other small, close-knit communities this seems quite astonishing, even allowing for the possible difficulties in giving injections to some patients with Down's syndrome.

I would be most interested to know if this unfortunate failure to offer BCG to these children, originating from preorganisation days, is merely a local quirk or a more widespread phenomenon.

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¹ Crofton, J. and Douglas, A. *Respiratory Diseases*, 2nd edn. Oxford, Blackwell Scientific, 1975.

Propriety of exotic treatments

SIR,—Not infrequently in the press there are reports about parents, friends, and volunteers collecting money to send patients abroad to receive medical treatments not practised in Britain, but which are claimed to be beneficial by those who offer them. For the sake of patients, their relatives, and doctors it is time that some responsible medical body investigated some of these treatments and the methods of their operators.

Recently, for example, the parents of a patient I know were in touch with a clinic abroad. On the basis of a brief medical report and without seeing the patient this clinic offered a course of expensive treatments. Hope springs eternal in the minds of most parents of the handicapped and in their anxiety they are tempted to seize any opportunity which seems to give a chance of improving the patient. The physician in these cases, who has usually little knowledge of the treatment involved, is placed in a difficult position. If he advises the parents against the treatment they might consider him biased or out of date. The layman, who often accepts what he sees in print as absolutely authentic, might regard the existence of allegedly successful treatments abroad as reflecting some deficiency, inadequacy, or conservatism on the part of British doctors or the National Health Service.

It is one of the ethical tenets of the medical profession that the doctor who discovers an effective treatment does not keep this secret for his own profit. The exploitation of the anxiety of parents and relatives who are persuaded to part with a fortune to obtain a treatment of doubtful or unproved efficacy is one form of private practice that deserves legitimate scrutiny.

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Misuse of tubular elasticated bandages

SIR,—The ready availability of tubular elasticated bandages has led to their increasing use for everything from dressings to fractures, from varicose veins to fallen arches. Not only are untapered tubular compression bandages seldom indicated, but indeed they can be positively damaging. My specific concern, which has prompted this letter, is their inappropriate application in vascular disease.

The fact that a leg in which there is venous or lymphatic insufficiency is likely to benefit from elastic compression has resulted in the application of these bandages in every sort of vascular condition. For example, one not uncommonly sees them applied to patients who have the dependent oedema of arterial

disease. In these circumstances, in which the arterial perfusion pressure is already severely reduced, elastic compression may be all that is required to devitalise tissue and precipitate gangrene. So far as venous conditions are concerned it is not unusual to see patients lying in bed with tubular elastic bandages applied from thighs to toes as prophylaxis or treatment of venous thrombosis. There is no evidence to justify this type of compression in the immobile patient. Furthermore, the effect of an untapered elastic tube applied to a limb is to act as a tourniquet at its proximal end. This is made worse by the fact that such a bandage applied to the thigh, unless special precautions are taken, always rolls at its upper end. Indeed, we have seen circumferential ulceration from this cause and aggravation of peripheral oedema is commonplace.

The benefit from elastic compression in venous and lymphatic disorders derives mainly from its combination with activity of the calf muscles. The elastic compression should be maximal distally and the girth should be graduated to match the leg. Only in the more severe forms of venous insufficiency or lymphoedema is elastic compression above knee level necessary. For male patients it is our practice generally to provide knee-length graduated hose. Women usually prefer support tights for cosmetic reasons, but if firm elastic support is required a better combination is knee-length graduated hose which can be disguised by wearing tights on top. Patients are instructed to put on their support hose before rising in the morning. If there is oedema the foot should be elevated above the level of the hip whenever the patient is resting. If the patient is confined to bed the affected leg should be elevated on pillows or the foot of the bed raised on blocks.

There is now a sufficient selection on the market of physiologically satisfactory elastic supports ranging from tapered tubular bandage to fully fashioned graduated hose. It is to be hoped that the untapered varieties will soon be allocated not to limbs but to limbo.

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Compression sclerotherapy of varicose veins

SIR,—The excellent article by Professor P H Fentem and others on leg bandaging for varicose veins (25 September, p 725) gives us more useful information about the effects of static compression. The range of compression (20–100 mm Hg) produced by bandages in his series illustrates very well how unpredictable the effects of bandaging are. While in theory bandages may be applied to give a desired compression, in practice their application cannot be standardised and therefore we feel they should be discarded altogether if they are being applied for their effect on blood flow.

