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 alkalinizing 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 acidaemia." But of the three articles cited the first[2] does not mention acetazolamide (or any carbonic anhydrase inhibitor) at all and the second[3] and third[4] 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 adults[1] 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.,

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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 azetazolamide. 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. Smith[6] 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