had a good initial response to tetracosactrin and those who did not, nor indeed between those with a low level of circulating plasma cortisol and those with a normal level. It is interesting, however, that the one patient who did show signs of a major complication during the attempted substitution had both a low level of plasma cortisol and a very poor response to tetracosactrin.

Another observation which we feel is worth making is that all our patients' asthma was well controlled on a maintenance dosage of prednisone before we attempted substitution with beclomethasone dipropionate. We have found that it is extremely difficult, if not impossible, to make this substitution if the asthma is not well controlled initially by corticosteroids administered other than by inhalation.—We are, etc.,

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Cholestyramine and Diabetic and Post-vagotomy Diarrhoea

SIR,—We wish to report four diabetic patients with intractable diarrhoea responding to oral cholestyramine, a resin which combines with bile acids to form insoluble complexes.

The first, a 37-year-old male diabetic with neuropathy and retinopathy, had had diarrhoea (8–20 stools daily) for 16 years. Pancreatic extracts, codeine phosphate, antispasmodics, diphenoxylate hydrochloride, and methylcellulose had little effect. Treatment with cholestyramine 4 g thrice daily with meals resulted in 1–2 solid stools daily. Diarrhoea recurred twice when cholestyramine was withdrawn, but restarting therapy resulted in relief. In the second patient, a 52-year-old man with neuropathy and diarrhoea unresponsive to other forms of therapy, solidification of the stools and reduction in frequency from 6–8 to one daily occurred when cholestyramine was given. Two other patients with intractable diarrhoea also responded to oral cholestyramine. In both cases withdrawal of cholestyramine caused a return of the diarrhoea, which was controlled again within 24–48 hours by restarting treatment.

The only known type of diarrhoea responding to cholestyramine is that following disease or resection of the terminal ileum,^{1,2} when excess bile salts enter the colon, inhibit absorption of water and electrolytes, and disturb colonic activity.^{3,4} Feeding bile salts induces diarrhoea^{5,6} and since no ileal disease is demonstrable in diabetic diarrhoea, it is possible that excess bile is entering the gut at "inappropriate" times.

Diabetic diarrhoea is associated with neuropathy,⁷ and the vagus, which supplies fibres to the biliary system, may be involved. Indeed post-vagotomy and diabetic diarrhoea have many features in common—watery stools, sometimes steatorrhoea, delayed gastric emptying, and disordered small-intestinal transit. Vagotomy may result in increased gall bladder volume and poor contraction.^{8–10} Diabetics also have large-volume poorly contracting gall bladders¹¹ and a high incidence of gall stones occurs both following vagotomy and in diabetics.^{12,13} Furthermore, there is no demonstrable disease of the gut and response to therapy is poor, though the incidence of post-vagotomy diarrhoea is reduced by preserving the coeliac and hepatic vagal branches.

Though bile flow is increased after vagotomy,¹⁴ the concentration of bile salts in the postprandial aspirate is low,¹⁵ suggesting that gall bladder contraction is out of phase with gastric emptying, resulting in poor admixture of bile and chyle. If gall bladder volume is greater than normal, increased amounts of bile salts might enter the small intestine, swamping its reabsorptive capacity and (deconjugated by bacteria) inducing "colonic" diarrhoea.

Both post-vagotomy and diabetic diarrhoea may therefore be related to excess bile salts entering the gut as a result of gall bladder dysfunction following severance or neuropathy of the vagus.—We are, etc.,

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Vaccination of Smallpox Contacts

SIR,—In his Milroy lecture on antiviral chemotherapy (4 August, p. 275) Dr. D. J. Bauer stated that "persons who have been in contact with smallpox infection are vaccinated routinely, but if they have not been vaccinated at some time in the past the development of immunity is too slow to protect them against developing the disease." With this latter phrase we disagree. It is generally accepted that successful vaccination within up to about 48 hours after exposure will usually protect contacts against smallpox whether they have been previously vaccinated or not. The earlier the vaccination after contact, the greater the chance of prevention of the disease, but vaccination even within three or four days of contact produces some measure of protection by modifying the disease. The reason for this protective effect is that the immunity which follows vaccination develops more rapidly than that following infection by smallpox virus. The efficacy of the vaccination of contacts (whether previously vaccinated or not) has been largely responsible for the nearly world-wide eradication of smallpox carried out by the World Health Organization and for the success of the epidemiological control

methods which are used when smallpox is imported into this country.

