objectivity in patients' management, so that the doctor might unwittingly be persuaded against a course of action which is ultimately in the patient's own good. In a practice where it is taken for granted that not only may patients choose a doctor but also the doctor frequently may decide to confer with his colleagues in the presence of the patient, this danger is considerably diminished.

So long as all partners are present in one central surgery at most, if not all, consulting times, so long as clinical notes are made concisely and clearly, and so long as regular (preferably daily) formal conversations take place between the partners in addition to in-surgery discussions, then we believe that encouraging the patients to consult any of the doctors can only enhance the standard of medical care in general practice.—We are, etc.,

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Discontinuation of Evening Surgery

SIR,—Many of Dr. T. Ternent's patients (1 April, p. 51) must now be attending his surgery during working hours. Since one's doctor is in one's home area (the dentist need not be), the commuters will also be taking time out for travelling. Is this trend to be encouraged? It is also likely to have serious effects for the patient with a long illness who is trying hard to hold down a job, and can at present obtain any necessary medical supervision in his own time.—I am, etc.,

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Contaminated Drip Fluids

SIR,—Early in January 1972 we investigated possible sources of contamination during the manufacture of sterile fluids, since some bottles of Darrow's solution (not for intravenous administration) were found to be contaminated. We also had a personal communication from Dr. Ian Phillips concerning similar difficulties, which he has since reported (18 March, p. 746).

The autoclave used for the production of sterile fluids was of a type designed originally for the process of canning, which involves sterilization under pressure and subsequent cooling of the hermetically sealed containers by water. We suspected that this process might be unsuitable for the type of screw-cap bottles now in general use, and that when these bottles are cooled in a spray-cooled autoclave, if there is the slightest imperfection in the seal, they may draw in some cooling water. (The cooling water is deionized, but not sterilized.) We tested this hypothesis by autoclaving some bottles inverted in a tray of red dye and also incorporating fluorescein in the cooling water in another cycle. In both experiments dye entered some bottles of each batch.

The thread of the bottles and the metal caps used for sterile fluids at present is relatively coarse. The type of closure which relies upon a rubber liner is altogether unsuitable as the liner cannot be relied upon to form a hermetic seal with the top of the bottle. Even when the cap is firmly screwed on some fluid may enter an apparently sealed glass bottle. For this reason we no

longer use spray-cooling for this type of bottle although leaving out this step prolongs the sterilizing cycle. It is clear that this type of autoclave although conforming to the specification for steam sterilizers for bottled fluids (British Standard, 3970, Part 2, 1966) is entirely incompatible with the type of bottle at present widely used.

So far we have not been able to demonstrate the contamination by cooling water of solutions autoclaved in M.R.C. bottles which are closed by means of a rubber plug and metal screw-cap.

We should also like to re-emphasize the importance of the inspection of bottles before use.¹ We have recently found visible fungal hyphae growing in commercially prepared dextrose solutions. The apparently intact bottles, on close inspection, were found to have fine cracks running through areas of mould growing in the gum on the back of the labels. The cracks were presumably due to rough handling in transit. An episode of this type has been reported and discussed by Robertson.²—We are, etc.,

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¹ Guy, J., and Jenkins, E. W., *British Medical Journal*, 1966, 2, 523.

² Robertson, M. H., *Journal of Medical Microbiology*, 1970, 3, 99.

Acute Salicylate Poisoning

SIR,—Your leading article (29 January, p. 263) on acute salicylate poisoning does not sufficiently emphasize the importance of alkalizing the urine, particularly to the pH of 7.6-8.0 by acetazolamide or acetazolamide and sodium bicarbonate. Morgan and Polak,¹ by the latter regimen, reduced salicylate half life in eight poisoning cases (from the normal of about 20 hours) to 6.3 hours, a considerable achievement. No toxicity or biochemical problems were encountered.

Your article states that "the use of acetazolamide in aspirin poisoning has shown that it may worsen metabolic acidemia." But of the three articles cited the first² does not mention acetazolamide (or any carbonic anhydrase inhibitor) at all and the second³ and third⁴ state explicitly that metabolic acidosis was not increased by the combination of acetazolamide and either lactate or bicarbonate. There are in fact no data to show that even acetazolamide alone could conspicuously worsen metabolic acidosis over the relatively short time of treatment.

