redress such an imbalance, but little has been achieved in this field as yet.

One can appreciate that protests will come from those threatened with cuts in their luxurious standards, but how can they justify the fact that our patients in the North pay the same taxes yet receive very different services? Were we in an idealistic world then the obvious solution would be extra funding to the underprivileged regions, but the financial crisis of the day surely demands a fairer spread of the resources currently available.

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Mobile chest radiography

SIR,—In 1967 mass miniature radiography was discontinued in the Bournemouth area owing to the low yield from a large number of examinations. A mobile unit continued (at weekly intervals for general practitioner referrals), visiting 16 sites from Amesbury in the north-east to Portland in the west. In addition, the unit is used for contact examination when a case of tuberculosis occurs in a factory or other sizeable institution.

Although essentially a screening procedure, the abnormality rate is appreciable—approximately 90 per 1000 for GP referrals. It is not suggested that all these patients would never have been x-rayed, but certainly they reach the consultant much more rapidly than through the usual means of referring for a chest x-ray, waiting for the report, and waiting once again for a hospital appointment. Among the less common abnormalities discovered by this method are coarctation of the aorta, arteriovenous fistula, right aortic arch, Meigs's syndrome, medullary carcinoma of the thyroid, and Hodgkin's disease.

It is now suggested that these units should be phased out on the grounds that there is an increased dose of radiation, but this is so small that it can be disregarded. It is said that they are expensive to run, but looked at from a broad point of view this is not true. The load taken off the hospitals is considerable, as is the saving of x-ray film. If a unit travels to the patients it saves petrol and decreases traffic congestion and car parking problems. The saving in production at a factory employing hundreds of people is considerable, as if the employees travelled to the hospital it would mean a day's loss of work. Another important factor is the small amount of space required to store the films, thus enabling them to be kept for a much longer period, and we all know the value of comparison with a previous film when an abnormality is detected.

A mobile chest radiography unit is of great benefit to the community and, so long as the yield remains as high as at present, it should not be curtailed in any way whatsoever.

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The end of excellence?

SIR,—Dr J W Todd writes sensibly in his letter (6 November, p 1130) in which he asserts that most patients are not helped by being treated in an institution filled with the

latest technological hardware and staffed with a galaxy of talent. However, the Hospital for Tropical Diseases in London is one of the genuine examples of a "centre of excellence." Tragic medical mistakes are frequently made in the management of tropical diseases in general hospitals. In Britain doctors have been sued over the past few years for negligence concerning patients who have died from malaria; patients die when ulcerative colitis rather than amoebic dysentery is diagnosed; and cases of leprosy and leishmaniasis are often missed.

This hospital, the only hospital in Britain primarily devoted to the treatment of tropical diseases, is a centre of excellence for the management of amoebiasis, filariasis, intestinal infections, leishmaniasis, malaria, leprosy, parasitic diseases, other protozoan infections, schistosomiasis, and other worm infections. The term "centre of excellence" is valid when applied to certain units throughout the world.

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SIR,—Your edition of 4 December (p 1376) carried an open letter from Dr Terry Davies to Sir Alec Merrison, chairman of the NHS Royal Commission, in which he rightly reiterated the singular contribution of teaching hospitals as centres of excellence and initiation in research and treatment. It has suddenly become fashionable to decry the lead such centres have given and so it is as well to list the result of a random survey of contributions in the same edition of the *BMJ*. Of the 15 signed articles, 10 were from teaching or university hospitals, two from other hospitals, and three from abroad. In the *Lancet* of the same date there were six from teaching hospitals, three from others, and two from abroad. It all seems to prove Dr Davies's point, and one the public at large may not fully appreciate.

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Propranolol absorption

SIR,—We were interested to read the suggestion of Dr B T Cooper and others (6 November, p 1135) that changes in the intestinal microclimate may affect drug absorption. While accepting this possibility, we feel that alternative explanations are more likely to account for reports of altered plasma propranolol concentration-time curves in coeliac and Crohn's diseases.

Plasma propranolol concentrations measured after oral dosing are affected by many factors which include: (1) the site and extent of drug absorption; (2) gastric and intestinal motility; (3) extraction and metabolism occurring in the liver (first-pass effect); and (4) plasma protein binding.

Higher than usual plasma propranolol concentrations in patients with intestinal disease cannot be attributed to improved overall absorption of the drug, since only 0.7–4.6% of a ¹⁴C-labelled dose can be recovered in the faeces, and urinary recovery amounts to 90% or greater.¹ Together, these observations indicate that absorption of propranolol is virtually complete in patients with normal intestinal function.

An alternative explanation for high plasma propranolol concentrations seen in patients with coeliac disease is an increased rate of absorption. This would be difficult to prove without cannulation of the portal vein since mathematical analyses are distorted by the first-pass effect. Nevertheless, accelerated gastric emptying has been described in coeliac disease² and we have recently found that metoclopramide (10 mg intravenously) elevates plasma propranolol concentrations in the first 2 h after dosing (see table below).

The most important source of individual variability in plasma propranolol concentrations appears to be the extent of metabolism at the first-pass. The most accurate way of measuring this effect is to compare the areas under the concentration-time curves in the same subject after both oral and intravenous administration. This was not done in the studies reported by Parsons *et al*³ or in those reported by Dr R E Schneider and his colleagues (2 October, p 794). There are, however, several groups of patients in whom reduced first-pass metabolism and increased bioavailability have been reported, including the elderly⁴ and those with hepatic disease.⁵ Since hepatic may complicate intestinal disease it remains a possible explanation for the differences reported in coeliac and Crohn's disease. Finally, as propranolol is extensively bound to plasma proteins the volume of distribution and thus the plasma concentration may be increased when protein binding is diminished.

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¹ Paterson, J W, *et al*, *European Journal of Clinical Pharmacology*, 1970, 2, 127.

² Moberg, S, and Carlberger, G, *Scandinavian Journal of Gastroenterology*, 1974, 9, 17.

³ Parsons, R L, *et al*, *Gut*, 1976, 17, 139.

⁴ Castleden, C M, Kaye, C M, and Parsons, R L, *British Journal of Clinical Pharmacology*, 1975, 2, 303.

⁵ Branch, R A, James, J, and Read, A E, *British Journal of Clinical Pharmacology*, 1976, 3, 243.

General practitioner's role in management of labour

SIR,—Had Professor Peter Curzen and Dr Ursula M Mountrose (11 December, p 1433) advanced their valuable figures in support of a conclusion that delivery in a unit where specialised monitoring in labour and afterwards was necessary one could not but have agreed with them. But they do not. Instead, they conclude that there is no place for the general practitioner to care for his own

Mean (and range) of plasma propranolol concentrations (ng/ml) following an 80 mg oral dose in four fasting normal subjects

	Time after dosing			
	½h	1h	1½h	2h
After intravenous physiological saline ..	26 (0–59)	39 (0–88)	45 (0–88)	49 (16–83)
After intravenous metoclopramide 10 mg ..	65 (18–106)	60 (13–107)	64 (22–113)	65 (33–113)