In view of Dr. Bauer's statement we reviewed some of the literature on the protective effect of vaccination of contacts of variola major in Britain.^{1–6} In general, the success of vaccination of contacts is indisputable, though there are some reports of failures in protection of up to 10% of contacts. Some of these failures appear to have been due to the fact that vaccination was done late in the incubation period and no one should be surprised that vaccination does not protect when done late in the first week of the incubation period. Other failures could be attributed to faulty technique during panic vaccination and low-titre lymph. However, we have no evidence to shake our belief in the protective value of immediate vaccination of contacts nor any data which support Dr. Bauer's statement that if contacts have not been vaccinated in the past, "the development of immunity is too slow to protect them against developing the disease." There is no evidence that there is anything better, apart from the concomitant use of antivaccinal immunoglobulin in close family and household contacts.—We are, etc.,

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Amyloidosis Complicating Actinomycosis

SIR,—In the discussion of the case of actinomycosis and amyloidosis that was the subject of the Clinicopathological Conference at the Royal College of Physicians of London in July (20 October, p. 149) the question was raised whether actinomycosis predisposes to amyloidosis. Since reporting a case of this association in 1956¹ I have been collecting material from cases of actinomycosis in connexion with a study of fungal infections. This series now includes 112 cases of visceral (including septicaemic) actinomycosis and 174 cases of faciocervical actinomycosis. In the visceral group 68 patients died with active actinomycosis; in 63 of these cases the infection (including its complications) was considered to be the cause of death; the other five patients died of unrelated causes. In the faciocervical group 19 died with active actinomycosis; in 17 the infection was the cause of death. In all the other cases in both groups the infection was considered to have been cured. All cases were characterized by sinus formation. No case of faciocervical actinomycosis was found to be associated with amyloidosis. In contrast, amyloidosis was found in 14 of the 112 cases of visceral actinomycosis; 12 of these were among the 63 patients whose death was attributed to the infection. The amyloid deposition was of only microscopic extent in five of these 12 cases; it was macroscopically evident in seven—six of these seven patients had symptoms that, in retrospect, were attributable to

amyloidosis. The other two patients with amyloidosis were among the 49 whose visceral actinomycosis was, reasonably, considered to have been cured. In one of these there was good evidence of regression of the amyloidosis since cure of the infection five years before; the other died of bacterial peritonitis complicating severe renal amyloidosis, symptoms of which antedated the completion of treatment for actinomycosis two years before death (there was no evidence of persistent actinomycosis at necropsy).

The average duration of the infection, judged from the clinical history, was about 23 months for the 51 fatal cases of visceral actinomycosis without amyloidosis and about 51 months for the 12 with amyloidosis (58 months for the six with symptoms due to amyloidosis).

Infection by other actinomycetes may also cause amyloidosis. Thus amyloidosis may complicate streptomycotic mycetoma, but—with rare exceptions—only in those uncommon cases in which the infection has spread to regional lymph nodes or viscera. The same is true of mycetomas caused by *Nocardia brasiliensis*. *N. asteroides*, an aerobic and often acid-fast actinomycete, has not, to my knowledge, been associated with amyloidosis, probably because the infections that it causes are usually rapidly fatal or rapidly cured.—I am, etc.,

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¹ Symmers, W. St. C., *Journal of Clinical Pathology*, 1956, 9, 212

The Suspended Retractor

SIR,—With reference to my letter on the use of the suspended Doyen retractor (4 August, p. 294) and the correspondence thereafter, I must record that this idea was first thought of by Mr. M. Planer in 1963 when he was working at Derby City Hospital. After his return to South Africa he published a description of its use in difficult vagotomies.¹ Mr. G. Dimopolous, to whom I wrongly attributed the idea, happened to be one of the audience in the theatre when Mr. Planer demonstrated this technique in 1969.—I am, etc.,

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¹ Planer, M., *South African Medical Journal*, 1970, 1, 13.

Granular Support in Prevention and Treatment of Bedsores

SIR,—A variety of measures used in the management of bedsores were mentioned in the seminar at the recent postgraduate meeting in Guernsey (13 October, p. 99). For many years "granular support" in the form of suitable-sized mattresses or cushions filled with granules and placed under pressure points has proved successful in the prevention and cure of ischaemic ulcers in my experience and that of a number of others to whom this method has been introduced.

The semi-fluid nature of granular support spreads the load over the whole of the under surface, as in floating, while the friction between the granules affords stability and allows relaxation in an infinite variety

of positions of comfort. In this way the joints of the spine or limbs get painless, relaxed support that cannot be ensured in other ways. Water- and air-filled mattresses or cushions require a constant degree of muscle tone to maintain any pain-free position selected for a painful joint, the patient waking in pain when relaxation into a painful phase of the joint occurs. The same is true with latex foam, while plastic foam on the other hand packs into a firmly lined indentation which limits adjustment from an uncomfortable posture.

