unto the off-chance that their organs might one day be suitable for transplantation.

It would have been more appropriate if the subcommittee had directed attention to the main ethical dilemma of organ transplantation—for the fact that only some 10% of the 2,000-3,000 young people needing treatment for terminal renal disease are being helped, the rest being allowed to die, despite the established fact that many of them would be provided with worthwhile therapy if more donor kidneys were available.

A change in the law with provision for "contracting out" is the only way in which there can be a solution to the shortage of donor organs. This was accepted by the majority of the MacLennan committee and legislation along these lines has already been adopted in Denmark, France, Israel, Italy, and Sweden.—I am, etc.,

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Chemotherapy of Bronchitis

Sir,—In your leading article on the chemotherapy of bronchitis (17 January, p. 125), a combination of fucidin and cloxacillin is suggested as an orthodox treatment for staphyloccocal pneumonia. However, you then observe that these antibiotics may be antagonistic under in vitro conditions. You therefore recommend a combination of fucidin with erythromycin for staphyloccocal pneumonia. Not only is there no possibility of antagonism, but erythromycin is more effective than cloxacillin against penicillin-resistant staphylococci, which may also be present.

Antagonism between fucidin and the penicillins, demonstrated by Erikson, is found when staphyloccoci are incubated for 24 hours in the presence of both antibiotics. Because of the inhibitory effect of fucidin, the staphyloccoci do not multiply and cannot therefore be destroyed by the otherwise bactericidal action of penicillin. However, this type of observation cannot be applied directly to conditions in vivo. It is also important to show if antagonism can be demonstrated after more than 24 hours, as a period of treatment of only 24 hours has no relevance to clinical practice. An investigation done in collaboration with K. A. Jensen (unpublished observations) showed that while antagonism can be observed after 48 hours' incubation of the staphylococci in the presence of the two substances, after 72 hours only very few staphyloccoci survived the combined effect. After five days, no antagonism can be shown. This corresponds most closely with the conditions in vivo where treatment is normally continued for at least a week. On this basis we have treated 86 cases of staphyloccoccal pneumonia with a combination of fucidin and methicillin (72 cases) or penicillin G, as part of a larger series of 270 cases of serious staphyloccoccal infection reported elsewhere.1 Of our 86 patients, 40 (47%) were aged over 60, and 64 (74%) had severe debilitating disease. The total mortality rate in the group was 40%, but in only 18 patients (21%) was the staphyloccoccal infection either the primary or contributory cause of death. In 10 of these 18 patients death occurred within the first 24 hours of therapy. It should be emphasized that in 22 patients without predisposing disorders there were no deaths.

These results may be compared with a series in which similar categories of patients were treated with a penicillinase-resistant penicillin alone (amoxaplan and Lin.2) treated 15 patients suffering from staphyloccoccal pneumonia with nafcillin or cloxacillin, with a mortality of 47%. Klein and Finland3 demonstrated that cutaneous staphylococcal infections can be treated with cloxacillin (mortality 46%), 27 patients were treated with oxacillin with a mortality rate of 44%, and 24 patients were treated with diphencillin with a mortality of 67%. Martin et al.3 treated 23 patients with penicillinase-stable penicillins, observing that 61% of the deaths were due directly to staphyloccocal infection. A treated with what is probably the most validated published results therefore suggests that the combination of fucidin and methicillin in the treatment of staphyloccocal pneumonia is at least as effective as the penicillinase-stable penicillin alone and may well be more effective. While our experience is limited to methicillin, and not cloxacillin, both drugs have similar bacteriological characteristics, and it is difficult therefore to concede a true clinical advantage of the combination used in vitro against the two drugs. In vitro antagonism, as measured by current routine techniques, may indeed have little relevance to what actually happens in practice.—I am, etc.,

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REFERENCES

Haemolytic Anaemia in Pregnancy in Nigerians

Sir,—I read with interest the article by Drs. A. F. Fleming and N. C. Allan (22 November, 1969, p. 461) on "Severe Haemolytic Anaemia in Pregnancy in Nigerians Treated with Prednisolone." The conspicuous absence of a reference to glucose-6-phosphate dehydrogenase deficiency (and, incidentally, to serum iron levels) needs a little comment.

Edington and Gilles1 state: "G-6-P-D deficiency plays an important role in the pathogenesis of a variety of haemolytic anaemias in the tropics." Now in West Africa between 10 and 15% of the population are deficient with a partial or total deficiency of this enzyme. Urinary tract infection in pregnancy is as common in West Africa as in Europe. Moreover West African countries are medicalian to treat in the open 28 patients with drugs which elsewhere can be dispensed only in hospitals, at chemists, or in a pharmacy. Quacks also abound; therefore patients often come to hospital having already dosed themselves with chloramphenicol and/or nitrofurantoin—two drugs which appear in a recent list by Beutler2 and some common drugs which may induce haemolysis of G-6-P-D-deficient red cells. It is my clinical impression that in Accra the total defect of the female (homozygote) is more severe than that of the male (homozygote). The haemolysis of the female total defect seen here in Accra can sometimes be so catastrophic as to precipitate acute renal shut down.

Having myself seen in Korle Bu Hospital cases of persistent haemolytic anaemia with obscure hepato-splenomegaly in pregnancy, I hasten to concede that the type of "severe haemolytic anaemia in pregnancy in Nigerians" described by Dr. Fleming and Dr. Allan is quite unlike the anaemia due to G-6-P-D deficiency. Nevertheless this hereditary quantitative erythrocyte defect could have played a minor role in one or two of the cases described, and should have been excluded as just another qualitative haemoglobinopathy was excluded.

