unters on 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—namely, 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 fusidin and cloxacin is suggested as an orthodox treatment for staphylococcal pneumonia. However, you then observe that these antibiotics may be antagonistic under in vitro conditions. You therefore recommend a combination of fusidin with erythromycin for staphylococcal pneumonia. Not only is there no possibility of antagonism, but erythromycin is more effective than cloxacin against pneumoccus, which may also be present.

Antagonism between fusidin and the penicillins, demonstrated by Erikson,1 is found when staphylococci are incubated for 24 hours in the presence of both antibiotics. Because of the inhibitory effect of fusidin, the staphylococci 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 An investigation done in collaboration with K. A. Jensen (unpublished observations) showed that while antagonism can be observed after 48 hours incubation of the staphylococci in the presence of the two substances, after 72 hours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 staphylococci survived the combined effect. After five days, no antagonism can be shown. This corresponds more 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 staphylococcal pneumonia and 23 cases of a combination of fusidin and methicillin (72 cases) or penicillin G, as part of a larger series of 270 cases of serious staphylococcal infection reported elsewhere.2 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 staphylococcal 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-stable penicillin alone and with fucidin.2 Luther and Martin treated 15 patients suffering from staphylococcal pneumonia with nafcillin or cloxacin, with a mortality of 47%. Klein and Finland3 treated 21 patients with lower respiratory tract infection caused by staphylococci, with a mortality of 46%; 27 patients were treated with oxacillin with a mortality rate of 44%; and 24 patients were treated with dihenicillin with a mortality of 67%. Martin et al.4 treated 23 patients with penicillinase-stable penicillins, observing that 61% of the deaths were due directly to staphylococcal infection.

A treated with penicillinase-stable penicillins, observing that 61% of the deaths were due directly to staphylococcal infection.

Having myself seen in Korle Bu Hospital cases of otherwise untreatable 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. Allen is quite unlike haemolysis 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 just as a qualitative haemoglobinopathy was excluded.

Incidentally, apart from the factors mentioned in the excellent discussion of Dr. Fleming and Dr. Allen, there are other things to suspect in haemolytic anaemia of obscure origin during pregnancy in a non-sickler include Thalassaemia minor, Hb CC disease,5 and systemic lupus erythematosus.—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. Allen (22 November, 1969, p. 461) on "Severe Haemolytic Anaemia in Pregnancy in Nigerians Treated with Penicillin." The conspicuous absence of a reference to glucose-6-phosphate dehydrogenase deficiency (and, incidentally, to serum iron levels) needs a little comment.

Edington and Gills1 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 have a partial or complete deficiency of this enzyme. Urinary tract infection in pregnancy is as common in West Africa as in Europe. Moreover West African countries are methylisouric to treat in the opening market 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 doses themselves with chloramphenicol and/or nitrofurantoin—two drugs which appear in a recent list by Beutler and colleagues which may induce haemolysis of G-6-P-D-deficient red cells. It is my clinical impression that here in Accra the total defect of the female (hemoglobin) is more severe than that of the male (hemizygote). The haemolysis of the female total defect seen here in Accra can sometimes be so catastrophic as to precipitate acute renal shut down.

Since the birth of her fifth child, six years previously, the mother had been taking, unknown to her, a proprietary asthma mixture. The contents of this mixture were: caffeine B.P. 2.8 W/V; sodium iodide 2.5 W/V; sodium benzoate B.P. 6-9 W/V; ephedrine hydrochloride B.P. 0-45%, W/V; glycine B.P. 20 W/V. The mixture was consuming 30 ml. of this mixture per day.

Investigations: Blood urea 68 mg./100 ml.; cholesterol 154 mg./100 ml.; potassium 6-0 mEq/l; cholesterol 75 mg./100 ml.; alkaline phosphatase 19 K.A. units; and

Gongenital Goitrous Cretinism due to Iodide

Sir,—The ingestion of iodides by the expectant mother is accompanied 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 Stobhill 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 cretinous facies. Temperature 99°F (33°C), pulse 132, respiration 49, with no enlarged. Respirations were irregular and there was poor air entry to both lungs. The child had no fingerbreadth and the spleen tip was palpable.

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