and was a consequence of the bacteraemia and, hence, of the primary infection. The fact that meningitis, once established, did not respond to therapy with cephalothin is a reflection of the low and erratic penetration of the antibiotic into the cerebrospinal fluid. There is absolutely no evidence to suggest that the meningeal infection was truly a "fresh infection" or, indeed, that it was "antibiotic induced."

The second misconception also occurs in the first paragraph. The comment is made that "apparently cephalothin is the only antibiotic against which this accusation can be levelled." This view is not shared by Mangi *et al.*,¹ who state that "meningitis is a possible complication when septicaemia is treated with any antibiotic that does not easily pass the blood brain barrier. A high degree of suspicion of meningitis is warranted for any patient with bacteraemia who does not respond to cephalothin or any other antibiotic with moderate cerebrospinal fluid penetration." Even a superficial review of the literature shows that meningitis developing during appropriate antimicrobial therapy has been reported for other antibiotics—for example, tetracycline,² penicillin,³ lincomycin,⁴ colistin.⁵ In fact, a series of 60 children had received such antibiotics as penicillin, tetracycline, ampicillin, erythromycin, and sulphonamide for respiratory tract infections, etc., prior to the development of meningitis.⁶ There is, therefore, nothing unusual or unique to cephalothin in this complication. Furthermore, the leader writer chose to ignore Mangi and colleagues' comments regarding such contributing factors as trauma due to lumbar puncture as a mechanism introducing bacteria into the spinal fluid in three patients; debilitating disease with probable diminishing host resistance in four; and rather low cephalothin dosage in two.

The conclusion in the leading article—namely, "what is in question here is not the treatment of meningitis but that of infections located elsewhere which may apparently be followed by meningitis if cephalothin is used in their treatment," requires clarification. Since cephalothin was introduced many millions of patients have been treated with it in many countries of the world. During that time the concomitant occurrence of meningitis has been mentioned in four reports involving a total of eight patients. It would be quite illogical to restrict the use of cephalothin for this reason.

Finally, the article refers to two reports^{10 11} from Paris and Rouen respectively, in each of which three patients are described who developed renal failure during treatment with cephalothin and gentamicin. As gentamicin is known to have nephrotoxic potential¹² it is difficult to determine what role, if any, cephalothin played in this matter.—I am, etc.,

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¹ Mangi, R. J., Kundargi, R. S., Quintiliani, R., and Andriole, V. T., *Annals of Internal Medicine*, 1973, 78, 347.

² Turck, M., Anderson, K. N., Smith, R. H., Wallace, J. F., and Petersdorf, R. G., *Annals of Internal Medicine*, 1965, 63, 199.

³ Almond, H. R., *New England Journal of Medicine*, 1969, 281, 218.

⁴ Lerner, P. I., *American Journal of Medical Sciences*, 1969, 257, 125.

⁵ Apicella, M. A., Perkins, R. L., and Saslaw, S., *American Journal of Medical Sciences*, 1966, 251, 266.

⁶ Torphy, D. E., and Ray, C. G., *American Journal of Diseases of Children*, 1970, 119, 336.

⁷ Hansman, D., and Gibbs, M., *New England Journal of Medicine*, 1972, 287, 201.

⁸ Price, D. J. E., and Sleight, J. D., *Lancet*, 1970, 2, 1213.

⁹ Jarvis, C. W., and Saxena, K. M., *Clinica Pediatrica*, 1972, 11, 201.

¹⁰ Kleinknecht, D., Ganeval, D., and Droz, D., *Lancet*, 1973, 1, 1129.

¹¹ Fillastre, J. P., *et al.*, *British Medical Journal*, 1973, 2, 396.

¹² Kahn, T., and Stein, R. M., *Lancet*, 1972, 1, 498.