For domestic use wheat (millable grain) has proved a practical and effective filling for a "half mattress" (28 lb for 24×36 in=12.6 kg for 60×90 cm) under the trunk from shoulders to hips on an ordinary spring bed.¹ Plastic granular fillings (beads of expanded polystyrene) are extremely light, are washable, and, suitably treated, have the right amount of friction between the granules, like those of wheat. The hollow propylene balls used for aircraft pilots' seats, regardless of expense, may have advantages, but clinically mattresses filled with the expanded polystyrene beads have been effective over the last two years in my personal experience in a limited number of cases in general practice and in nursing homes and a mental hospital (waterproof covered). The design must be right. The cost of this filling is less than the cost of the materials for the covers and manufacture, so that it cannot be called expensive. Present supplies are limited, as are those of all plastics.—I am, etc.,

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¹ Tracey, J. B., *Practitioner*, 1970, 204, 845.

The Magic Diploma

SIR,—Professor James H. Hutchison's Personal View (4 August, p. 288) was bound to stimulate a lively correspondence, and all the overseas doctors who have responded are to be congratulated on many constructive observations.

Dr. K. Raghavan's letter (13 October, p. 108), raises some important points. Being from India, he will appreciate that though official statistics on our overall dependence on overseas doctors in the junior hospital grades do not distinguish between the various countries of origin, it is widely recognized that doctors from the Indian sub-continent, particularly from India itself, greatly preponderate. For various reasons, readily understandable, these doctors also aspire far more than those from other countries to a permanent career in Britain. Dr. Raghavan rightly deplores the apparent failure of the Government of India—after nearly 30 years of independence—to provide a reasonable livelihood, congenial prospects, and proper working conditions for more than a minority of those of her doctors wishing to specialize within their own communities surrounded by their own cultures. Rather are they often compelled to accept relatively less satisfying posts in Britain or the U.S.A., in order legitimately to earn a reasonable living. Furthermore, while a satisfactory solution to our own problems of hospital staffing seems endlessly evasive, it is scarcely deniable that the ultimate responsibility rests squarely on successive U.K. Governments for continuing to rely so heavily, in the

junior hospital grades, on so many overseas doctors instead of insisting that most of them return home in due course, where some at least of the skills they have learnt from us are so desperately needed.—I am, etc.,

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Delayed Drug-induced Dystonias

SIR,—Dr. M. A. Simpson's comments from Ontario (20 October, p. 174) amply confirm my own recent experience while providing intermittent general medical intake cover at a hospital in Cardiff. Over a two-year period I attended approximately six patients (including two in one evening), all in the casualty department, all suffering from variants of drug-induced extrapyramidal disorder, all undiagnosed by the attending casualty officers, several referred from other units undiagnosed, and all causing considerable distress to those accompanying them. All of these cases responded within minutes to the intravenous administration of procyclidine.

It would appear from my experience with the phenothiazines and butyrophenones over some years (and most of us are caught out initially) that many doctors at all levels are not aware of the hazards for some of their patients when given such drugs, though these are well documented,¹ and that they are often given to patients with, or who are thought to have, personality disturbance, temporary or long-term. In particular, all casualty officers should consider the possibility of side effects to these groups of drugs in patients who present with recent dysarthria, torticollis, muscle spasm, tongue protrusion and retraction, and abnormal movements of eyes or limbs. As Dr. Simpson rightly says, the intake does not have to have been on the day the patient is seen.—I am, etc.,

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¹ Shepherd, M., Lader, M. H., and Lader, S. R., in *Side Effects of Drugs*, ed. L. Meyler and A. Herxheimer, vol. 7, p. 69. Amsterdam, Excerpta Medica, 1972.

SIR,—Some time ago (12 February 1972, p. 441) you published a short comment of mine about the use of single injection of massive doses of phenothiazines. Dr. M. A. Simpson offers a helpful warning (20 October, p. 174) concerning "delayed dystonia," but I think it is a pity that he does not question the use of a single large dose of haloperidol for a "suicidal young male." Would not a different preparation in suitable (repeated) doses have been more appropriate?—I am, etc.,

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Non-accidental Injury to Children

SIR,—Few will disagree with the recommendations of the Tunbridge Wells Study Group (13 October, p. 96). However, we are all fallible and I would ask the first doctor and consultant dealing with a case of suspected child abuse to be especially careful. Incautious questioning, with the subsequent interviews by well-meaning people, can