additionally breast fed by a lactating relative or the child's natural mother.

In my experience in Papua New Guinea this method worked well in all cases in which the adoptive mother was motivated and received the necessary support from her family. Side effects of the drugs used were rarely complained of, and only once was it necessary to add methyldopa in the sequence as described above.

Clearly, in industrialised countries, where induction of lactation is not considered to be essential for the survival of the infant, the relative importance of side effects and concerns regarding excretion of the drugs into the milk are greater. Nevertheless, at least some of the aspects of the above method could well be adopted in industrialised countries.

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Author's reply

EDITOR,—Padraig Kramer's recommendations on the use of oestrogens, metoclopramide, and chlorpromazine to induce lactation are helpful, and I too have used chlorpromazine for this purpose, in Africa. Opinion seems to be divided over the drugs' value in Western countries, and drugs other than nasal oxytocin (not now available in Britain) are little used in Britain, though they are used in the United States. All the drugs mentioned boost prolactin concentrations but are less effective in increasing the output of milk and may be of mainly psychological benefit. Other methods seem to be equally effective in helping relactation and have fewer side effects. Documented trials, however, are scanty. The drugs mentioned are not licensed for use in inducing lactation in Britain.

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Accuracy of local data on skin cancer

EDITOR,—The paper by Clive Richards and colleagues on underregistration of melanoma in the South Western Cancer Registry¹ was published alongside our paper reporting trends in registrations of melanoma in the Oxford registry.² This naturally raised doubts in the mind of some readers about the validity of our findings. In particular, Dafydd L Roberts questions whether an artefact due to underreporting could have exaggerated the apparent decline in melanoma in women from 1988 to 1992.³

Apart from the fact that sex specific artefact is hard to explain, we can respond to Roberts, and other similarly sceptical readers, on several counts. Throughout the period of our study the Oxford Cancer Registry routinely received reports from pathology laboratories, except for one of the eight districts. The paper by Richards and colleagues shows the value of using pathology records to identify melanomas, if indeed this has ever been in doubt. From 1992, the reporting process in this region has been largely automated by use of systematised nomenclature of medicine (SNOMED) codes to identify potentially regis-

trable cancers.⁴ Thus a process similar to that used by Richards and colleagues to validate the South Western statistics has already been adopted for routine registration.

With respect to our study of melanomas, there were specific concerns about completeness in only two districts. Staff of the cancer registry therefore visited the relevant laboratories and reviewed all pathology reports for the periods in question, looking for melanomas. This laborious process added to the number of registrations of melanoma in those districts by about 10% but did not alter the secular trends. As a further test of completeness we obtained a list of patients with melanoma diagnosed in a single year (1992) from a comprehensive diagnostic register held by a dermatology department in one of the districts. Only two confirmed cases (5%) were not already registered, both in private patients (one male and one female).

It would have been helpful if Richards and colleagues had pointed out in their paper that the South Western registry was one of only three registries in England that were not using pathology reports routinely for registration in 1989.⁵ Clearly, it would be wrong to generalise from their study to those cancer registries that do use such reports.

We are confident that the trend in incidence in women in Oxford is not an artefact, and a similar trend has now been reported by another registry (North Western). Why this trend occurred and whether it will be sustained remain open questions.

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Targets for glycated haemoglobin

Glycated haemoglobin targets are attainable

EDITOR,—Chris Butler and colleagues criticise the targets set for glycated haemoglobin concentration by various organisations and guidelines on the grounds that they are not based on normative data and seem to be largely unattainable in clinical practice.¹ The authors are incorrect on both counts.

The targets set by the British Diabetic Association and St Vincent Declaration have a common history, which can be most easily traced to the consensus guidelines of the European Non-Insulin Dependent Diabetes Mellitus Policy Group published in 1988.² Those guidelines, adopted by a consensus meeting of over 200 European doctors, were compared by the policy group's chairmen with local results. At the time (1988), using an assay of haemoglobin A_{1c} (normal concentration <7.5%), the Newcastle service was achieving results in the "good" or "acceptable" range (<8.8%) in 64% of people with non-insulin dependent diabetes and in 44% of those with insulin dependent diabetes. Results achieved in primary health care in Newcastle (non-insulin dependent diabetes: 62%) were similar.³ Furthermore, in a progressive disorder such as non-insulin dependent diabetes the figures for satisfactory control will always be diluted by patients approaching the next step of intervention treatment.

