criticism it would be a shame to miss seeing the unique niche this drug has in the treatment of urinary infections by organisms previously only susceptible to intramuscular therapy. We were fortunate in having some of the earliest clinical trial material of carfecillin and were therefore able to witness a number of occasions when its use cured urinary infections which would previously have been treated with a parenteral antibiotic. In particular, the life of a middle-aged paraphegic was transformed by carfecillin. Previously he had been subject to repeated febrile episodes with malaise and sweating accompanied by the isolation of a *Pseudomonas aeruginosa* from the urine and treated by admission to hospital and intramuscular therapy. Though no antibiotic (including carfecillin) ever cleared his urinary tract of infection permanently, a course of carfecillin started at the onset of a febrile episode controlled it quickly, enabling him to remain at home. Eventually confidence in the therapy enabled him to take a holiday, his first since the onset of his illness two years previously.—I am, etc.,

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Skin Reactions to Beta-blockers

SIR,—I was interested to read the comments of Dr. J. B. Cumberbatch (30 November, p. 528) on an apparent reaction to oxprenolol.

For the sake of accuracy, however, I should make it clear that in my letter of 26 October (p. 229) I did not intend to imply that the reactions to practolol I noted were in fact "exacerbations of psoriasis," though they appeared at first sight to be so. As I pointed out in the earlier part of my letter, by virtue of its psoriasiform appearance a practolol rash may be missed when superimposed upon the psoriasis and mistakenly thought to be an exacerbation of the psoriasis.—I am, etc.,

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Medical Nemesis

SIR,—Congratulations on your thought-provoking leading article (7 December, p. 548) and the three well-written reviews under the same title about Ivan Illich's book[1] (7 December, p. 573).

I was reading them last night and half listening to Michael Parkinson's second interview with Muhammad Ali, which my wife had on the television. The vocal boxer was just commenting that he did not feel criticism came well about fighter's techniques from those who had never been in a boxing ring—and thinking back to my memories of schoolboy boxing three-round bouts I agreed with him. But also I felt that similar views were being expressed by your three reviewers in their own way about the criticisms of medical practice levied by Ivan Illich.

Like your reviewers I feel that the "insatiable and ill-informed" demand for much of modern medicine is from those outside the medical profession rather than from those practising it. It is too often the politicians and academic sociologists who "shout from the rooftops appropriately festooned with television aerials the benefits and breakthroughs of modern technology."—I am, etc.,

COLIN R. PORTEOUS

Ormskirk, Lancs

1 Illich, I., *Medical Nemesis*. London, Calder and Boyars, 1974.

Acute Calf Swelling

SIR,—We were interested to note the new physical sign of calf haematoma described by Dr. D. A. Tibbutt and Mr. A. J. Gunning (26 October, p. 204). In discussing the problem the authors do not mention another cause of acute calf swelling which may mimic deep venous thrombosis and if incorrectly treated can produce calf haematoma.

Acute synovial rupture of the knee joint with leakage of synovial fluid into the tissues of the calf can resemble the symptoms and signs of deep venous thrombosis very closely.[1] It occurs in patients with a previous history of knee arthritis and effusion when the joint is subjected to a sudden or prolonged increase in pressure such as on rising from a kneeling position.[2] Distinction from a deep venous thrombosis is usually possible if an arthrogram is performed shortly after the event. The patient should be exercised in the standing position and contrast medium will then be seen to pass down into the calf. Incorrect treatment of this condition with anticoagulants can produce a persistent calf haematoma.[3]

The condition is not uncommon in departments of rheumatology, and Jayson *et al.* have described 20 patients seen over a period of 18 months.[2] We have seen 10 patients with acute synovial rupture in one year in our department attached to a district general hospital. Like other authors[4] we feel the condition of acute synovial rupture of the knee joint producing acute calf swelling is underdiagnosed. It is not well documented in the general medical literature and merits consideration in a patient with a previous history of knee effusion who presents with acute calf swelling.—We are, etc.,

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1 Dixon, A. St. J., and Grant, C., *Lancet*, 1964, **1**, 742.
2 Jayson, M. I. V., *et al.*, *Annals of Physical Medicine*, 1969, **10**, 175.
3 Tait, G. B. W., Bach, F., and Dixon, A. St. J., *Annals of the Rheumatic Diseases*, 1965, **24**, 273.
4 Hughes, G. R., and Pridie. R. B., *Proceedings of the Royal Society of Medicine*, 1970, **63**, 587.

Lorazepam Poisoning

SIR,—Lorazepam (Ativan) is a new member of the benzodiazepine group of tranquillizers which is not yet widely prescribed. We record the case of a child with moderate overdosage of this drug which produced surprisingly severe effects.

A 6-year-old boy ingested some of his mother's individually foil-wrapped 1-mg lorazepam tablets and presented at hospital two hours later with drowsiness and ataxia. The maximum possible dosage ingested appeared to be 30 mg and was probably considerably less. Careful questioning of the mother excluded the concurrent ingestion of any other drug and positively identified the tablets taken as lorazepam. Gastric lavage was carried out and no tablets were recovered from the washings. The child was admitted for observation. On examination he was drowsy but responded maximally to minimal stimuli. His pulse and blood pressure were normal but he was demonstrably ataxic. No other abnormality was present. About four hours after the self-poisoning episode the child became manifestly hallucinated, reaching out at invisible objects, grasping them, examining them, and chattering incoherently. We were unable to establish the nature of these hallucinatory objects. This behaviour continued intermittently for nine hours and examination on several occasions confirmed the movements to be purposeful and related to the hallucinated state rather than to extra-pyramidal dysfunction. The central nervous system was otherwise normal on examination, apart from generalized hyper-reflexia, and the plantar reflexes remained flexor throughout. After 27 hours the child appeared bright and alert, there were no abnormal neurological signs, and he was discharged.

We have been unable to find any other recorded case of lorazepam overdosage in children. The fatal dose in an adult is thought to be around 1·85 g. Known side effects of overdosage are drowsiness and stupor. Hallucinations are a recognized complication of overdosage with diazepam, a more widely used member of the benzodiazepam group. On a body weight basis the fatal dose for a child of this age would be between 500 and 600 mg. It was therefore surprising to find severe toxic signs with marked hallucination in an otherwise healthy child at a fraction of that dosage. Clinicians should therefore be alert to the potential central nervous system toxicity of this drug in children with even mild to moderate degrees of overdosage.—We are, etc.,

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The Roseolar Reaction

SIR,—Your leading article on fourth, fifth, and sixth diseases (23 November, p. 429) offers no information about the aetiology of the last of these, exanthem subitum or roseola infantum.

In 1966[1] and 1969[2] I published my hypothesis that this is not an infection sui generis due to one particular pathogen but a slow febrile immunizing reaction against many different, mainly intestinal rather than respiratory, viruses. Perhaps for this reason roseolar rashes are commonest in summer and autumn. The fast febrile immunizing reaction or "one-spike" fever is, as its name implies, all over in 24-36 hours; the roseolar reaction takes three or four days from onset to rash. Family doctors are usually called not for the illness but for the rash, sometimes ascribed to teething or possibly rubella. The rash consists of small pink macules surrounded by a pale areola; this bird's eye effect becomes more obvious as the skin cools after the child is uncovered.

In family studies I have seen a 13-month-old baby develop a roseolar reaction while her sister aged 2½ years suffered only a "one-spike" fever; from each Coxsackie A6 virus was isolated. Two other children have each had two separate roseolar rashes, two months and 13 months apart respectively. A mother was ill with fever and meningism; Coxsackie B2 virus was isolated from her