

lesions arise as a result of sunlight on uraemic skin and are not necessarily a drug reaction.

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<sup>1</sup> Gilchrist, B, Rowe, J W, and Mihm, M C jun, *Annals of Internal Medicine*, 1975, 83, 480.  
<sup>2</sup> Korting, G W, *Dermatologica*, 1975, 150, 58.

### Myocardial bleeding in haemophilia

SIR,—We have recently studied 20 severe haemophilic boys at a residential school for evidence of myocardial bleeding. They were aged between 13 and 17, and all had factor VIII levels of less than 1%. So far as we could ascertain there was no clinical evidence of significant myocardial bleeding at any time in their life. We also studied their electrocardiograms for mean frontal axis, T wave changes, and conduction defects. We considered them all to be within normal limits.

It is recognised that muscle bleeding is the second most likely site of haemorrhage after joints in haemophilia.<sup>1</sup> We find it difficult to understand why this does not appear to occur in such an actively contracting muscle bulk as the heart. We could find no report of myocardial haemorrhage in the literature, although pericardial bleeding has been noticed as a post-mortem finding in dogs known to be haemophiliacs.<sup>2</sup> We would be interested to hear if experience in other centres is similar. If it is, why does myocardial haemorrhage not appear to be a clinical problem?

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<sup>1</sup> Favre-Gilly, J, *Hémostase*, 1964, 4, 231.  
<sup>2</sup> Webster, W P, et al, *Proceedings of Symposium on Haemophilia*, Sidney, Australia, ed I R Vanderfield, 1966.

### Hypophosphataemic osteomalacia in patients receiving haemodialysis

SIR,—In some haemodialysis centres osteomalacia is a major component of the progressive osteodystrophy that occurs in a large proportion of patients on regular treatment. There is an increasing awareness of the potential importance of phosphate depletion and hypophosphataemia as part of the aetiological mechanism for the osteomalacia in these patients. The recent paper by Dr J F Mahony and his colleagues (17 July, p 142) was therefore of interest in this respect. In three of their four patients, as in the patient reported by Baker *et al*,<sup>1</sup> the regular oral intake of a phosphate binder in the form of aluminium hydroxide was undoubtedly a major aetiological factor for the phosphate depletion hypophosphataemia and osteomalacia. We were, however, particularly interested in Dr Mahony's fourth patient (case 4), who had progressive osteomalacia and hypophosphataemia but was not taking aluminium hydroxide. We have studied<sup>2</sup> four haemodialysis patients who had persistent predialysis hypophosphataemia and developed osteomalacia but who had not taken oral phosphate binders for two years or more. The persistence of the hypophosphataemia in our patients was

apparently due to intestinal phosphate malabsorption accentuated by poor dietary intake and continual loss during dialysis.<sup>3</sup> Treatment of our patients with oral dihydrotachysterol and increasing the dietary intake of phosphate resulted in marked improvement in four months. Serum 25-hydroxycholecalciferol concentrations in our osteomalacic patients were either normal or increased, an observation which is of considerable interest.

The early detection of phosphate depletion in haemodialysis patients may be difficult, as plasma phosphate concentrations do not necessarily reflect phosphate balance, and in some phosphate-depleted patients plasma alkaline phosphatase activity and parathyroid hormone concentrations may be normal. The position would be considerably helped by identification of the optimal predialysis concentration. This might lead to the avoidance of the two extremes of tissue calcification on the one hand and phosphate depletion osteomalacia on the other.

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<sup>1</sup> Baker, L R I, et al, *British Medical Journal*, 1974, 3, 150.  
<sup>2</sup> Ahmed, K Y, et al, *Lancet*, in press.  
<sup>3</sup> Bishop, M C, et al, *Proceedings of the European Dialysis and Transplant Association*, 1971, 8, 106.

### Inaccessible specialist

SIR,—With some difficulty I recently persuaded a young professional to seek psychiatric help for panic attacks. Because the consultant I wanted him to see was full-time, he attended the outpatient clinic at his local teaching hospital. There was a 3-4-week delay.

On arrival he was confronted by a student to whom he told his story with some diffidence and diminishing confidence. He was next confronted by the registrar, who in spite of protests insisted on a full repeat of the story. He was spared a third run because he was denied access to the consultant with whom a specific appointment had been made.

Obviously students have to be taught, but I doubt if this is the best way to encourage patient acceptance of psychiatry. But, much more important, why is it so difficult to see a specific specialist in the Health Service, or has the Service decided, like comprehensive education, to deny all choice? If so, why do they bother with specialists' names in clinics?

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### Fetal transfusion syndrome

SIR,—Placental vascular anastomoses in mono-chorionic twins are not infrequent. However, they are usually fairly well balanced, so that neither twin suffers. The decision to perform an immediate blood transfusion in a severely anaemic "donor twin" or to bleed a "recipient twin" must be based on clinical judgment.

The following report seems to illustrate the above principle. In our unit a pair of mono-chorionic male twins were born via spontaneous vaginal delivery, each weighing 2.300 kg. Several hours later they did not

appear well. The first-born twin (twin A) was pale, tachycardic, and his haemoglobin was 11 g/dl and haematocrit 32%. The second-born twin (twin B) grunted and was plethoric and tachypneic. The edge of his liver was palpable 3 cm below the costal margin. His haemoglobin was 23 g/dl and haematocrit 69%. Their blood group was AB+.

We decided that both deserved treatment. We placed them one alongside the other, withdrew 35 ml blood through an umbilical vein from twin B, and transfused 30 ml of it through a peripheral vein to twin A. We used heparinised 10 ml syringes, and the procedure lasted about 15 minutes. Both infants improved almost immediately and seemed content. Their haemoglobins on the following day were 14 and 18 g/dl respectively.

To summarise, inequality of nature was corrected in an economic way and to the benefit of all parties involved.

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### Carpal tunnel syndrome and tennis elbow

SIR,—May we thank Drs T C Beer and N Memon for the suggestions in their letter (31 July, p 299). We note with interest the figures of symptomatic tennis elbow in their series of cases of the carpal tunnel syndrome. In our series of patients reviewed after surgery for the carpal tunnel syndrome eight patients (18.5%) had previously sought medical advice on account of tennis elbow, and in five of these there was still clear evidence of tennis elbow when we saw them. A further six patients reviewed by us in this study had evidence of tennis elbow, making up our overall figure 14 (33%).

In relation to the second suggestion that our patients with carpal tunnel syndrome and our controls may not have been matched in respect of occupation, the figures in the table would seem to suggest that our two groups were in fact very comparable in this respect:

	Carpal Tunnel (43)	Control (43)
	Women (39)	Women (39)
Full time housewife ..	9	7
Office/clerical work ..	13	13
Light manual ..	13	16
(tailoress) ..	(8)	(5)
Moderate/heavy manual ..	2	3
	Men (4)	Men (4)
Light manual ..	3	3
Heavy manual ..	1	—
Unemployed ..	—	1

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### Fear of ECT

SIR,—In the course of Professor W H Trethowan's irascible and contentious review of Dr Willis's new textbook of psychiatry (31 July, p 306) he makes several statements with which one might disagree. Most odd, however, is the passage in which, referring to the administration of ECT in pre-anaesthetic days, he describes patients who "would sometimes develop an inexplicable fear of it."