A much more effective way of providing static compression of known degree is by a properly designed, fully tailored, and carefully fitted elastic stocking. I must stress that I do mean properly made elastic stockings and not cylinders of elastic material (such as Tubigrip) which, although often referred to as stockings, quite obviously are not stockings. The degree of compression produced by a stocking may be varied as required by altering the amount of

elastic material incorporated in the fabric from which it is made. Not only does this allow alteration in overall compression but, more important, it allows alteration in compression at local sites on the limb. For example, it is now perfectly simple to make a stocking that gives higher levels of compression over the ankle and calf than over the knee and thigh or vice versa. What level of compression should we apply? This depends entirely on what function is required. Suffice it to say that it will be very different for supine and upright ambulatory patients.

In 1948 Halperin *et al*¹ studied the effects of incremental rises in pressure on the circulation of the limb in supine patients. A 10-mm Hg compression produced a decline in blood flow of 10% and a 30-mm Hg compression a decline in blood flow of 25%. In 1954 Litter *et al*² indicated that a compression of greater than 60 mm Hg was likely to lead to arterial insufficiency. In Professor Fentem's series a level of compression higher than this occurred nine times (18%). It would seem likely, therefore, that a large number of patients are having bandages inappropriately applied. It really is time we discarded bandages as a compressive device. At best they are of limited value and at worst they are positively dangerous.

A sensibly designed stocking which provides a constant, safe compression of known degree would appear to be the answer. Then it may be possible to obtain an accurate assessment of the benefits of static compression in a variety of circumstances.

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¹ Halperin, M H, Friedland, C K, and Wilkins, R W, *American Heart Journal*, 1948, **35**, 221.

² Luter, J, and Wood, J E, *Journal of Clinical Investigation*, 1954, **33**, 798.

Bilateral injuries in childhood: an alerting sign?

SIR,—The examination of children following injury increasingly presents family practitioners with difficulty in establishing whether the trauma sustained has occurred accidentally or not. In order to provide assistance in this situation the area child health service in Leicestershire has nominated a small number of senior clinical medical officers who have developed an expertise in the diagnosis of non-accidental injury by seeing most of the cases which arise within the area and are then available to give advice and, where necessary, attend court to give evidence.

Experience so far has shown that with severe injuries the diagnosis is relatively straightforward and in any event transfer to hospital is essential. Where children present with minor trauma, however, such as repeated bruising and small burns, the diagnosis may prove extremely difficult. After discussions with the staff concerned the problem seemed to indicate a need for more information about the normal range of minor injuries experienced by children under school age.

As a result arrangements have been made through the social services department for Mr M J Sargeant, a reader at Loughborough University of Technology, to undertake a project in which children attending 10 day nurseries in Leicestershire were inspected every day and all injuries, however slight,

recorded. This research is not yet completed and full details of the project will be published in due course. In the meantime it seemed important to give early notice of the following observation.

During 25 days in November and December last year 9253 inspections were made on 481 children on the registers and some 1543 injuries were located and recorded. None of these injuries were found to be bilateral symmetrically about the main axis of the body—for example, affecting both sides of the head or both arms.

This suggests that symmetrical bilateral injury is a comparatively rare occurrence and indicates the need to alert all doctors and staff to be particularly vigilant when seeing children with bilateral trauma.

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Postmenopausal urinary symptoms and hormonal replacement therapy

SIR,—Recent discussion on the management of the menopause has failed to mention the possible effects of oestrogen deprivation on the lower urinary tract. In a study of a large number of women with lower urinary tract symptoms¹ it was shown that a significant proportion first experienced their urinary symptoms after the menopause, often in association with a senile atrophic vaginitis, an association previously noted by Everett.² It was suggested that the urinary symptoms were due to oestrogen-deficient changes in the urethra similar to those producing the senile vaginitis.

The natural variations in oestrogen activity occurring throughout a woman's life are reflected by changes in the maturation of the squamous epithelium covering the distal urethral segment.³ Adequate levels of oestrogen are necessary for maturation of this epithelium, failure of which may give rise to atrophic changes similar to those of senile vaginitis. At first these postmenopausal urinary symptoms were thought to be due to the presence of distal urethral stenosis, and hormone replacement therapy was combined with urethral dilatation.¹ However, in a recent clinical study 18 postmenopausal women with lower urinary tract symptoms were treated by oestrogen therapy alone (Premarin 0.625 mg daily for three weeks out of four, for a total of three months). Of the eight women whose urinary symptoms had been present on average for less than 12 months this treatment relieved the symptoms in six. In the remaining 10 patients symptoms had been present for more than 12 months and only two of these were relieved of their symptoms by this hormone therapy alone.

This suggests that the early symptoms of postmenopausal atrophic urethritis are probably due to epithelial changes alone and hence can be corrected by hormone replacement therapy. At a later stage fibrosis and stricture formation may complicate the epithelial changes and urethral dilatation is required in addition. This hypothesis now forms the basis of a more detailed prospective study, but it would suggest the prompt use of hormone replacement therapy in those postmenopausal women presenting with lower urinary tract symptoms of frequency and dysuria. It would be interesting to hear from other workers whether hormone replacement therapy has

been effective in treating such symptoms and whether the duration of urinary symptoms has been a significant factor in the success of the treatment.

