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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 production⁶ 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 oxid 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

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 described¹ and recently reported² 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. We² and others³

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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- 1 Parker, M T, and Jevons, M P. *Postgraduate Medical Journal*, 1964, **40**, (Dec Suppl), 170.
- 2 Dyke, K G H, Jevons, M P, and Parker, M T, *Lancet*, 1966, **1**, 835.
- 3 Dyke, K G H, *Journal of Medical Microbiology*, 1969, **2**, 261.
- 4 Dornbusch, K, and Hallander, H O, *Journal of General Microbiology*, 1973, **76**, 1.
- 5 Hewitt, J A, and Parker, M T, *Journal of Clinical Pathology*, 1968, **21**, 75.
- 6 Orstavik, I, and Odegaard, K, *Acta Pathologica et Microbiologica Scandinavica*, 1971, **79** (Sect B), 855.
- 7 Brown, D F J, and Kothari, K, *Journal of Clinical Pathology*, 1974, **27**, 420.

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-

venously per day. We are continuing to investigate the underlying mechanisms to develop a convenient and completely effective prophylactic regimen and to assess the role of other types of parenteral nutrition in the genesis of this complication.

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¹ International Society of Haematologists' Meeting, London, August 1975.

² Wardrop, C A J, *et al*, *Lancet*, 1975, 2, 640.

³ Chanarin, I, and Davey, D A, *British Journal of Haematology*, 1964, 10, 314.

Shoulder pain from subluxation in the hemiplegic

SIR,—In a study of shoulder pain in hemiplegia¹ seven other senior physiotherapists and myself found it to be a major barrier to progress in rehabilitation. In your leading article on shoulder pain from subluxation in the hemiplegic (14 June, p 581) you stated that supporting the arm in a sling made the patient more comfortable.

We designed several slings or supports for trial in each of our major hospitals. These increased sensory deprivation in the arm, did not solve the bed problems, and, from observation, had little effect on pain once it had started. The trials did not work because none of us could get total co-operation in keeping the appliance on. Some designs were discarded because nurses could not apply them. We tried to keep records of patients who could be put into a support on admission to casualty. But this scheme also failed as no hospital would run it—when life was perhaps at stake who could be bothered? Furthermore, the damaging traction could well have already happened when the family lifted the patient from the floor or into an ambulance. Many patients are treated at home and do not get to a physiotherapy or rehabilitation department till later in their illness. We also questioned the use of slings over a period as patients seemed to become addicted to them after they became unnecessary. And, lastly, wearing a support when walking, though good for the subluxed shoulder, adversely affected the gait pattern and, once formed, it could not be changed.

The practical problems of management are many. The doctor who orders a sling may be prescribing a treatment which cannot or will not be satisfactorily carried out. However, if doctors are more aware of the problems and try to tackle them from the start better solutions may be found.

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¹ Spencer, K, *Australian Journal of Physiotherapy*, 1974, 20, 4.

Low-dose heparin and the prevention of venous thromboembolic disease

SIR,—I would like to congratulate you on the leading article (23 August, p 447) on low-dose heparin and the prevention of venous thromboembolic disease. This presented a balanced view of the present state of prophylaxis in this most important problem. However, the following two articles

were disappointing and indeed misleading.^{1,2}

A plea for standardisation of the radioactive fibrinogen uptake test is a worthy aim. The most important components of the test are the quality of the fibrinogen and the equipment used. There is considerable variation in different batches of radioactive fibrinogen. When partially degraded fibrinogen with a high free radioactive iodine content (more than 5%) is used, bizarre counts are obtained over the limbs during the first 72 hours. This is probably due to the fact that circulating free iodine is rapidly excreted and gives low counts on subsequent days. As regards the equipment, when dealing with low counts the ratemeter is less sensitive and errors in interpretation of results are commonly seen. If the precautions outlined by Kakkar *et al*³ are followed, then such mistakes are eliminated. It is essential that after the counts in one leg have been completed the radioactivity over the reference point (heart) is again recorded to check the baseline before the second limb is scanned. This reduces the effect of "drift" in the machine. I have never observed the pattern of leg counts shown in table II of the article under discussion.¹

In the second article² the claim is made that intermittent calf compression results in a reduction of 90% in the incidence of deep vein thrombosis. It follows from this observation, therefore, that one could hope at the very most for a 10% improvement. Under such circumstances the all-important consideration is the number of patients in the trial. To be confident of showing a 10% improvement one would require about 1000 patients in the study.⁴ With 85 patients the chance of obtaining a significant result is that of the toss of a coin. No conclusions from the study can be reached.

