by inclusion within the National Health Service. The Faculty fellows, members, and medical associates are all registered medical practitioners, who obtain a diploma of MFHOM or FFHOM as a postgraduate degree. Any other homoeopathic qualification in the UK is not a medical qualification, and anyone who advertises as a homoeopath is a lay practitioner.

We are aware that there is still a great deal of antipathy and opposition to homoeopathy in this country in certain medical quarters, but I would appeal to those who hold this view to support those of us who sincerely believe in the value of homoeopathy, used with judgment and skill together with a sound knowledge of clinical medicine and therapeutics. Failure to do so will not cause homoeopathy to wither away—it will only encourage more lay practice, to which patients will turn if there are not enough qualified doctors practising homoeopathy.

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24,25-Dihydroxycholecalciferol and calcium absorption in uraemia

SIR,—We were interested to read of the apparent lack of effect of 24,25-dihydroxy-vitamin D₃ (24,25(OH)₂D₃) on calcium absorption in uraemic patients reported by Drs J Szymendera and K Galus (25 November, p 1465), which contrasts with our own previously reported findings.¹

This contrast may reflect differences in the type of patient studied (our patients were anephric); in the dose and duration of treatment with 24,25(OH)₂D₃; or, as they suggest, in the method used to measure calcium absorption. Although the differences between tests for calcium absorption were clearly not critical in demonstrating effects of 1,25(OH)₂D₃ or 1 α -hydroxycholecalciferol, we have additional evidence that the effects of 24,25(OH)₂D₃ on calcium absorption differ from those of 1,25(OH)₂D₃. Using a low dose of carrier calcium (20 mg) for the ⁴⁵Ca tracer dose, we have noted little change in plasma ⁴⁵Ca curves in anephric patients given 24,25(OH)₂D₃, whereas large changes were seen after 1,25(OH)₂D₃ (Cochran *et al*, unpublished). From our inability to demonstrate effects of 24,25(OH)₂D₃ in this way, and the apparent increases in calcium absorption noted using the whole body counter,¹ we deduce that 24,25(OH)₂D₃ either increases intestinal calcium transport at distal sites in the gastrointestinal tract or affects its metabolism following absorption, but has little if any effect on duodenal calcium transport.

It seems possible that Drs Szymendera and Galus have additional information which might be helpful. In an earlier paper² Szymendera, using his double isotope technique, demonstrated that maximal rates of absorption occurred only 16 minutes after ingestion of the tracer dose. It seems reasonable to assume that the kinetics of calcium absorption at the time of its peak rate are particularly indicative of its absorption in the proximal gut. We would be interested to know if they have been able to calculate maximal rates of absorption from the data obtained in their series of uraemic patients,

and, if so, whether these rates changed as little as the fractional absorption in response to 24,25(OH)₂D₃. We have previously shown that the maximal rate of absorption changed more markedly in response to therapy than the fractional absorption,³ so the former variable should have the twin advantages of increased specificity and increased sensitivity in determining whether 24,25(OH)₂D₃ has any measurable effect on calcium absorption in the upper small intestine of uraemic patients.

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¹ Kanis, J A, *et al*, *British Medical Journal*, 1978, **1**, 1382.

² Szymendera, J, Heaney, R P, and Saville, P D, *Journal of Laboratory Clinical Medicine*, 1972, **79**, 570.

³ Reeve, J, Hesp, R, and Veall, N, *British Medical Journal*, 1974, **3**, 310.

* * * We sent a copy of this letter to the authors, whose reply is printed below.—Ed, *BMJ*.

SIR,—We appreciate Dr Kanis and others' comments. But we feel that the data presented in our report do not contrast with those they reported.¹ Our data simply provide evidence that 24,25-dihydroxycholecalciferol (24,25(OH)₂D₃) does not influence active calcium transport in the proximal jejunum. This is also their contention—they state in their letter that this agent has little if any effect on calcium absorption in the duodenum. It may be deduced from the apparent difference between the increased absorption shown by whole-body counter and the unchanged absorption shown by our double-isotope technique that 24,25(OH)₂D₃ increases intestinal calcium absorption at distal sites. But this contention needs straightforward evidence. The second possibility is that 24,25(OH)₂D₃ affects calcium accretion only; this effect might explain the increased retention of calcium tracer as well. The latter contention is in agreement with a recent report by Ornoy *et al*,² which presents evidence that 24,25(OH)₂D₃ is intimately associated with bone formation and mineralisation.