* * Few readers are likely to accuse us of the first of these "misconceptions." We wrote that four of these five patients had positive blood cultures, and that the same organism caused the subsequent meningitis. This infection was clearly "fresh" only in the sense of its new location. As regards the second, there are naturally many instances in which meningitis has developed during treatment with other antibiotics, but if the cases described by Mangi and his colleagues were not at least unusual, if not indeed unique, why should they have been considered worthy of publication in a leading American journal? Their essential feature is the very short interval lapsing between the start of treatment and the onset of meningitis.—Ed., *B.M.J.*

Hypomagnesaemia after Parathyroidectomy

SIR,—Having had a similar experience to that described by Dr. C. T. A. Jones and others (18 August, p. 391) we wish to confirm their suggestion that hypomagnesaemia should be regarded as an important development after parathyroidectomy in patients with bone disease in hyperparathyroidism.

Our patient, a man aged 35, presented early this year with pathological fractures. Radiologically there were characteristic changes of hyperparathyroidism and gross demineralization. Serum calcium concentration was 7.3 mEq/l., with total protein 6.9 g/100 ml, plasma phosphate 0.9 mg/100 ml, serum magnesium 1.4 mEq/l., and serum alkaline phosphatase 414 IU/l. Serum parathyroid hormone concentration was 13.8 ng/ml. A carcinoma of the left upper parathyroid gland was excised (Mr. C. W. A. Falconer).

As a rapid fall in serum calcium was anticipated, a single dose of 15 mg of calciferol was given intravenously immediately after the operation and calcium gluconate was infused intravenously in an amount equivalent to 20 g of elemental calcium in five days. Mild symptoms of paraesthesiae in the extremities and around the mouth appeared when oral calcium (Sandocal) was substituted. These symptoms became more severe, with a positive Chvostek's sign and occasional spontaneous facial twitching, despite increasing the oral calcium intake to the equivalent of 12 g of elemental calcium daily along with 2.5 mg daily of oral calciferol. On the 20th day he developed dysarthria and weakness of the right lower face when the serum calcium level had fallen to 2.5 mEq/l., with serum magnesium 1.15 mEq/l. Intravenous calcium gluconate (20 ml of 10% solution) raised the serum calcium to 3.3 mEq/l. but had little effect on the symptoms. The dysarthria and facial weakness disappeared immediately after the intravenous injection over two minutes of 500 mg of magnesium chloride. Intravenous infusion of magnesium chloride

and calcium gluconate was maintained for 48 hours and then oral magnesium (Sandoz Ltd.) and calcium (Sandocal) were given. Despite the subsequent fall of serum calcium concentration to a level at which a positive Chvostek's sign with moderate paraesthesiae had previously occurred, these features did not return, possibly because the serum magnesium was now normal.

We would therefore support the contention implied by Dr. Jones and his colleagues that it is important to monitor the serum magnesium concentration after parathyroidectomy for hyperparathyroidism in patients with bone disease. We hope to publish a more complete account, including balance data, in the future.

We wish to thank Dr. J. L. H. O'Riordan for the parathyroid hormone assay.

—We are, etc.,

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Incidence and Prevalence

SIR,—It is apparent that the author of your leading article "Screening for Glaucoma" (8 September, p. 511) has regrettably failed to realize that the terms "incidence" and "prevalence" are not synonymous.

Prevalence covers all cases detected at a designated time in a certain population, whereas incidence implies new cases occurring during a defined period of time in a certain population. Thus the 55 cases detected from examining a population of 5,941 on a single occasion (a cross sectional or point prevalence study) represents a prevalence (not incidence) of 0.93%.

However, the five new cases detected from following up 212 persons over five to seven years (a prospective, follow-up, or longitudinal study) represents an incidence of 2.63% (presumably allowing for patients lost to follow-up).—I am, etc.,

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Impatient Patients

SIR,—One appreciates Dr. J. Mahmood's (8 September, p. 546) over-sensitivity and possible frustration at the mini-epidemic of patients requesting referrals for investigation before asking his advice on diagnosis and treatment. Granting these desires and then having the patient removed from his list is certainly one possible remedy, as Dr. Mahmood suggests. Another possible approach is to put the patient at ease by agreeing to all the demands for investigations, or pills and opinions, and then gently coax out the reasons for these requests. This can be quite revealing and rewarding to both doctor and patient, and usually dispenses with the need for any tests, potions, or opinions. (Description before prescription.) Should however the patient be correct in his guess he can be complimented and appropriate tests, or therapies, instituted.

Though this approach takes longer initially, the doctor fulfils his role as teacher