It was decided to try the effects of immunosuppressant therapy in this case since there were certain features similar to those of massive pulmonary fibrosis and a possibility that a delayed immune response might account in part for the lung damage. He was started on azathioprine 50 mg four times daily and in addition was given potassium aminobenzoate as an antifibrotic measure. The effect of the immunosuppressant therapy was monitored by frequent blood counts and immunophoresis of gammaglobulins. The latter showed proportional reductions after therapy started, the IgG level being reduced to 5·83 g/l (583 mg/100 ml).

On 23 December the lung appearances began to improve, starting on the right side and to a lesser extent the left, and on 30 December there was further resolution of the lesions. Concomitantly with this his blood urea level began to fall and reached 10·3 mmol/l (62 mg/100 ml), the creatinine level being 61·9 μmol/l being (0·7 mg/100 ml), and Po_2 steadily rose. These changes were all associated with a progressive clinical improvement which has been maintained, though his lung changes have not yet completely resolved.

I hope to publish further details concerning this patient at a later date but feel that while this isolated case may "prove nothing," in view of the usually grave prognosis associated with severe paraquat poisoning, especially when associated with marked pulmonary radiological signs, this patient's response may be of interest to clinicians who may be dealing with such cases, particularly late cases in which the use of adsorption agents such as Fuller's earth and haemodialysis are not applicable.—I am, etc.,

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Septicaemia on the Increase

SIR,—Your leading article on septicaemia (14 December, p. 615) reminds us that "the intravenous tubes now so commonly used are a convenient portal of entry for these infections." One way in which such infection can enter is by accidental contamination of the piercing needle of the giving set when it is removed from an empty bottle and inserted into a full one. This change over is made some 1300 times each week in the surgical wards in Aberdeen hospitals. Therefore the procedure should be as simple and safe as possible.

Currently we use Avon 11 giving sets, as we have done for 16 years since a Medical Research Council subcommittee[1] devised and recommended them, and they have proved highly satisfactory. They have a combined air-inlet and piercing needle, a good grip can be obtained on the rigid filter chamber, a flange prevents the operator's fingers touching the needle which pierces the bung, and nurses find them easy and quick to manipulate. It is therefore surprising to find that official recognition has been withdrawn from this set. There are now only two giving sets listed in the D.H.S.S. schedule of medical and surgical equipment (13 May 1974, p. 1408)—the Avon A 15 and the Travenot F.K.C. 2005. Both have been designed for use with plastic bags containing stored blood and other derivatives and are excellent for this purpose. Because a plastic bag collapses as it empties there is no need to incorporate an air-inlet into these sets. When, however, these sets are used to give fluids supplied in glass bottles a separate air-inlet assembly must be inserted through the bung in order to admit air while fluid leaves the rigid bottle.

The A 11 set seems to have been withdrawn because blood and its byproducts will soon be generally distributed in plastic bags, and it is not safe to penetrate a plastic bag with the piercing needle of the A 11 set. No one disagrees that the A 15 set is essential here. However, our pharmaceutical colleagues expect intravenous electrolyte and dextrose solutions, dextran, amino-acid solutions, and fat emulsions to be supplied in glass bottles for some years to come. In Aberdeen during 1974 the pharmacy has manufactured infusion fluids in 135 000 500-ml glass bottles and half these bottles have been used on the five general and paediatric surgical wards. The transfusion of blood is largely confined to the day of operation, and many patients do not require any blood. On the other hand, most patients who undergo major alimentary-tract surgery or who come in with peritonitis or intestinal obstruction cannot drink for some days. During this time they receive solutions of sodium, potassium, and dextrose, and in my adult ward it is usual to have about eight patients on this treatment at one time. If we have to use the two official sets for all these infusions then every time a bottle is changed two needles must be changed instead of one on some 1300 occasions each week. Not only is this unnecessary work but there is a serious additional risk of contamination, because the separate air-inlet needles supplied are difficult to hold and to introduce through the thick bungs of the bottles. The likelihood of a nurse's fingers contaminating the lower end of this needle is high.

