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 fucidin and cloxacillin 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 fucidin with erythromycin for staphylococcal pneumonia. Not only is there no possibility of antagonism, but erythromycin is no more effective than cloxacillin against pneumococci, which may also be present.

Antagonism between fucidin 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 fucidin, 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 An investigation done in collaboration with K. A. Jensen (unpublished observations) showed that while antagonism can be observed after 48 hours' incubation of the clostric in the presence of the two substances, after 72 hours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 with Erikson's finding that 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 with a combination of fucidin 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 treatment. 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. Gravant and Lentz3 treated 15 patients suffering from staphylococcal pneumonia with nafcillin or cloxacillin, with a mortality of 47%. Klein and Finland4 treated 14 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 cephalothin with a mortality of 67%. Martin et al.5 treated 23 patients with penicillinase-resistant penicillins, observing that 61% of the deaths were due directly to staphylococcal infection.

A treated with the proposal in vitro antagonism between 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 Pendrinolone." 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 is known to be deficient in this enzyme. Urinary tract infection in pregnancy is as common in West Africa as in Europe. Moreover West African countries are menacising to treat in the open 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 dosed themselves with chloramphenicol and/or nitrofurantoin—two drugs which appear in a recent list by both. So some common drugs 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 (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 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 as just another haematological quagmire was excluded. Incidentally, apart from the factors mentioned in the excellent discussion of Dr. Fleming and Dr. Allan's cases, there are other things to suspect in haemolytic anaemia of obscure origin during pregnancy in a non-sickler include Thalassaemia minor, Hb CC disease,6,7 and systemic lupus erythematosus,—I am, etc.,

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REFERENCES

Gonadal Goitre in Cretinism due to Iodide

Sir,—The ingestion of iodides by the pregnant mother is the inadvertent cause of gonadal cretinism in the infant. Cases still occur despite recognition of the hazards of proprietary substances containing iodides.

A male infant weighing 5 lb. 8 0z. (2.5 kg.) was admitted to the Stobhill Hospital nursery on 16 September 2023 by guest. Protected by copyright.http://www.bmj.com/ Br Med J; first published as 10.1136/bmj.2.5701.112 on 11 April 1970. Downloaded from http://www.bmj.com/ on 16 September 2023 by guest. Protected by copyright.