infants in the home. With respect, we suggest that you read our paper again and then ask this question: Would I be prepared to expose an infant or a pregnant woman in my care to a drinking water lead level of 800 $\mu\text{g/l}$?

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¹ Beattie, A D, *et al*, *Lancet*, 1975, **1**, 589.

² Bacon, A P C, *et al*, *Lancet*, 1967, **1**, 264.

³ Beattie, A D, *et al*, *British Medical Journal*, 1972, **1**, 488.

⁴ Campbell, B C, *et al*, *British Medical Journal*, 1977, **1**, 482.

⁵ Parry, W H, *Lancet*, 1967, **2**, 1207.

Molar solutions and molar concentrations

SIR,—An article by Dr S E Williams (15 May 1976, p 1189) concerning the use of hydrochloric acid for the correction of severe metabolic alkalosis perpetuates a small but significant error originally contained within one of the references.¹ The author states that 150 ml of 1-M HCl in one litre of sterile water yields 300 mmol of H^+ and Cl^- per litre. By definition a 1-M solution of any preparation contains 1000 mmol of solute. Therefore 150 ml of the 1-M HCl solution will contain 150 mmol of H^+ and Cl^- . When the 150-ml aliquot is added to the sterile water the molarity of the final solution is 0.15 M, not 0.3 M as would be calculated using the 300 mmol figure.

Such miscalculations may be critical when one is attempting rapidly to correct severe metabolic alkalosis. Furthermore, while the author, as well as others,^{1,2} confirms the safety of dilute solutions of HCl (0.1–0.2 M), several reports^{2,3} indicate that HCl in 0.3 M concentrations or greater may precipitate acute haemolysis of red blood cells. Thus great care should be directed toward ordering and preparing intravenous hydrochloric acid solutions for reasons both of efficacy and of safety.

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¹ Abouna, G M, Adrete, J A, and Stangl, T E, *Surgery*, 1974, **75**, 139.

² Shaville, H G, and Parke, R, *Surgery*, 1975, **78**, 439.

³ Whelton, O, *Lancet*, 1974, **1**, 1055.

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

SIR,—I am indebted to Dr Cloyd for pointing out a possible source for confusion in my article concerning the use of intravenous hydrochloric acid for correcting severe metabolic alkalosis.

One hundred and fifty millilitres of 1-M HCl made up to one litre with sterile water will produce a solution containing 300 mmol of H^+ and Cl^- (that is, 150 mmol of each ion). In retrospect this may have been ambiguous and I am sorry if it has caused confusion.

In the patient described the calculated Cl^- deficit and H^+ deficit suggested that a total of 300 mmol of H^+ was required—that is, 2 l of solution as made up above.

I would again emphasise the dangers of using solutions of hydrochloric acid of higher

molarity than 0.15 M (0.15 mmol HCl/l) for intravenous use.

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Patients' awareness of melaena

SIR,—During the past two months I have seen three elderly patients who have had melaena while taking fenoprofen. One presented with abdominal pain, the second with shortness of breath, and the third with shortness of breath and palpitations; their haemoglobin concentrations were 13.1, 7, and 9 g/dl respectively. They had had black stools for between two and four days but none of them complained of this and had continued to take the drug. The bleeding was discovered only when the patients were specifically asked about their stools.

Patients often complain of two or three loose stools but it appears that few realise the significance of change in colour. I would suggest that all patients being given drugs which might cause gastrointestinal bleeding should be made aware of the significance of black stools and given advice regarding the action to take.

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Teaching leprosy to medical students

SIR,—Professor H Scarborough (5 February, p 379), in defending the teaching of leprosy in leprosaria such as Garkida, Nigeria, states that physiotherapy, occupational therapy, and surgery are therapeutic measures. Instead, patients receiving such measures should be seen as examples of a tragic failure to prevent irreversible nerve damage, usually as a result of late diagnosis. Thus students taught in leprosaria may well conclude that deformity in leprosy is inevitable and that its management is time-consuming, needs expensive equipment, and is not very rewarding. If students are taught that the diagnosis of leprosy is difficult or that it is desirable to do skin smears to establish a definitive diagnosis they may lose interest in the subject, and it is not surprising that leprosy continues to be neglected by the medical profession.

In 1970 the Faculty of Medicine at the University of Dar-es-Salaam, Tanzania, carried out a teaching and health survey for two weeks on Mafia Island. The students were to detect cases of leprosy and tuberculosis, vaccinate the population of 14 000 against smallpox, and immunise the children against poliomyelitis and measles. There were no registered cases of leprosy on the island. None of the students had previously seen a case of leprosy. I gave one illustrated lecture followed by a clinical demonstration. The current classification of the disease was not mentioned. The students were instructed to screen the population for hypopigmented skin lesions, nodulation, and infiltration of the face and ears. Those having these signs were then referred on a later day to the WHO leprologist and the leprosy specialist for Tanzania, who made a definitive diagnosis. Seventy-one newly diagnosed cases of leprosy were detected, an estimated 80% of the leprosy on the island, and many were at an early, curable stage.

Thus the students learnt that the diagnosis of leprosy is easy and deformity can be prevented.

The capital and running costs of leprosaria like Garkida are enormous and this approach to leprosy is now completely outmoded. What is required is low-cost, effective, and practical management of the disease, and Ross was one of the few leprologists in the world to provide such measures. The decline in leprosy after 10–15 years of dapsone administration as shown by the figures from northern Zaria is a good example of successful preventive medicine. It is therefore a strange decision by the Faculty of Medicine of Ahmadu Bello University, Zaria, not to demonstrate these findings to the students, especially as the clinics are in the immediate vicinity of the medical school.

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CMV retinitis

SIR,—We are very grateful to Dr H W Murray for his letter (30 October, 1976, p 1071) drawing attention to the various reported episodes of the ocular manifestations of cytomegalovirus (CMV) retinitis and we look forward to reading his article when it is published. We apologise if we failed to make sufficiently clear that the patient we reported (31 July 1976, p 281) had a retinitis without visible choroidal involvement. Pure cytomegalovirus (CMV) retinitis was not the lesion in five of the 19 patients that Dr Murray cites^{1–5} and the diagnosis of cytomegalovirus inclusion disease was no more than presumptive in three patients.^{1,3,5} In particular we agree with Dr M E Smith⁶ that the diagnosis in the otherwise asymptomatic patient with a unilateral uveitis briefly described by Dr Helen Foerster¹ must remain unproved and speculative. Indeed, Dr Foerster herself presented her patient as a diagnostic problem.

It may well be that our patient is not the first reported case of CMV retinitis in an otherwise healthy adult but we remain unable to find a similar patient in the English literature.

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¹ Foerster, H W, *Survey in Ophthalmology*, 1959, **4**, 296.

² Maler, G H, and Disker, W A, *American Journal of Surgery*, 1970, **119**, 334.

³ Madge, G E, *Medical College of Virginia Quarterly*, 1972, **8**, 251.

⁴ Porter, R, *et al*, *British Medical Journal*, 1972, **3**, 133.

⁵ Coulson, A S, *et al*, *Western Journal of Medicine*, 1974, **120**, 1.

⁶ Smith, M E, *Archives of Ophthalmology*, 1964, **72**, 44.

Hip fractures up to date

SIR,—Congratulations on an excellent series of orthopaedic articles over the past few weeks, among which I particularly enjoyed Mr P A Ring's on hip fractures (11 December, p 1429). I should, however, like to endorse Mr E H J Smyth's comments (5 February, p 377) regarding the displaced subcapital fractures which he describes as "the problem-