The sensitivity of most PTH assays currently available is such that the PTH concentrations present in normal subjects are either unmeasurable or very close to the limits of detection of the assay and hence subject to serious artifactual errors. In their previous paper¹ describing the assay the serum PTH concentration in normals is reported as being less than 400 ng/l, concentrations which, according to their standard curve, would have caused less than 10% displacement of tracer in the assay and hence giving results which we feel are of very dubious significance and perhaps more safely considered as being undetectable. Their view that "any detectable immunoreactive PTH in peripheral blood is considered raised in hypercalcaemic patients" has to be questioned, as the finding of apparently normal levels could easily be produced by very slight non-specific changes in binding in the assay. As in their previous publication 25% of patients with proved hyperparathyroidism had levels below 400 ng/l the measurement of serum PTH may be of dubious significance or frankly misleading in up to a quarter of cases. In addition Dr Posen and his colleagues found that a further 5% of hypercalcaemic patients with elevated PTH levels had no abnormal parathyroid tissue at operation. Whether they consider their diagnosis of hyperparathyroidism to have been wrong or the surgical exploration inadequate they do not tell us. Should it be the former, then the case for PTH assays becomes even weaker.

Excluding patients with overt malignancy, the great majority of patients with hypercalcaemia seen in routine medical practice have mild elevations of the serum calcium level. As there is a reasonable correlation in hyperparathyroidism between serum calcium and PTH concentrations, it follows that it will be in this large group of mild hypercalcaemics that the assay will be most frequently unhelpful or actually misleading, a point rarely understood by the requesting physician or surgeon.

One of the most difficult differentiations in our experience is that of hyperparathyroidism from a case of occult malignancy. The incidence of elevated PTH levels in malignancy has varied very considerably in different series, but the finding of 25% in the Posen series means that PTH levels cannot reliably differentiate malignancy from hyperparathyroidism except when the hypercalcaemia is relatively severe and the PTH levels are undetectable—probably the minority of cases.

The value of PTH measurements early on in the management of hypercalcaemia of hyperthyroidism, in surgical hypoparathyroidism, and renal failure, we feel, is minimal or non-existent.

While we agree that the PTH assay may occasionally be necessary to confirm the clinical diagnosis and that in certain patients neck vein catheterisation carried out by a radiologist experienced in the technique may be a useful procedure, nevertheless the assay can be quite misleading unless clinicians appreciate its deficiencies, most of which are alluded to in the paper by Dr Posen and his colleagues, but perhaps not sufficiently emphasised.

There is nothing in this paper to dissuade us from our view that serum PTH levels are not required for the investigation of the majority of cases of hypercalcaemia or of renal stones. Where doubt concerning the diagnosis exists it is better for the patient to be assessed by a clinician with experience in the diagnosis

and management of hypercalcaemia who may occasionally require a PTH assay for the elucidation of the case. Future improvements in the PTH assay may perhaps enable us to change our views.

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¹ Kleerekoper, M, et al, *Clinical Chemistry*, 1974, 20, 369.

SIR,—We would like to comment on the recent comprehensive account by Dr S Posen and others (3 January, p 16) of the clinical value of serum parathyroid hormone (PTH) assays. One point made in their paper merits further emphasis—namely, that any detectable immunoreactive PTH is considered to be abnormal in hypercalcaemic patients. In other words, patients with primary hyperparathyroidism may have serum PTH values within the "normal" range. But this normal range is, of course, in subjects who also have normal levels of serum calcium. We would therefore urge those who report the results of PTH assays not to present them in the form "serum PTH 0.6 µg/l (normal range less than 0.9)" without further comment. Such a value in a hypercalcaemic patient would be inappropriately high, but it could easily and wrongly be interpreted as evidence against primary hyperparathyroidism.

In deciding whether or not to explore the neck of a patient with hypercalcaemia serum PTH assays may sometimes be helpful in another way by discriminating between primary hyperparathyroidism and the ectopic PTH syndrome of malignancy¹ (in the latter condition serum immunoreactive PTH levels tend to be lower for any given value of serum calcium). It is a pity then that the authors, rather than considering just one value (in the text), did not publish a graph relating serum PTH to serum calcium in the two conditions over the whole range of observed values. If all laboratories performing PTH assays could construct their own graphs of these relationships the usefulness of the assay in clinical decision-making might be further enhanced.

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¹ Benson, R C, et al, *American Journal of Medicine*, 1974, 56, 821.

SIR,—I am concerned at the low value placed by Dr S Posen and his colleagues (3 January, p 16) on venous catheter studies in the diagnosis and location of overactive parathyroid tissue and would like to comment.