Nevertheless, the group with "poor" control was

subsequently split into two ("poor" and "very poor") by the joint audit working party of the Royal College of Physicians and British Diabetic Association, so that analyses of population distributions could be more informative.⁴ In the working party's paper data from a hospital in southern England were reasonably evenly distributed among the four groups.

In 1993, under the auspices of the St Vincent Declaration initiative, the guidelines on non-insulin dependent diabetes were revised, and the new European Insulin Dependent Diabetes Mellitus Policy Group liaised with the initiative to produce identical cut off values.⁵ At that time the categories were again reduced to three and the cut off values raised slightly, partly in recognition of the problem that assays of haemoglobin A_{1c} gave different (higher) distributions of patients for glycated haemoglobin than assays of haemoglobin A₁.

More importantly, even before the results of the diabetes control and complications trial were available it was evident that the relation between glycated haemoglobin and microvascular complications steepened considerably at a haemoglobin A_{1c} concentration of around 7.5%.^{6,7} With this assay (normal concentration <6.1%) 65% of people with non-insulin dependent diabetes and 39% of those with insulin dependent diabetes achieve satisfactory blood glucose control (good plus borderline control) in our service. Without such targets some of these people would be denied freedom from the risk of microvascular complications and the many with concentrations just above the cut off value would face much higher risks.

The guidelines accept that target concentrations are not achievable by everyone and emphasise that therapeutic targets must be adjusted for individual people in accordance with other factors, such as personal experience of risk of hypoglycaemia. At present, review of any person with diabetes with a haemoglobin A_{1c} concentration over 7.5% requires consideration of whether blood glucose control can be optimised further.

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Targets set reference points for clinic comparisons

EDITOR,—Chris Butler and colleagues show impressively how systematic collection and use of case based information can help identify problems in the search for improved quality of care in diabetes.¹ Pointing to the difficulties with setting targets in the treatment of diabetes and in its care, they argue that targets for haemoglobin A_{1c} concentration may be set more on an idealistic

than on a realistic basis. They also argue that inappropriate targets, in many instances, may do more damage than good to the ultimate goal of reducing morbidity and mortality through improved metabolic control.

A central question about setting targets is whether they should be "visionary" or attainable by a certain proportion of the people concerned. On the one hand, there is wide agreement that the development of long term complications of diabetes is related to the degree and duration of hyperglycaemia in the same way that the likelihood of developing lung cancer is related to the number of cigarettes smoked. On the other hand, as good medical practice is about a person and not a disease, different targets will often be set for a given person with diabetes than are set for the population with diabetes in general. The doctor and the well informed person with diabetes, having discussed the possible implications of the decisions, should agree on a level of glycaemic control that is appropriate for the person.

Thus the doctor must decide to what extent overall poor glycaemic control (as shown by the haemoglobin A_{1c} concentration) in a local population should set the standard for an individual person with diabetes. Might it be possible to improve the glycaemic control by renewed efforts, such as referring the patient to a colleague, extra education sessions with the diabetes nurse, or participation in a summer camp?

The real question is whether the authors' work to develop the quality of treatment and care in diabetes, with each general practice receiving a confidential report enabling it to compare results from year to year, has resulted in improvement. Although the paper refers to improved results for indicators of both process and outcome, no results are presented and nothing is said about any difference among centres.

In the St Vincent Declaration action programme one of the mechanisms for improving quality is anonymous comparison of indicators of process and outcome from different centres and practices by the method of "benchmarking." Chosen indicators in diabetes are ranked in such a way that each centre, but no one else, will know its position compared with that of others. The best will be asked to identify themselves for others to learn from them. So far, anonymised information on about 15 000 people with diabetes from about 20 countries in Europe has been collected (third St Vincent meeting on diabetes care and research in Europe, Athens, March 1995).

Should targets set for heavy smokers be different from those for people who smoke less, or should stopping smoking be the target for all? Should targets depend on what each practitioner judges to be attainable or should they be visionary and based on the results of scientific documentation? Are targets set after everything reasonably possible has been done to improve glycaemic control in those people likely to benefit, or has the status quo been accepted too early?

The diabetes control and complications trial showed that improvement is possible and worth striving for.² It is therefore imperative that data should be collected and used to improve not only the performance of the patients but also that of the doctors.

