("an observation not previously made") Hillyer⁵ wrote in 1971: "the results suggest that auto-immune and/or localised *in vivo* antigen-antibody reactions may play a significant role in the pathogenesis of schistosomiasis. . . ." In 1930 Muller and Stender reported a similar case.⁶

EEG, EMG, and spinal cord evoked response studies of the reported case must be most relevant. It is regrettable that these studies were not mentioned in the article. The treatment given to this patient (niridazole 25 mg/kg twice daily for 14 days and then prednisolone 10 mg three times daily, reducing to 5 mg daily after one week) must be considered too little and too late. Why were hycanthone and higher doses of prednisolone not tried in this utmost neurological emergency? What could have been the relative or absolute contraindications?

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- ¹ Ross, G L, Norcross, J W, and Horray, G, *New England Journal of Medicine*, 1959, **246**, 823.
² Phillips, T M, and Draper, C C, *British Medical Journal*, 1975, **2**, 476.
³ Wakefield, G S, et al, *Brain*, 1962, **85**, 535.
⁴ Bird, A V, *Neurology*, 1964, **14**, 647.
⁵ Hillyer, G V, *Proceedings of the Society for Experimental Biology and Medicine*, 1971, **136**, 880.
⁶ Muller, H R, and Stender, A, *Archiv für Schiffs- und Tropenhygiene*, 1930, **34**, 527.

Professorial pulchritude

SIR,—Referring to the obituary tribute to the late Professor M F Lucas Keene (28 May, p 1422) in which this comely teacher is described as "the first woman professor of anatomy," I am prompted to draw attention to the following fact.

In Wilfred Blunt's book *Sebastiano* about the travels of a Bolognese priest to Paris and back in 1664-5 it is stated that "Bologna was the first university to institute a chair of anatomy, and the first perhaps to have a woman professor—Novella d'Andrea, in the fourteenth century. (Such, however, was that lady's beauty that she was obliged to deliver her lectures behind a curtain.)" Similar restrictions were not practised 600 years later when the histrionic Professor Lucas Keene gave her lectures, which, no doubt, were a conspicuous success.

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Megaloblastic anaemia associated with sulphasalazine treatment

SIR,—Drs R E Schneider and Linda Beeley (25 June, p 1638) report a case of anaemia with megaloblastic change in the marrow which, they feel, resulted from treatment with sulphasalazine. The use of this agent may be questioned in a patient in whom a diagnosis of ulcerative colitis was excluded by normality of the rectal mucosa. The symptoms reported may merely have been the result of an irritable bowel syndrome and haemorrhoids.

The microbiological serum folate level would be invalidated by concurrent antibacterial therapy,^{1,2} and only an isotope dilution technique would have been acceptable.² However, the serum vitamin B₁₂ level (presumably measured by the *Euglena gracilis* or isotope dilution method) would be resistant to antibacterial interference and was low compared with stated normal ranges. Since no details of gastric antibodies, peak acid output, or Schilling test are reported B₁₂ deficiency

cannot be excluded, and the response to high-dose folate therapy is not specific to folate depletion. It has been suggested that patients with B₁₂ deficiency may be especially prone to sulphonamide-induced blood dyscrasias.³

More information, including adequate documentation of dietary folate and some investigation of the small bowel, is required before sulphasalazine is blamed for the megaloblastic anaemia in this patient.

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- ¹ Herxheimer, A, *Journal of Antimicrobial Therapy*, 1975, **1**, 346.
² Bateson, M C, Hayes, J P L A, and Pendharkar, P, *Lancet*, 1976, **2**, 339.
³ Chanarin, I, and England, J M, *British Medical Journal*, 1972, **1**, 651.

Bites from house martin parasites

SIR,—Insect bites are common but often the causative insect is not identified.

In late April neighbours started to suffer a series of severe insect bites, with bullae and marked surrounding erythematous induration, principally around the ankles but also elsewhere on the limbs. The pruritus lasted for over a week despite treatment with local and systemic antihistamines. One of the insects was identified by Dr S F Barnett, of the Department of Clinical Veterinary Medicine, Cambridge, as *Oeciacus hirundinis*. These small brown parasites of swallows and martins are similar to bed bugs. There have been no recent published reports of this insect emerging from house martins' nests to bite humans.