This risk is unacceptable in any patient but it is especially dangerous practice among patients on long-term intravenous feeding. The great care taken over the insertion of the necessary central venous catheters and over their maintenance over days and weeks can be frustrated by the use of giving sets never designed to be used with glass bottles. It is no argument to claim that these sets are currently used without trouble. Everyone knows that intravenous lines can become infected and how serious this is; can we exclude faulty technique of needle insertion into solution bottles as one source of infection? Happily we can still purchase A 11 sets and avoid this risk, but for how long will manufacturers continue to make a set which is not officially recognized?

The D.H.S.S. appears to have restricted recognition of the A 15 and F.K.C. 2005 sets on the grounds that it is more economical and less confusing to have only one type of giving set available which will safely enter a bag of blood. This is understandable, but in fact we have to keep both the A 11 and A 15 sets at present, because blood sometimes arrives in bags, and no confusion arises. The major fact which has been overlooked is the very large amount of electrolyte and dextrose solutions used on medical wards, all of which are supplied in glass bottles. I and many of my nursing and surgical colleagues believe that there is an overwhelming case for allowing the A 11 to join the A 15 as an officially recognized giving set.—I am, etc.,

PETER F. JONES

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1 Medical Research Council Blood Transfusion Research Committee, *Lancet*, 1957, 1, 595.

Ischaemic Heart Disease in Young Women

SIR,—Though he gives no figures of his own, Dr. I. McD. G. Stewart (14 December, p. 653) doubts the validity of my assessment (2 November, p. 253) of the prevalence (34%) of hypertension in women who developed ischaemic heart disease under the age of 45, and some clarification appears necessary.

(1) All women classified as having hypertension were recorded by me as having a diastolic blood pressure of 100 mm Hg or more and the lowest reading of three taken over a period of 10 minutes was used for this criterion. Of 50 women so classified, 43 (28 out of 32 with myocardial infarction) had a diastolic blood pressure of 100 mm Hg or more recorded on three or more separate clinical examinations. None were regarded as hypertensive solely on the basis of treatment "at the hands of their family doctor."

(2) In 23% the electrocardiogram showed left ventricular hypertrophy. This was defined in the customary way as $R_{aVL}>11$ mm or $R_{aVF}>20$ mm and $S_{V1}+R_{V6} \geqslant 35$ mm: obviously, S-T and T-wave abnormalities would be of little value in the assessment of left ventricular hypertrophy in patients with previous myocardial infarction.

(3) The prevalence of diastolic hypertension (28%) or of left ventricular hypertrophy in those presenting with angina was only slightly, and not significantly, less than that in those with myocardial infarction. This supports the view that hypertension is an important pre-infarction risk factor in young women.

(4) While a prospective study would probably give more reliable data about the prevalence of each risk factor, Dr. Stewart should consider the magnitude of implementing such a study in women of this age group, in which the incidence of myocardial infarction is so small, and ask himself why no comparable data are available even from the Framingham survey or from the Inter-Society Commission for Heart Disease Resources (Pooling Project)—two of the largest prospective surveys of risk factors in relation to ischaemic heart disease.

(5) The majority of women with diastolic hypertension also smoked cigarettes or had hyperlipidaemia. Thus hypertension should not be regarded as the only risk factor contributing to the early development of myocardial infarction.

At present there are no alternative data from which to deduce the prevalence of diastolic hypertension in young women with ischaemic heart disease, and 28-40% (mean 34%) is as near as we can get.

Finally, it was certainly no surprise to me to find that coexisting hypertension worsened the prognosis in each group. This correspondence may help to emphasize the need for assiduous control of diastolic hypertension in young women.—I am, etc.,

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Tuberculosis of the Spine

SIR,—In your leading article under the above heading (14 December, p. 613) you refer to our work in Nigeria and state that we treated our ambulant patients with "chemotherapy alone, using neither rest, nor splintage, nor operations."