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¹ Roberts, V C, *British Medical Journal*, 1975, 3, 455.

² Roberts, V C, and Cotton, L T, *British Medical Journal*, 1975, 3, 458.

³ Kakkar, V V, *et al*, *Lancet*, 1970, 1, 540.

⁴ Boag, J W, *et al*, *British Journal of Radiology*, 1971, 44, 122.

Deaths in asthma

SIR,—A 46-year-old known asthmatic but otherwise healthy woman was admitted to hospital after a severe episode of asthma. She had been at a night club and had had a few drinks when the attack started. With the help of a friend, she retired to the cloakroom and was seen to take repeated puffs from her isoprenaline inhaler. On leaving the cloakroom she collapsed to the floor and made further efforts to use the inhaler. An ambulance was called but by the time of arrival in hospital she was in asystole. Despite resuscitation the patient died without recovering consciousness. Identification of propellant gases in a blood sample taken some hours after admission indicated that a substantial amount of propellant gas had been inhaled and confirmed excessive use of the inhaler. At necropsy no other underlying condition was found.

The epidemic of asthma deaths in the 1960s coincided with the increased use of pressurised aerosols.¹ The dangers associated with their overuse have become well recognised.² Selective sympathomi-

metic drugs are now available and we wondered whether our patient would have been better off using a bronchodilator with selective B₂ adrenergic stimulant effects rather than isoprenaline.

The Committee on Safety of Drugs informed us that since 1970 it has had reports of 31 asthmatic deaths among patients using isoprenaline inhalers and of a similar number of deaths (31) in patients using salbutamol inhalers. All the deaths were due to cardiorespiratory failure with underlying ventricular arrhythmias. Although in recent years selective B₂ bronchodilators may have been used in greater quantities than isoprenaline, it is also likely that abuse of either bronchodilator can have serious consequences. Another question raised by this case is whether the cardiostimulant effects of bronchodilators are potentiated in subjects drinking alcohol.

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¹ Inman, H W, and Adelstein, A M, *Lancet*, 1969, 2, 279.

² Committee on Safety of Drugs, *Aerosols in Asthma*, Adverse Reactions Series, 1967, No 5.

Management of acute asthma

SIR,—Your leading article under this title (11 October, p 65) was in fact concerned with *persistent* severe asthma or status asthmaticus, and the diagnostic and therapeutic suggestions which you gave were therefore relevant only to management in hospital, to which you say all such patients should be sent. But acute asthma in the sense of a sudden worsening of chronic asthma or a severe attack in a mild asthmatic (from exposure to an allergen or bronchial irritant or coming out of the blue) must be treated *before* the patient is sent to hospital. It is when general practitioners take your advice and just send for an ambulance to take such patients to hospital that they too often arrive dead, tragically and quite unnecessarily.

No patient with acute asthma should be sent to hospital without first being given 200 mg of hydrocortisone or prednisolone intravenously or intramuscularly. You suggest that intravenous steroids take an hour to have any effect, but the study on which you base this opinion was based on changes in ventilatory tests such as peak flow, FEV₁, and PEF₁.¹ Symptomatic relief may appear much sooner than that—within as little as 15 minutes in my experience. This may be due to a reduction in total lung capacity which can relieve asthmatic distress without affecting ventilatory capacity.² I have seen considerable relief even from oral prednisolone within half an hour. Even if this relief of bronchospasm may be partly psychogenic owing to the patient's confidence in the value of steroids based on past experience, it is none the less real and may be life-saving.

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¹ Ellul-Micallef, R, and Fenech, F F, *Thorax*, 1975, 30, 312.

² Woolcock, A J, and Read, J, *Australian Annals of Medicine*, 1966, 15, 196.