The data in the literature suggest strongly that in both adults¹ and children^{3,4} aspirin poisoning can generally be handled safely and well by acetazolamide and sodium bicarbonate, in addition to the supportive treatment outlined in your article.—I am, etc.,

THOMAS H. MAREN

Department of Pharmacology and Therapeutics,
University of Florida,
U.S.A.

¹ Morgan, A. G., and Polak, A., *British Medical Journal*, 1969, 1, 16.

² Hoffman, W. S., and Nobe, C., *Journal of Laboratory and Clinical Medicine*, 1950, 35, 237.

³ Schwartz, R., Fellers, F. X., Knapp, J., and Yaffe, S., *Pediatrics*, 1959, 23, 1103.

⁴ Feuerstein, R. C., Finberg, L., and Fleishman, E., *Pediatrics*, 1960, 25, 215.

. The two major regimens of treatment discussed in the leading article were forced diuresis with bicarbonate and the acetazolamide and bicarbonate regimen described by Morgan and Polak.¹ These would not have been stressed if the promotion of an alkaline polyuria had not been considered extremely important.

If when Professor Maren refers to "salicylate half line" he means the mean time for the plasma salicylate level to fall to half the peak level, and the various treatments for acute salicylate poisoning are judged on this basis alone, then the following results are obtained.¹⁻³

Regimen	Mean Time (Hours) to Reduce Plasma Salicylate Level to Half Peak Level
Forced oral	22.0
Forced saline/laevulose	10.0
Forced alkaline	5.0
Forced "cocktail"	6.7
Acetazolamide and bicarbonate	6.3

On this basis forced alkaline diuresis is the best, but the differences between the three most effective regimens mentioned are not of practical importance. The final choice of alkalizing regimen must be made with consideration of complications of acid-base and electrolyte imbalance resulting from treatment other than the rate of fall of plasma salicylate level. Despite Professor Maren's comments to the contrary rapid biochemical and acid-base changes do occur with acetazolamide and bicarbonate regimens, and at the least close biochemical monitoring is required, which is not necessary with forced "cocktail" diuresis.³

Schwartz *et al.*⁴ described a clinical study of three children treated with acetazolamide. The therapy was successful from the point of view of salicylate recovery but there were very severe biochemical changes encountered and large quantities of intravenous bicarbonate had to be administered to control the systemic acidosis which developed in the patients. As evidence of the severity of the changes two of the children had convulsions. Feuerstein *et al.*⁵ gave sodium lactate infusion before and during administration of acetazolamide, and so any conclusions which they made about acetazolamide and metabolic acidosis must be treated with reserve. Other authors raised serious objections to the clinical use of acetazolamide in salicylate poisoning. Smith⁶ suggested there may be an adverse synergistic action between salicylate and acetazolamide and concluded that in view of available information there was doubt about the clinical use of acetazolamide particularly in severe salicylate poisoning.—Ed., B.M.J.

¹ Morgan, A. G., and Polak, A., *British Medical Journal*, 1969, 1, 16.

² Lawson, A. A. H., M.D. Thesis *Management of Acute Salicylate Poisoning*, 1968, Edinburgh, p. 266.

³ Lawson, A. A. H., *et al.*, *Quarterly Journal of Medicine*, 1969, 38, 31.

⁴ Schwartz, R., Fellers, F. X., Knapp, T., and Yaffe, S., *Pediatrics*, 1960, 25, 215.

⁵ Feuerstein, R. C., Finberg, L., and Fleishman, E., *Pediatrics*, 1960, 25, 215.

⁶ Smith, M. J. H., in *The Salicylates*, p. 282, London, Interscience, 1966.

Sickle-cell and Altitude

SIR,—The lengthy letter of Dr. R. L. Green and others (25 March, p. 803) failed to tell your readers how the categorical statement

in their original article (4 December 1971, p. 593): "Haemoglobin studies later showed that she was a sickle-cell trait carrier" could now be reconciled with the statement in their letter that "the available haematological investigations have not excluded a diagnosis of sickle-cell disease." Your readers, especially those from Africa, would want to know how a Ghanaian nurse said to have needed laparotomy for intestinal infarction after the brief 45-minute flight from Kumasi to Accra was said by the authors to have the "sickle-cell trait" in one article only to be told in their next communication that the diagnosis of sickle-cell disease was not excluded. Their original article with the sickle-cell trait intestinal infarction story was promptly quoted not only in *The Times*¹ but also in *Nature*,² and in the case of the former the conclusion of its Science Correspondent was such that Dr. Green and his colleagues found it necessary to write to dissociate themselves from it.³

