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Correspondents are urged to write briefly so that readers may be offered as wide a selection of letters as possible. So many are being received that the omission of some is inevitable. Letters should be signed personally by all their authors.

Penicillin-"sensitive" methicillin-resistant Staphylococcus aureus

SIR,—The occurrence of penicillin-sensitive methicillin-resistant strains of *Staphylococcus aureus* has been mentioned to me three times recently, and one of the strains, isolated at Lincoln Public Health Laboratory, was available for investigation.

Penicillin resistance in *Staph aureus* is primarily by production of penicillinase, whereas methicillin resistance is intrinsic and temperature sensitive. Penicillinase-negative methicillin-resistant strains are resistant to penicillin,[1-5] and it seemed likely that the penicillin - "sensitive" methicillin - resistant strains were in fact penicillinase-negative and that penicillin resistance would be demonstrable under conditions used for detection of methicillin resistance.

The Lincoln strain, five penicillinase-positive methicillin-resistant strains, their penicillinase-negative variants, and one other penicillinase-negative methicillin-resistant strain (all from the Cross-Infection Reference Laboratory, Colindale) were tested for penicillinase production[6] and for penicillin and methicillin sensitivity by disc tests carried out under conditions favourable (on Difco Mueller-Hinton agar at 34°C) and unfavourable (on oxoid DST agar at 37°C) for the detection of methicillin resistance.[7] Methicillin 10 μg and penicillin 2 unit discs were used.

The penicillinase-producing strains gave no zones of inhibition around penicillin discs under any conditions tested, whereas six of the penicillinase-negative strains gave zones with diameters of 21-28 mm around penicillin discs on DST at 37°C. These zone sizes were close to that of the sensitive Oxford control (30 mm) and undoubtedly some of the strains would have been reported penicillin sensitive. Under similar conditions methicillin resistance was not always obvious—10 of the 12 methicillin-resistant strains gave zones with diameters of 15 mm or greater around methicillin discs. On Mueller-Hinton agar at 34°C no methicillin-resistant strain gave zones with diameters greater than 13 mm around penicillin or methicillin discs, whereas the control zones were 32 mm and 24 mm respectively.

Thus, with the strains tested, methicillin resistance conferred resistance to benzylpenicillin and this resistance could often be demonstrated under conditions which allowed expression of methicillin resistance. In practice attention is likely to be drawn to these unusual strains even if methicillin sensitivity has not been tested because methicillin-resistant strains are almost invariably resistant to tetracycline and streptomycin and often resistant to other antibiotics. Sensitivity to penicillin in an otherwise multiply resistant strain should be viewed with scepticism, and such strains should be tested against methicillin. Methicillin-resistant strains should be reported penicillin-resistant even if apparently penicillin sensitive when tested at 37°C.

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Folic acid deficiency during intensive therapy

SIR,—We read with much interest the report of Dr R M Ibbotson and others (18 October, p 145) of two patients showing the haematological manifestations of acute folate deficiency arising in the context of critical surgical illness. These cases closely parallel those we have previously described[1] and recently reported[2] in surgical patients, particularly those receiving intravenous amino acid/ethanol mixtures. Moreover, the authors inform us that case note review has shown this form of parenteral feeding to have been used in both patients.

We would support the contention of Ibbotson *et al* that patients in intensive care units are at risk for folate deficiency for the reasons they cite; but reports of folate deficiency associated with renal failure and dialysis have indicated gradually developing blood and marrow changes over periods of many weeks, in contrast to the acute syndrome we have described. We[2] and others[3] have reported acutely developing thrombocytopaenia, leukopaenia, and megaloblastic haemopoiesis responsive to folic acid in some patients where parenteral feeding was not involved. Such cases would suggest that the stress of infection or surgery might precipitate acute changes in patients whose folate stores are already severely compromised. However, in our experience the majority of patients in whom this complication has developed over short periods of one to two weeks have been treated with amino-acid/ethanol intravenous nutrition.

While the benefit of parenteral nutrition in selected patients is undoubted, it is clear that it can be associated with a complication which, if unrecognised, may be life-threatening. We are about to submit for publication a report of a trial of folate prophylaxis which we believe is safe and effective in minimising this complication. The dose we currently use is 0·5 mg folic acid intra-