The catheterised patient to whom they refer (case 3) had a left innominate vein hormone gradient and a left upper adenoma. This emphasises the fact that thyroid venous drainage occurs predominantly via the inferior thyroid veins into the left innominate.¹ Thymic and other mediastinal veins may also drain into the innominate; hence the poor localising value of an innominate gradient. For an informative study it is mandatory to catheterise not merely the innominate but also the inferior thyroid veins.^{1 2} The potential

morbidity of venography, unlike arteriography, is very low—no appreciable morbidity occurred in several large series reported.³⁻⁵ The fact that bilateral thyroid vein hormone gradients occur in normal subjects⁶ does not detract from the usefulness of the procedure. Since hypercalcaemia will suppress normal glands,¹ any hormone gradient in a patient with hypercalcaemia is indicative of overactive parathyroid tissue.

The procedure can resolve other problems mentioned by the authors. Correctly performed, it can permit preoperative differentiation between an adenoma (unilateral gradient) and hyperplasia (bilateral gradients).¹ Mediastinal adenoma has been suggested preoperatively by finding absence of a gradient in either inferior thyroid vein in a patient with hyperparathyroidism.¹

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¹ Powell, D, et al, *New England Journal of Medicine*, 1972, 286, 1169.

² Shimkin, P M, et al, *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine*, 1973, 118, 654.

³ Bilezikian, J P, et al, *American Journal of Medicine*, 1973, 55, 505.

⁴ Tomlinson, S, et al, *British Journal of Surgery*, 1974, 61, 633.

⁵ Eisenberg, H, Pallotta, J, and Sherwood, L M, *American Journal of Medicine*, 1974, 56, 810.

⁶ Shimkin, P M, and Powell, D, *Annals of Internal Medicine*, 1973, 78 714.

Hospitals for the developing world

SIR,—Your leading article on this subject (8 November, p 309) states that "the cream of the medical graduates from developing countries, who have basked in the facilities" offered by the big hospitals in the western world "have persuaded their governments that similar structures are needed in their own countries." Allow me to say that such an assumption is misplaced. I come from the developing world myself and know that, while doctors who have been trained in the West appreciate the opportunities they themselves have been offered, few are so ill-advised as to imagine that their countries can afford either the capital outlay or the recurrent expenditure which such "vast structures" involve. And to those so naive as to have recommended their construction I would still allow the privilege of sharing the blame with their teachers, who must have failed to impart reason.

Anyone familiar with conditions in the developing world must bear witness to the fact that so far most advisers have not been indigenous and that it is the expatriate experts and consultants who have been largely responsible for the "vast structures," the need for which you have perhaps correctly questioned. To my personal knowledge the British General Medical Council was at one time advised by expatriates to withhold recognition from the Makerere Medical School in Uganda until the new hospital was built. To blame indigenous medical advisers for the new Mulago Hospital would therefore be unfair. Furthermore, would the money that built Mulago Hospital have been made available for something else rather less impressive? I doubt it.

This brings me to another aspect of the problem. While the developing world still needs external aid, the donor countries have retained the right of choice as to what projects qualify for support and, as always, he who pays the piper calls the tune. Almost invariably a country in need of aid has to modify its plans to meet the requirements of the donor and as a short cut some have in fact had to shape development plans for sale. Where aid is concerned motives are often mixed, and the

possibility of separating aid from politics is remote. On this score, for example, would the United States be content to build 30 health centres in relatively inaccessible rural areas while a large USSR-built hospital stood supreme in the capital? Publicity thus tends to carry more weight than utility, and size is made a virtue. And it must be admitted in fairness that indigenous authorities have often been swayed by the favourable domestic publicity that "vast structures" can bring. Nevertheless, the indigenous adviser has so far played a limited part in deciding to build them.

One possible solution would be for donors to channel most of their aid through international organisations such as UNDP, UNICEF, WHO, and the Commonwealth Fund for Technical Co-operation. Aid would then lose much of its national identity and comparisons would be limited to the amounts contributed rather than to the grandeur of structures built, and Third World planning could then be geared more closely to need.

Perhaps developing countries should stick firmly to their own plans and priorities and seek external aid on a "take it or leave it" basis. There are few countries which can afford to do this, however, and when cases arise in which "vast structures" are the result it is a mistake automatically to place the blame for them on indigenous advisers.

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A place to be born

SIR,—We have read with concern your leading article of 10 January (p 55) because it suggests that 100% hospital confinement is a desirable objective—thereby removing from women the possibility of choice—although the evidence for this is far from conclusive.