The bulk of the letter of Dr. Green and his colleagues was taken up with "fresh" evidence to prove that the true sickle-cell trait (with more HbA than S) could lead to splenic infarcts in unpressurized aircraft. But the burden of my earlier criticism was that in their article they produced evidence which did not stand the test of scientific scrutiny, the most glaring example of which is the Ghanaian nurse in whom, on the authors' own present admission, sickle-cell disease was not excluded. Your readers, Sir, are interested in scientific truth. I am one of them, and the very least I am entitled to expect is that if evidence obtained from my own country cannot be scientific it should at least be true.—I am, etc.,

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¹ *The Times*, 9 December 1971.

² *Nature*, 1971, 234, 326.

³ Green, R. L., Huntsman, R. G., and Serjeant, G. R., *The Times*, 8 February, 1972.

Smoking and Vascular Disease

SIR,—Your leading article (1 April, p. 3) states unequivocally, "The medical profession is the only professional group in which cigarette smoking has lessened," without the support of any references. I would doubt if each and every professional group has been adequately sampled and reported on. *Smoking and Health Now*¹ reports that after the publication of the 1962 report there was a sharp reduction in the number of men smoking cigarettes, a reduction maintained in social classes I, II, and III. The professional groups fall in these classes.

Your article goes on to point out that Pozner and Billimoria² (in a small selected sample) found that "fasting plasma turbidity, cholesterol, and β and pre- β lipoproteins were significantly increased in heavy smokers," but you failed to make the most important point that in male heavy smokers only pre- β lipoprotein means were significantly higher than those of non-smokers. In a sample of 2,483 middle-aged males Howell³ failed to find significant correlation between smoking and haemoglobin, E.S.R., serum cholesterol, β lipoprotein, and uric acid levels. The one striking finding—namely, a significantly increased white blood cell count in heavy smokers—has now been

confirmed by Corre, Lellouch, and Schwartz.⁴ In some small way this may provide confirmation that the sample used by Howell was not atypical for males of that age group with an average smoking history extending over 20 years.—I am, etc.,

R. W. HOWELL

Tilehurst, Berks

¹ Royal College of Physicians, *Smoking and Health Now*. London, Pitman, 1971.

² Pozner, H., and Billimoria, J. D., *Lancet*, 1970, 1, 1318.

³ Howell, R. W., *Lancet*, 1970, 2, 152.

⁴ Corre, F., Lellouch, J., and Schwartz, D., *Lancet*, 1971, 2, 632.

SIR,—I would agree with your leading article (1 April, p. 3) that the case against cigarette smoking in peripheral vascular disease is a very strong one. I am sorry that you take exception to my finding¹ fewer than 1% of male non-smokers among those seen with lower limb ischaemia. The figure that you quote, of less than 5%, is not strikingly different from mine. Perhaps the disparity may be due to my criterion of a non-smoker's status in this respect: these people were lifelong non-smokers and not ex-smokers. It has been interesting to review the final diagnosis in some of these cases. Entrapment of the popliteal artery, cystic adventitial disease, attrition over an exostosis, as well as other non-vascular causes have been suspected and mostly confirmed on the leading evidence of the non-smoker's status.

There is most certainly the need for more careful review of the relationship between cigarette smoking and occlusive arterial disease in the lower limbs. References are few, indeed you have given most of them.—I am, etc.,

H. H. G. EASTCOTT

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¹ Eastcott, H. H. G., *Arterial Surgery*. London, Pitman, 1969.