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Targets are suggestions to be modified

EDITOR,—In presenting the results of their audit of glycaemic control in the diabetic population of South Glamorgan, Chris Butler and colleagues report yearly recording of glycated haemoglobin in 76% of an estimated 70% of the total diabetic population. Mean haemoglobin A_{1c} concentrations were 10.5% and 10.0%, depending on the assay used.¹ Although the paper's title refers to a comparison between local and external targets for haemoglobin A_{1c}, the authors do not state what these local targets were or how they were reached. International and British guidelines advocate measurement of haemoglobin A_{1c} concentrations at least yearly and ideally every three to four months, and they set target concentrations for good control of less than 6.5%.^{2,3}

Butler and colleagues claim that these targets are inappropriate and not attainable in their population. They have, however, misinterpreted the guidelines. The guidelines on both insulin dependent² and non-insulin dependent diabetes³ state that these are strict targets for ideal long term control and may be inappropriate or undesirable for individual patients. In both guidelines the need for individualising targets after discussion with the person with diabetes is strongly emphasised. The Insulin Dependent Diabetes Mellitus Policy Group specifically states that its guidelines are suggestions and should be adapted to local circumstances.

The diabetes control and complications trial has shown a reduction in the risk of the development of progression of microvascular complications of at least 50% with a drop in haemoglobin A_{1c} concentration from 9.0% to 7.0%—that is, from poor to borderline control according to the international guidelines.⁴ If attempts are not made to achieve this worthwhile reduction, health care professionals may run the risk of being accused of providing inadequate care. Rather than deride the international and national guidelines, perhaps we should use them to organise our current resources better, to the benefit of all.

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Author's reply

EDITOR,—We are heartened by the responses to our paper, which questioned the wisdom of setting targets for glycaemic control in the absence of normative data and an understanding of the psychology of motivation. P D Home asserts that the targets were based on normative data. Unfortunately, the data he quotes from the Newcastle hospital sector do not seem to be available for scrutiny, and general practice data were obtained from 186 patients in four practices specially selected for their interest in diabetes and audit.¹ We found several consensus statements about targets but no normative data in the documents Home cites. In contrast, our conclusions were based on a district-wide approach, involving 37 practices and 3022 patients. We also considered

data on a further 2880 patients from four other centres.

Home's experiences regarding the attainability of targets for large numbers also run counter to ours and to those of the diabetes control and complications trial, in which less than 5% of patients receiving intensive treatment achieved control that would be categorised as "good" by the targets.² Home seems to suggest yet another target, based on the trial's finding that microvascular complications rise steeply with a haemoglobin A_{1c} concentration above 7.5%. But if the original targets are well grounded why are haemoglobin A_{1c} values between, say, 7% and 7.5% (which are now so clearly associated with improved outcomes) classified as "poor" by the targets Home defends?

Questioning targets does not imply a lack of support for optimal metabolic control; it simply means that we doubt the value of one approach based on metabolic idealism. With regard to our local targets, our paper makes it clear that these are based on the previous performances of the practices and comparison with peers.

It may be helpful in the future to be explicit about the purpose and status of any target. If general systems theory is used as a framework,³ this could be: (a) longer term, population targets based on normative data from representative samples that may be used as in the Health of the Nation; (b) consensus targets based on experts' views that set metabolic ideals for the single minded; and (c) targets, based on negotiation with individual people, that recognise the relevance of metabolic ideals, informed choice, the psychology of setting targets, and people's contexts. Those of us who work in primary care will find the third category most helpful, ethically sound, and the least maternalistic.

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Malaria prophylaxis

Mefloquine toxicity should limit its use to treatment alone

EDITOR,—Reviews of chemoprophylaxis against malaria for travellers are welcome; numerous problems surround the advisability of current regimens.¹ Two recently published studies have supported the advisability of using mefloquine for short term chemoprophylaxis in sub-Saharan Africa.^{2,3} As a result, the authors of the recent update advocate use of mefloquine for most parts of this continent even though the combination of chloroquine plus proguanil remains moderately effective in most countries under consideration.⁴

Safety and freedom from side effects are of paramount importance in chemoprophylaxis and should override complete efficacy; no regimen even approaches 100% prevention against infection with *Plasmodium falciparum*. Advocates of widespread use of mefloquine have produced figures purporting to support a rarity of side effects (in particular neuropsychiatric ones), which are seemingly far less common when this agent is used in chemoprophylaxis than when it is used in chemotherapy.⁵ A great deal of clinical experience