloestradiol and low doses of both steroids.

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Incompetence in medical practice

SIR,—I was amazed, infuriated, and hurt by the letter you published from an anonymous registrar (31 May, p 1459). By publishing that letter and placing it in such a prominent position, you have displayed a surprising degree of bad taste and poor judgment.

I showed that letter to seven resident doctors, two white and five non-white. The whites were non-committal; all the non-whites felt as I did to varying degrees. Some made unprintable comments while others shrugged it off as just another slur on non-white overseas doctors. Everyone

noticed the sentence: "He came to Britain from a developing country and qualified here two years ago." This sentence needs no elaboration. The rest can be deduced. Some of the non-white doctors had either seen or heard of gross incompetence by British born white doctors, and they proceeded to give details. They said that these episodes of malpractice were often covered up and never written about in journals.

There are several other aspects of this letter that call for comment, but I shall limit myself to a few. Firstly, a reference about a colleague is a confidential document intended to be read by consultants only. A registrar should never know the contents of his senior house officer's reference. Secondly, to qualify as a doctor one needs average or greater than average intelligence and a considerable amount of knowledge. I suggest that if that senior house officer really is "inexcusably incompetent," he might well be ill (perhaps mentally ill) or simply inexperienced. A doctor who qualified two years ago can have done only one year as a senior house officer.

Thirdly, I am fortunate in being able to practise at high levels in two specialties. In my capacity as a locum consultant obstetrician and gynaecologist I have witnessed gross incompetence by residents both local and overseas. My action has always been repeatedly to show the residents concerned how a procedure should be done to prevent injury to patients. I have never thought of writing anonymously to a journal about incompetence in a colleague. As a locum registrar in anaesthetics I have had to supervise several senior house officers who were in their first weeks or months in the practice of anaesthesia. I have been frightened by the amazing lack of basic knowledge in practical respiratory physiology and pharmacology in some of them. If the faults are discovered in a clinical setting I simply gently take over and quietly discuss the relevant topic. Frequently, the colleague benefits from this and I often learn or revise something as well. This is the way to get rid of incompetence. Thus I suggest that our anonymous colleague should get his boss to ask the personnel department to extend his senior house officer's contract and then he should get on with the business of teaching him but should not cover up for him. Allowing him to get another job elsewhere is simply passing the buck, which ensures that only the patients suffer in the end.

Fourthly, your anonymous writer's idea of setting up a committee to "shadow" training is as daft as it is banal; and he poses questions that point to the absurdity of a shadowing committee. Finally, I think there is always something unsavoury about an anonymous letter and feel that if the writer cannot append his name to it, then it is probably best not published.

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** We made the decision that the letter should be printed anonymously, not to protect the writer but to obscure the identity of the senior house officer. —Ed, *BMJ*.

SIR,—Your anonymous registrar seeks a didactic answer to a problem of professional incompetence in a junior colleague and from my experience as clinical consultant and manager of a haematology laboratory I offer the following advice borne of practical experience.

Professional conscience must override social conscience in this matter. The registrar should first inform his junior colleague that specific actions have been unsatisfactory and offer guidance if he wishes to accept the advice. Then any failings must be documented and further findings cross