Concerning the relation of total and maximal absorption of calcium in the intestine, the data presented in the accompanying table show that maximal rates of absorption tended to increase with the increased total absorption, and vice versa. However, the changes were of about the same size, which is contrary to expectation. If the changes in the maximal rate of absorption were affected by treatment with 24,25(OH)₂D₃, one would expect that they would be many times greater than the increase in total absorption.³

In conclusion, we contend that differences in both the peak rate of absorption and the

percentage absorption represent natural variability and do not seem to be influenced by the treatment.

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¹ Kanis, J A, *et al*, *British Medical Journal*, 1978, **1**, 1382.

² Ornoy, A, *et al*, *Nature*, 1978, **276**, 517.

³ Reeve, J, Hesp, R, and Veall, N, *British Medical Journal*, 1974, **3**, 310.

1-alpha-hydroxy vitamin D₃ in primary hyperparathyroidism

SIR,—We have read with interest the paper from Dr D A Heath's group on the value of 1 α -hydroxy vitamin D₃ (1 α HCC) in the treatment of primary hyperparathyroidism before parathyroidectomy (17 February, p 450). While we agree on the need for careful monitoring of the serum calcium we would like to record our conclusion that the Birmingham experience may relate to patients with less severe bone problems than the group previously described by ourselves.¹

It is difficult to grade the severity of skeletal disease in primary hyperparathyroidism; but, in addition to the serum alkaline phosphatase concentration and the results of routine radiology, we believe that quantitative bone histology, serum 25-hydroxy vitamin D₃ concentration, radiocalcium absorption, ^{99m}Tc-diphosphonate retention, and even symptoms relating to musculoskeletal involvement, may be relevant indices, which also relate to the probability of severe postparathyroidectomy hypocalcaemia and related complications. Each of Dr Heath's bone disease groups of six patients contains at least one patient with a normal serum alkaline phosphatase concentration, and the highest concentration in the group not receiving 1 α HCC was only about three times normal; whereas six patients with primary hyperparathyroidism initially described by ourselves as having been treated by 1 α HCC had levels ranging from three to 12 times normal. In our experience the combination of severe osteitis fibrosa and a normal alkaline phosphatase concentration occurs only in the presence of marked magnesium deficiency. It is a pity that, with the exception of the serum calcium concentrations, the data for the Birmingham patients have been averaged as there must be real doubt about the true comparability of the two groups with bone disease.

It was not possible to judge the variation in the serum calcium from day to day before the administration of 1 α HCC in the Birmingham patients, but, from the experience of ourselves and others,² the fluctuations in five of the six patients after starting 1 α HCC could be accounted for by the spontaneous changes in the serum calcium which occur in this disease. The sixth patient, whose serum calcium concentration rose by 1.5 mmol/l (6 mg/100 ml) after starting on 2 μ g of 1 α HCC daily, is the first such report we have encountered—and we ourselves have experience of some 15 patients with primary hyperparathyroidism and bone disease managed in this fashion. It would have been of great value to have had further details of this particular case.

Calcium absorption in uraemic patients treated with 24,25-dihydroxycholecalciferol

Case No	Total absorption (% oral dose)		Maximal rate of absorption					
			Time (min)		% dose/min		μ mol Ca/min	
	Before	After	Before	After	Before	After	Before	After
1	8.25	8.24	22	22	0.0603	0.0726	3.00	3.50
2	23.07	24.28	13	16	0.2574	0.1681	12.75	8.25
3	12.34	7.55	36	86	0.1120	0.0410	5.50	2.00
4	5.72	14.34	155	74	0.0203	0.0573	1.00	2.75
5	14.06	3.80	46	175	0.1243	0.0142	6.25	0.75
6	9.76	28.19	23	15	0.0734	0.4990	3.75	24.50

Conversion: SI to traditional units—Calcium: 1 μ mol/min = 40 μ g/min.