Incidentally, apart from the factors mentioned in the excellent discussion of Dr. Fleming and Dr. Allan's paper, one other thing to suspect in haemolytic anaemia of obscure origin during pregnancy in a non-sickler include Thalassaemia minor, Hb CC disease,2,3 and systemic lupus erythematosus,—I am, etc.,

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REFERENCES

Gongenyal Goitrous Cretinism due to Iodide

Sir,—The ingestion of iodides by the expectant mother is a documented cause of gongenital cretinism in the infant. Cases still occur despite recognition of the hazards of proprietary substances containing iodides.

A male infant weighing 5 lb 8 oz (2.5 kg) was admitted to the Stohill Hospital nursery when one day old, having been born at home after a spontaneous vertex delivery. He was transferred to hospital because of slight cyanosis and failure to maintain a satisfactory temperature. He was lethargic, cold, oedematous and had strikingly cretineous facies. Temperature 36.2°C (98°F), pulse 120 per min, and respiration 60 per min. Respirations were irregular and there was poor air entry to both lungs. The infant was cyanosed, with frothy sputum and the spleen tip was palpable.

Since the birth of her fifth child, six years previously, the mother had been taking, unknown to her general practitioner, a proprietary asthma mixture. The contents of this mixture were: caffeine B.P. 2.5 W/V; sodium iodide B.P. 2.5 W/V; sodium benzoate B.P. 6.9 W/V; ephedrine hydrochloride B.P. 0.5%, W/V; glycerin B.P. 20% W/V. The mixture was consuming 30 ml. of this mixture per day.

Investigations: Blood urea 68 mg./100 ml.; chlorides 94 mEq./L.; sodium 140 mEq./L.; bicarbonate 20 mEq./L.; potassium 60 mEq./L; cholesterol 75 mg./100 ml.; alkaline phosphatase 19 K.A. units; and
Convulsions
Age over 60 9 6
Pneumonia 4
Coma

Factors Related
Pyogenic Meningitis
Steroids in 81

Steroids and Acute Pyogenic Meningitis
Sir,—Your leading article (3 January, p. 6) on pyogenic meningitis and steroid treatment prompts us to make some comments.

We have investigated the results of steroid treatment in pneumococcal meningitis in 81 patients. The results are given in Table I. More patients in the steroid-treated group suffered from coma and pneumonia at admission. It is seen that among patients with bacteriemia the fatality rate was considerably higher in the control group. This corresponds to the experience gained from animal experiments, in which early treatment with hydrocortisone in pneumococcal bacteriemia revealed a favourable effect.² It is stressed that the steroid treatment in our material was initiated at an early stage of the infection. No side-effects were observed and the incidence of sequelae was similar in the two groups (Table II).

Though the difference in mortality between the two groups is not significantly different at the 5% level, our findings might suggest the effect of steroid treatment in pneumococcal meningitis.—We are, etc.,

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REFERENCES

Pharmacological Aid for Faecal Incontinence
SIR.—The difficulties in keeping continent patients clean are great. Children particularly live in constant fear of an “accident,” of the odour involved, of the shame, and of becoming a social outcast. We have not found the simple recommendations of the Spina Bifida Study Group¹ helpful in the treatment of our twenty incontinent children who wish to go to school, to social events, and to work. Enemata are not a good solution.²

We have had remarkable successes with the following method of bowel-training, which invokes a conditioned reflex. This can be reinforced by the gastro-colic reflex, by stimulation of intestinal motility with neo-stigmine, and also by suppositories which irritate the rectal mucosa. The programme must of course be adapted to the patient’s usual daily schedule, and the advantages of completing this duty early in the morning have to be weighed against the early hour which the two-hour programme must be commenced.

The patient is given a tablet of neostigmine 15 mg. (up to two tablets if one is inadequate) at a fixed hour, preferably before a meal, on two or three days a week depending on the frequency of the patient’s earlier bowel habits. One-and-a-half hours later, or soon after the meal, the patient is given two glycerin suppositories per rectum.³ If these cannot be retained, the larger glycerin ovule, probably for vaginal use, will usually remain in situ. Twenty minutes after insertion of the suppositories the patient is seated on the toilet for ten minutes. If there is a small result, or no result, at the end of this time, an enema is given immediately. The need for this has been found to diminish progressively. Rectal (digital) examination is performed after the bowel action to confirm adequate emptying, especially in the early stages of training. Complete emptying of the rectum is essential, and if necessary the enema is repeated immediately.

By frequent trial over the weeks or months, the neostigmine dose is gradually reduced, and finally eliminated. Suppositories are continued at the scheduled time. Our physiotherapists have helped with some patients by education of the abdominal muscles.

57 patients with this regimen has enabled us to discharge a resident patient for fourteen years from all care except three-monthly supervision. Now aged 23, she had been admitted for meningo-myletcele with complete incontinence, but when examined after two years for enema was eliminated that she became confident of being able to cope at home.

The failures so far have been in younger children, probably because they are too young to collaborate actively.—I am, etc.,

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Appendicitis in Umbilical Hernia Sac
SIR,—Although acutely inflamed appendices have been found in inguinal hernias¹ and even in femoral hernias,² I could find only one reference to an appendix being found in an umbilical hernia. From Russia, Bulgakov reported an acutely inflamed appendix in a strangulated umbilical hernia of a ten-month-old baby.³ The rarity of the finding is probably due to the fact that the appendix is seldom found in the proximity of the umbilicus. It would, therefore, appear worthwhile to report the occurrence of a chronically infiammed appendix in an umbilical hernia of an adult.

The patient, a 63-year-old man, was first seen for the out-patient department on 30 July 1969 complaining of a red and painful swelling in the umbilical region. He had first noticed this swelling three months previously. It had subsidec the patient reported several days later. His own doctor had treated him with tetracycline. He had never vomited, his bowel action was normal, and at no time had he lost his appetite. In the past he had had bilateral femoral