In fact our paper[1] reporting the management of patients suffering from spinal tuberculosis certainly mentioned surgery, albeit of a simple nature. Perispinal abscesses were drained when they were associated with paraplegia and the patient was unable to walk (that is, could not yet undergo ambulant treatment). Believing that the paraplegia is primarily caused by the pressure of the abscess on the cord we contented ourselves with the removal of the inner end of a rib or ribs and emptying the abscess under direct vision (costectomy). Twenty-seven out of a total of 207 patients were

treated in this way. Surface abscesses were also drained when aspiration failed to clear them.

We would like to emphasize these points so as to spare any disappointment to our colleagues in the tropics when they are faced, as they will be, with such situations. —We are, etc.,

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ARY BLESOVSKY

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Shotley Bridge, Co. Durham

1 Konstam, P. G., and Blesovsky, A., *British Journal of Surgery*, 1962, **50**, 26.

SIR,—Your leading article on this subject (4 December, p. 613) was no doubt justified by the recent reports you quoted. There was, however, one outstanding flaw in your review which was to my mind almost criminally negligent. You did not draw attention to the fact that it was in the cases treated in an ambulant manner that paraplegia developed during treatment. I feel that you should draw attention to this and that any one treating tuberculosis of the spine in the ambulant manner for the first time should confine his experiment to patients with the disease located below the termination of the dura and particularly not in the dangerous vertebrae, dorsal 4-8.—I am, etc.,

RANDLE LUNT

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** Mr. Lunt has done well to draw attention to spinal cord involvement in tuberculosis of the spine. Because of limitation of space the leading article to which he refers dealt almost solely with healing of the vertebral disease. However, Mr. Lunt, as a consultant in orthopaedic tuberculosis, will certainly have studied the four reports referred to, even though he has drawn conclusions diametrically opposed to the evidence that they showed.

Of 268 followed-up patients treated as ambulant outpatients, three who had been free from central nervous signs on admission to the trials developed paraparesis. None became unable to walk. Two recovered completely on ambulant chemotherapy alone and the third after an operation which hindsight suggests might have been unnecessary. Mr. Lunt will recollect that the leading article did not advise ambulant chemotherapy as the sole method of treatment in circumstances in which radical surgery is safe. When he has demonstrated, not merely implied or asserted, that any other method of treatment produces a lower incidence of paraplegia his charge of "almost criminal" negligence in recommending ambulant treatment in places where surgical facilities are scarce will call for an answer. On the evidence of the Medical Research Council's reports it does not do so at present.—ED., *B.M.J.*

Cyclophosphamide in Treatment of Nephrotic Syndrome

SIR,—We would like to comment on the excellent article by Professor J. S. Cameron and others (5 October, p. 7). They reported sustained remission after cyclophosphamide treatment in 20 out of 58 children with corticosteroid-sensitive but relapsing nephrotic syndrome and "minimal-change" lesions on renal biopsy.

We recently reported[1] the results of short-term cyclophosphamide therapy in 13 adults and four children presenting with the nephrotic syndrome or persistent proteinuria, normal renal excretory function, and either mild but definite glomerular lesions (14 patients) or no glomerular abnormalities on light microscopy (3 patients). Sustained remission of proteinuria for 5-22 months (mean 12·7 months) was obtained in 11 of the 17 patients and clinical improvement seen in four other patients. The onset of remission was usually prompt (within three months of starting treatment in nine patients). The response was independent of the duration of disease and histological diagnosis—60-75% of patients with focal proliferative glomerulonephritis, diffuse proliferative glomerulonephritis, and mesangiocapillary glomerulonephritis obtaining complete remission. Two patients conceived normal children after cyclophosphamide treatment. In only one patient, aged 14, was amenorrhoea seen seven months after therapy.