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¹ Roberts, M, and Smith, P, *British Journal of Urology*, 1968, **40**, 694.

² Everett, H S, *American Journal of Surgery*, 1941, **52**, 521.

³ Smith, P, *British Journal of Urology*, 1971, **44**, 667.

Drug treatment of typhoid fever

SIR,—I read with great interest the paper by Surgeon Lieutenant Commander P D Clarke and others on "Mecillinam: a new antibiotic for enteric fever" (3 July, p 14). I agree that further information is required on the question whether mecillinam is also effective in typhoid fever due to organisms with the R-factor-mediated multiple resistance to chloramphenicol, tetracyclines, sulphonamides, and streptomycin which has been known for several years now.¹ I do not agree, however, with the statement of the authors that "chloramphenicol-resistant *Salmonella typhi* strains, such as those that caused the large typhoid epidemic in Mexico, are sensitive to ampicillin." R-factor-mediated ampicillin resistance among *S typhi* strains has been reported from that same typhoid epidemic in Mexico² and, apart from individual cases in France and Algeria, has also been reported from South Vietnam and Thailand.³ Ampicillin-resistant *S typhi* strains have been noted in India⁴ as well. If there exists no cross-resistance to the closely related ampicillin and amoxycillin, then mecillinam could be a valuable "addition to the agents available for treating typhoid." The exact mode of action of this new amidino penicillin is, however, not yet known and the question of cross-resistance is still open.⁵

Considering the risk of irreversible aplastic anaemia, the various drawbacks in the treatment of acute typhoid fever (lack of influence on the relapse rate³ and reconvalescent excretor state³ and the occurrence of a toxic crisis in 5-10% of treated cases^{3 6}) and the alarming spread of R-factor-mediated chloramphenicol

resistance throughout the world (see figure³), I doubt whether it is still justified to call chloramphenicol the drug of choice in typhoid (except for economic reasons—for example, in developing countries). Ampicillin was for years the only valuable alternative agent, but the response of acute typhoid fever to ampicillin is at least 1-3 days slower than that to chloramphenicol and the failure rate can reach 30%.³ Amoxycillin gave promising results,⁷ but there is a complete cross-resistance to ampicillin.

Reviewing the literature on the chemotherapy of typhoid fever³ I found that co-trimoxazole has not only a therapeutic efficacy equal to that of chloramphenicol as measured by the time for defervescence and for improvement of the patient's general condition, but has moreover the advantage of not causing a toxic crisis in the cases treated^{3 6} and of even being effective, although somewhat more slowly, in cases of multiple drug-resistant *S typhi* infections (including sulphonamide resistance).⁷ It is worth noting that the synergistic effect of the two components of co-trimoxazole (sulphamethoxazole and trimethoprim) has been shown not only in infections due to various sulphonamide-resistant bacteria in vitro⁸ as well as in clinical trials in man⁸ but also in R-factor-mediated sulphonamide resistance in *S typhi* in vitro,⁹ the latter finding being, however, contrary to the findings of Anderson.¹ In view of the above-mentioned qualities and its good tolerance¹⁰ co-trimoxazole seems to be at present the drug of choice in acute typhoid fever.

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¹ Anderson, E S, *Lancet*, 1973, **2**, 1494.

² Olarte, J, and Galinder, E, *Antimicrobiological Agents and Chemotherapy*, 1973, **4**, 597.

³ Herzog, C, *Infection*, in press.

⁴ Solomon, S, Subramaniam, S, and Madanagopalan, N, *Current Medical Research and Opinion*, 1976, **4**, 229.

⁵ Roholt, K, Nielsen, B, and Kristensen, E, *Chemotherapy*, 1975, **21**, 146.

⁶ Kamat, S A, *British Medical Journal*, 1970, **3**, 320.

⁷ Gilman, R H, et al, *Journal of Infectious Diseases*, 1975, **132**, 630.

⁸ Acar, J F, Goldstein, F, and Chabbert, Y A, *Journal of Infectious Diseases*, 1974, **128**, suppl, p 470.

⁹ Bushby, M R, and Bushby, S R M, in *Proceedings of 9th International Congress of Chemotherapy*, London, 1975. In press.

¹⁰ Havas, L, Fernex, M, and Lenox-Smith, I, *Clinical Trials Journal*, 1973, **10**, 81.

● single cases ▲ epidemic outbreaks □ endemic occurrence
Incidence of single cases ● and epidemics ▲, and endemic □ occurrence of typhoid fever due to *S typhi* with R-factor-mediated resistance to chloramphenicol and other antibiotics throughout the world, 1967-74.³