It appears that the insects over-wintered in the remains of last year's nests and became active at the start of spring, just before the martins returned from Africa. Many of the insects entered the houses around the window frames and, usually at night, crawled over the ceiling, falling on to the bed and biting their human victims. Hiding from light by day they were not initially apparent.

The victims had not suffered similarly in previous springs, although the martins had nested under the eaves within inches of the windows for two years past. The succession of good summers and mild winters may have allowed the bugs to build up to large numbers, such that with the coming of the warmer weather they spread further afield in search of their usual prey and found humans instead.

This year attacks abated with the complete clearance of all traces of old nests and extensive spraying of the area with domestic fly spray, to which the bugs are susceptible. As an ornithologist may I ask that the nests are not knocked down till after the end of the breeding season?

N I MCNEIL

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Beckett on biofeedback

SIR,—Your informative leading article (11 June, p 1491) on biofeedback considers some recent publications on this concept, but perhaps the most extreme example of voluntary cardiac control was vividly described as long ago as 1938.¹

"Murphy had lately studied under a man in Cork called Neary. This man, at that time, could stop his heart more or less whenever he liked and keep

it stopped, within reasonable limits, for as long as he liked. This rare, faculty, acquired after years of application somewhere north of the Nerbudda, he exercised frugally, reserving it for situations irksome beyond endurance, as when he wanted a drink and could not get one, or fell among Gaels and could not escape, or felt the pangs of hopeless sexual inclination."

It is, perhaps, not surprising in a land in which the heart more often than not rules the head, that Murphy himself should have some cardiologically peculiarities, but Beckett's insight into cardiology—or, perhaps more correctly, into cardiologists—is fascinating; might not Neary have as easily created the words inotropy, chronotropy, or bathmotropy?

"Murphy's purpose in going to sit at Neary's feet was not to develop the Neary heart, which he thought would quickly prove fatal to a man of his temper, but simply to invest his own with a little of what Neary, at that time a Pythagorean, called the Apmonia. For Murphy had such an irrational heart that no physician could get to the root of it. Inspected, palpated, auscultated, percussed, radiographed and cardiographed, it was all that a heart should be. Buttoned up and left to perform, it was like Petrushka in his box. One moment in such labour that it seemed on the point of seizing, the next in such ebullition that it seemed on the point of bursting. It was the mediation between these extremes that Neary called Apmonia. When he got tired of calling it the Apmonia he called it the Isonomy. When he got sick of the sound of Isonomy he called it the Attunement. But he might call it what he liked, into Murphy's heart it would not enter. Neary could not blend the opposites in Murphy's heart."

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- ¹ Beckett, S, *Murphy*. London, Routledge, 1938.

Zinc, acrodermatitis, acne, and prostaglandins

SIR,—You have twice recently drawn attention to the importance of zinc in human metabolism.^{1,2} In zinc poisoning there are gastrointestinal upsets, fever, muscle pain, hypotension, cardiac arrhythmias, and renal and hepatic damage.¹ In acrodermatitis enteropathica there is a zinc deficiency.^{2,3} Acrodermatitis responds to treatment with either halogenated 8-hydroxyquinolines or zinc.³ In one patient zinc treatment brought the plasma level of the metal from 400 µg/l up into the normal range of 700-1150 µg/l.³ A patient with acrodermatitis and acne showed improvement in both conditions during zinc treatment⁴ and zinc has been shown to bring about improvement in other acne patients.⁵

In acrodermatitis enteropathica there is evidence of abnormal essential fatty acid metabolism and prostaglandin deficiency.⁶ Using a rat vascular preparation we have recently shown that zinc is a powerful stimulator of the production of a substance which has the characteristics of prostaglandin E₁. The threshold zinc concentration for the effect was 500 µg/l, which is very close to the threshold for a therapeutic effect in acrodermatitis. Excess stimulation of prostaglandin synthesis could account for a number of the features of zinc poisoning. It suggests that prostaglandin synthesis inhibitors, like aspirin and indomethacin, and prostaglandin antagonists might be of value in this situation.

We have not tested the halogenated 8-hydroxyquinolines, but we have shown that the related compounds chloroquine and quinine can act as either prostaglandin agonists