We suggest that cyclophosphamide given for a short period has a definite role in patients with glomerular lesions other than minimal change and that short-term therapy may avoid the reported toxicity to gonadal function.—We are, etc.,

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1 Ibels, L. S., *et al.*, in *Drugs and the Kidney*, ed. K. D. G. Edwards. *Progress in Biochemical Pharmacology*, vol. 9, Basal, Karger, 1974.

Fish Tank Granuloma

SIR,—A woman aged 46 cut her hand cleaning a fish tank. After 12 months a small unhealed lesion remained over the head of the fourth metacarpal. No foreign body was found on exploration. Some weeks later the back of her hand and wrist became swollen. At operation a chronic thickened synovium of the extensor tendon was excised. No bacteriological examination was made, but sections of a substantial portion of the thickened synovial tissue showed a granulomatous reaction with small numbers of giant cells but no caseation or necrosis. No acid-fast bacilli were found. Urine, E.S.R., and chest radiograph were all normal. After the biopsy a comparative skin test was carried out using five units of tuberculin (P.P.D.) and an equivalent dose of *Mycobacterium marinum* antigen. Induration 20 mm in diameter resulted from the latter but less than 6 mm from the P.P.D.

Myco. marinum lives in water and is an opportunist pathogen of aquatic animals, including man in the role of swimmer or keeper of pet fish. Risks from it in the latter hobby have been noted.[1,2] The organism grows very poorly at 37°C, and presumably for this reason human infections are always cutaneous. They present in two main ways: (1) as indolent localized but sometimes extensive lesions of the skin, first papular and then nodular, which may become verrucose or may develop pustules or liquefying granulomata; (2) a sporotrichoid-like infection initially seen at the site of an injury but followed by the lymphatic spread of skin lesions, which may be nodules or abscesses and from the hand may reach to the axilla.

The present case is of interest because a rather trivial lesion was activated by surgical exploration and the extension of inflammation resulted in tenosynovitis, a complication which appears not to have been previously recorded and which represents a deeper incursion into the tissues than is usual. Histology of the synovium suggested the possibility of a *Myco. marinum* infection and prompted the skin test, which strongly supported the hypothesis. Unfortunately the connexion of indolent skin lesions with aquaria is still not generally known. An initial bacteriological examination and skin test in this case would have led to effective treatment and saved two operations. Perhaps recognition would be helpful by encouraging the memorable name "fish tank granuloma," which has been suggested for the condition. —We are, etc.,

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1 *British Medical Journal*, 1970, **2**, 438.
2 Barrow, G. I., and Hewitt, M., *British Medical Journal*, 1971, **2**, 505.

Complication of Austin Moore Prosthesis

SIR,—I read with interest the letter of Mr. O. N. Vyas and Mr. A. K. Varshneya (14 December, p. 658), and I am surprised to hear that insertion of the Austin Moore prosthesis is performed in some centres under *x*-ray control. Surely this is unnecessary as this procedure is virtually performed under direct vision.

This point, therefore, leads to a comment on the purpose of their letter—namely, the insertion of a probe into the femur. If the femoral shaft is carefully reamed out the alignment of the reamer is readily checked and thus will ensure that it passes along the line of the femur. Similarly, the shaft of the femur below the trochanter may be palpated while reaming and when the prosthesis is being inserted. The problem of lateral protrusion of the stem is thus easily avoided. I therefore feel that this additional step seems unnecessary in an operation that has been performed on countless occasions in different parts of the world.—I am, etc.,

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Mithramycin for Hypercalcaemia Associated with Myeloma and other Malignancies

SIR,—In his review of myelomatosis Dr. J. S. Malpas (30 November, p. 520) recommends that neutral phosphate be used to treat associated hypercalcaemia when dietary restriction, fluids, and prednisone prove ineffective. Oral phosphate is, however, impractical if the hypercalcaemic patient is nauseated or vomiting and intravenous phosphate infusion carries the risk of ectopic calcification, which may prove fatal.[1] We