Recurrent Urinary Infections in a Girl

SIR,—I read with interest the warm flow of correspondence between Drs. Andrew Smith and Hugh Jackson (12 February, p. 428), and the latter's observation that the chief shortcoming to the accurate diagnosis of urinary tract infection, in the early age-group cited, was the practical difficulty of proving conclusively that bacteria were multiplying within the urinary tract itself. In sympathy with the above writers, to be sure, are the experiences of many in the past that false positive cultures on voided urines from neonates, infants, and children may exceed 60%, even when collections are achieved by the bag or mid-stream techniques done under trained supervision.¹ No mention, however, of the established technique of obtaining urine by suprapubic bladder aspiration was proffered by Dr. Jackson or Dr. Smith; nor did there once appear in their stream of thinking a single drop of support for this widely used, simple, safe, efficient, and economical procedure.¹⁻⁵ Hopefully, this exclusion will not retard the rapidly growing appreciation for suprapubic aspiration, which eliminates both the necessity for frequent voided urine collections and also quantitation of the bacterial content of these specimens, with the delays attendant in making a definitive diagnosis. Their omission was all the more astonishing, since

suprapubic aspiration relieves general practitioners of the urgency for specimen culture, the aspirated specimens can be safely posted to the laboratory, and the chief problem noted by Dr. Smith, namely that of contamination, has been eliminated.^{2,3}

To direct the question of "proven bacteriuria" back into mainstream thinking, it is my view that the first such episode in neonates, infants, or children should lead to both an intravenous pyelogram and a micturating cystourethrogram being performed, precisely because up to 50% of such patients with bacteriuria, symptomatic or not, have been shown to have vesico-ureteric reflux.⁵⁻⁷ In his letters, Dr. Jackson made insufficient mention of the degree of reflux present in his patient, but presumably because the pelvic-ureteric systems had become distended with contrast medium the reflux could be termed "gross" by the criteria of Rolleston *et al.*⁶—the only degree of reflux apparently associated with progressive renal damage (occasionally regardless of whether there is coexisting infection).

Dr. Smith's experience with ampicillin provides additional minimal evidence for condemning the use of this drug for the treatment of urinary tract infections, prior to availability of sensitivities of the given pathogens. The place of ampicillin as the drug of general convenience should be seriously questioned, as was strongly suggested by the large multicentre study in the United Kingdom where the sensitivities of over 23,000 different urinary tract pathogens were evaluated: in both inpatient (particularly) and outpatient isolates ampicillin dribbled in a mediocre fourth behind the "big three"—nitrofurantoin, trimethoprim-sulphamethoxazole combination, and nalidixic acid.⁸

Finally, nitrofurantoin is undoubtedly a useful agent in the prevention of recurrent urinary tract infections, but in a 2-year-old girl a dose of 25 mg t.d.s. would seem excessive, particularly when it has been shown by Professor de Wardener's group⁹ that a single nightly 50 mg dose was very effective in preventing recurrent urinary tract infections in adult women. The dose of drug used by Dr. Smith and Dr. Jackson could reach toxic levels in the serum if the child had coexisting renal functional impairment (which the urea estimation of 50 mg/100 ml suggests); moreover, if the child's glomerular filtration rate is less than about 60 ml/min/1.73 m², the drug may not be attaining adequate antibacterial concentrations in the urine.¹⁰—I am, etc.,

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¹ Shannon, F. T., Sepp, E., and Rose, G. R., *Australian Paediatric Journal*, 1969, 5, 97.

² Bailey, R. R., and Little, P. J., *British Medical Journal*, 1969, 1, 293.

³ Bailey, R. R., and Little, P. J., in *Renal Infection and Renal Scarring*, ed. P. Kincaid-Smith, and K. F. Fairley, p. 81. Melbourne, Mercedes Publishing Services, 1971.

⁴ Abbott, G. D., and Shannon, F. T., *Clinical Pediatrics*, 1970, 9, 277.

⁵ Abbott, G. D., *British Medical Journal*, 1972, 1, 267.

⁶ Rolleston, G. L., Shannon, F. T., and Utley, W. L. F., *British Medical Journal*, 1970, 1, 460.

⁷ Kunin, C. M., in *Renal Infection and Renal Scarring*, ed. P. Kincaid-Smith, and K. F. Fairley, p. 287. Melbourne, Mercedes Publishing Services, 1971.

⁸ McAllister, T. A., *et al.*, *Postgraduate Medical Journal*, Sept. suppl., 1971, 47, 7.

⁹ Bailey, R. R., Roberts, A. P., Gower, P. E., and de Wardener, H. E., *Lancet*, 1971, 2, 1112.

¹⁰ Sachs, J., Geer, T., Noell, P., and Kunin, C. M., *New England Journal of Medicine*, 1968, 278, 1032.