You imply that the reduction in neonatal mortality has occurred as a direct result of increasing hospital confinement. This is an over-simplification, for other factors such as higher standards of living, improved general health, size of families, and lowering of child-bearing age have contributed to this reduction quite apart from the higher standards of maternity care, whether carried out in hospital or at home. Sweden, with 100% hospital deliveries, has the lowest perinatal mortality rate, closely followed by Holland, with a more than 50% domiciliary rate, suggesting that it is not the place of confinement but rather the quality of life and care that is the critical point at issue.

The study by Dr C A Cox and others (10 January, p 84) gives an account of poor prenatal selection and some instances of questionable medical practice, but this is not in itself an argument against domiciliary confinement any more than examples of poor hospital care argue against hospital delivery.

At present there is an approximately 90% hospital confinement rate in England and Wales, but independent studies have shown that 80% of women who have experienced childbirth in both hospital and home preferred birth to take place in the family setting. As soon as one accepts that human and emotional factors are important elements in childbirth the problem arises as to how the balance between the different considerations for care can be achieved. We fully share the concern of all for the safety of mother and baby; however, if physical safety measured in terms of

mortality and morbidity becomes the overriding preoccupation there is a real danger that other important factors will be disregarded.

The critical question we would like to pose is, How are the decisions concerning the management of childbirth to be made? As a group looking at this question we have become aware that it is one in which various disciplines, together with the personal views of the mothers themselves, have an important contribution to make.

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SIR,—I was relieved to read your leading article on this subject (10 January, p 55). I consider its timing particularly appropriate as there seems to be a growing wave of nostalgia for a return to the "good old days" of home delivery, a trend which many paediatricians and obstetricians view with great anxiety. In my view a recent series of articles in the *Sunday Times* and a current series on BBC "Nationwide" have painted an altogether too rosy picture of the joys of having a baby in an atmosphere of friendly domesticity and informality as compared with the noisy, lonely, and impersonal surroundings of a large maternity hospital. Too often the feelings and wishes of the mother and her attendants, although important, are given precedence over concern about the safety of the baby. We even read statements written by individuals whose qualifications indicate that they should know better suggesting that forceps and breech deliveries can be carried out relatively easily in the home. Many of us who work in large maternity hospitals are uncomfortably aware of their deficiencies, but we must get our priorities right.

We all know that a number of very serious complications can occur during labour, even when the pregnancy has been entirely uneventful and a normal birth is expected. These may require the attention of staff and use of facilities that are available only in a properly equipped hospital. A potentially normal infant who dies or is crippled because of delay in getting such help represents a tragedy that can and should be prevented. The past few decades have seen major improvements in perinatal care based on increased understanding of the "what, why, and when?" of obstetric and neonatal complications. Unfortunately, too little of this knowledge is currently being applied throughout the country, and recent financial and staffing trends in the NHS make me very pessimistic that it ever will be. The trend in perinatal mortality has been most encouraging, but we still have a long way to go. I am afraid that over the next few years we may see that trend reversed. Failure to provide high-quality centralised maternity facilities and encouragement of home confinements are two ways of increasing perinatal mortality and morbidity. Our efforts must be directed towards improving maternity hospitals (including the attitudes of the staff) so that labour and delivery can not only be safe for the baby but a satisfying emotional experience for the

parents as well. Better facilities will help, but we can make a simple beginning by insisting on "family-centred maternity care" in which fathers are not treated as outsiders or visitors but are welcomed as part of the family unit.

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Carrier solutions for low-level intravenous insulin infusion

SIR,—Dr P H Sönksen (17 January, p 151) criticises the recommendation in our book¹ that albumin does not need to be added to the insulin infusion in the treatment of diabetic coma. He concludes from experimental work on the absorption of insulin in high dilutions to glass and plastic that human albumin should always be added to the insulin infusion.

The new treatment of diabetic ketoacidosis with low-dose insulin infusion owes much to the work of Dr Sönksen, who, with his colleagues,² showed that very low doses of insulin were metabolically active. In one of the original reports of the results of this treatment, of which Dr Sönksen himself was a joint author,³ it was stated that the rate of fall of blood glucose was the same in cases treated with and without the addition of human albumin (see figure).

It was for that reason, and because of our experience of the technique with about 100 cases since then, that we said in our book, and still think, that the addition of human albumin is not necessary. Perhaps instead of baldly saying that insulin in the concentration we use (about 1 U/ml) "does not adhere" to plastic we should have added the words "to a significant extent." At higher dilutions (as when insulin is added directly to the infusion solution, which gives a concentration about one-hundred times less than we use) adsorption may be important. That would be an extra reason, in our opinion, for preferring the infusion pump technique.

Diabetic ketoacidosis is a serious condition and its treatment always demands careful monitoring. With the continuous low-dose infusion technique a change in dose can be made (simply by altering the infusion rate) according to the patient's response, but in practice it is rarely necessary to do so. The