treatment and the child died five weeks after his admission. Post-mortem examination showed no evidence of functioning marrow.

Although aplastic anaemia can arise spontaneously, it was felt that chloramphenicol was the causal agent in this case.—We are, etc.,

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1 Ioannidis, A. H., and Murdoch, J. McC., *Brit. med. J.*, 1957, **1**, 1157.
2 Dameshek, W., *J. Amer. med. Ass.*, 1960, **174**, 1853.

Why Drink Milk?

SIR,—I think it is agreed by all that in the vast majority of cases mother's milk is the natural and adequate food for the infant mammal. But the articles by Drs. K. B. Taylor and S. C. Truelove (October 7, p. 924) and Drs. E. D. Acheson and S. C. Truelove (p. 929), and the letter from Dr. B. McNicholl (November 4, p. 1219) confirm my belief that after the age of weaning further consumption of milk (especially a "foreign" one—e.g., cow's milk) is neither natural nor necessary—and there are other and many milk-borne diseases besides ulcerative colitis.

Is it not time the British Medical Association should state unequivocally whether or not milk is a desirable article of diet? There is no particular virtue or value in milk that cannot be replaced by "safer" foods. Again I ask, why drink milk?—Baby Food!—I am, etc.,

Dundee. F. R. BROWN.

Bathing in a Plaster

SIR,—I have found recently great benefit from the use of polythene plastic bags placed over a limb encased in plaster-of-Paris or adhesive strapping when a patient wishes to take a bath. It is, generally speaking, very difficult to have a bath with a below-knee plaster propped on the edge, and the result of such efforts is almost always some degree of wetting and softening of the cast. If, however, a polythene bag is put over the limb and secured with a couple of elastic bands, the patient can bath with relative safety. Often, being able to have a bath is the one item that determines whether a patient will accept a plaster cast.—I am, etc.,

Winchcombe, Glos. JOHN FINCH.

Hypertension of Pregnancy

SIR,—In the annotation on hypertension of pregnancy (October 14, p. 1007) you discussed hysterotonin as an aetiological factor in its production. Your evaluation, however, failed to include many pertinent criticisms to which I wish to draw attention. When substances are seriously considered as responsible for sustained hypertension certain criteria must essentially be satisfied, since there are over a score of polypeptides with hypertensive properties that have been rejected as agents in the production of hyperpiesia in the human. Importantly, they need identification by their chemical structure; then their amounts in circulation must be considered sufficient to maintain a raised blood-pressure; and again specific substances need to be found that antagonize their hypertensive effects; whilst finally intra-arterial (*not* intravenous) injection must be employed in demonstrating their activity.

None of these criteria have been satisfied. The authors claiming this specificity[1 2] had failed to recognize that both renin and vaso-excitor motor substance have already been found to be present in pre-eclampsia. The properties of the octapeptide of angiotensin II entirely correspond to those enumerated as peculiar to hysterotonin, for not only does it elevate the blood-pressure but it produces uterine contractions and is not destroyed by adrenolytic agents. The sole point of difference—quite reasonably accounted for by the impurity of hysterotonin—is the fact that the angiotensin is dialysable whereas the hysterotonin is not.

In their claim for the aetiological significance of hysterotonin they set out the underlying influence of an increased uterine tension, which, by effecting myometrial ischaemia, causes an insufficient oxygenation of the decidua that is responsible for the product. It is already well recognized that the utero-renal reflex depends on the resistance of the uterus to stretch for its foundation—a common ground thereby shared. That the placenta or decidua cannot have a primary role in the production of pre-eclampsia is entirely borne out by the fact that pre-eclampsia is recovered from on the death of the retained foetus only to recur days later at the onset of labour. Reflection will show that such a happening denies any relevance of the primary intrusion of placenta or decidua in the toxaemic aetiology, but underlines the undeniable significance of the utero-renal reflex that in all the circumstances can alone explain this recurrence.—I am, etc.,

Worthing, Sussex. J. SOPHIAN.

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1 Hunter, C. A., and Howard, W. F., *Amer. J. Obstet. Gynec.*, 1960, **79**, 838.
2 —— ibid., 1961, **81**, 441.

A Case for Diagnosis

SIR,—The diagnosis of porocephalosis proposed for the case presented (September 23, p. 820) cannot be rejected so easily (October 14, p. 1027). (1) The patient didn't eat any snake during his trip to Africa. The eggs of *Armillifer armillatus* can be absorbed with snakes, but also with water soiled by snakes. So this infestation is very common in Africa: Vaucel[1] states that 5–25% of Africans in certain regions have porocephalosis. (2) The typical horseshoe calcifications, well described by Drs. Zaida M. Hall and R. R. Wilson (September 23, p. 280), appear many years after infestation, as Pellegrino[2] stated in a recent paper. The period of ten years between an African trip and appearance of calcifications is not unusual.[3 4] (3) The biopsies photographed by Drs. Hall and Wilson in the paper showed only connective tissue changes, and no calcification. Therefore it seems reasonable to think that the radiological, horseshoe-like object was missed by the biopsies.—I am, etc.,

Swiss Tropical Institute, Basle. MICHEL FERNEX.

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1 Vaucel, M., *Médecine Tropicale*, 1955. Flammarion, Paris.
2 Pellegrino, A., and Cave, L., *Méd. Afr. noire*, No. Spécial, August, 1960, p. 128.
3 Woithelet, G., *Méd. trop.*, 1956, **16**, 379.
4 Van Wymeersch, H., and Wanson, M., *J. Radiol. Électrol.*, 1955, **36**, 22.

"A Case of Renal Failure"

SIR,—The Clinicopathological Conference (November 4, p. 1208) was most interesting indeed. The most puzzling aspect of the case was a very rapid deterioration with failure of response to several attempts at dialysis. Renal-vein thrombosis was mainly blamed for this dramatic downhill course.