common in the enterobacteria of the infant population of Dublin.

Dr. Anderson suggests that the R factors in the Tees-side strains had probably been transferred from non-pathogenic R⁺ *E. coli*. Our findings support his view. One possible method of preventing the spread of R factors within a hospital is to isolate all infants excreting R⁺ enterobacteria on admission. As it is a routine practice in most children's hospitals to culture the faeces of infants on admission for pathogenic enterobacteria, I suggest that a direct sensitivity test should also be done on the faeces. All infants carrying R⁺ strains should be isolated within the hospital. It would not be possible or necessary to screen for R factors, because it is most probable that if the organism is multi-resistant it is R⁺.—I am, etc.,

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REFERENCE

- ¹ Moorhouse, E. C., *Irish J. med. Sci.*, 1966, 375.

Misuse of Antibiotics

SIR,—In the last paragraph of your leading article on "Transferable Antibiotic Resistance" (3 February, p. 263) you raise the question of whether or not antibiotics should be prescribed for mild respiratory infection in view of their possible effect on intestinal flora. The same question may be asked with regard to mild sporadic gastrointestinal upsets in children.

For a period of about a year between 1965 and 1966 I was in charge of a military families' clinic in Malaya. Diarrhoea was a common cause of attendance and stool specimens were obtained from all but a small minority of patients. In one child a pathogenic *E. coli* was isolated, and in one woman a clinical diagnosis of dysentery was confirmed. In all the other cases recovery followed dietary restriction with the prescription of kaolin or codeine phosphate, and no bacterial pathogens were isolated.

One wonders how often antibiotics are prescribed "for safety." It has certainly been my impression that the admission of a child to hospital sometimes appears to be precipitated by vomiting following the administration of (usually) sulphonamides. These same children get well as a rule without further antibiotics, and bacterial pathogens are not isolated.

It would be of interest and value to know how frequently antibiotics are prescribed for diarrhoea and vomiting in England, and whether recovery is in fact speeded by their use. Perhaps this would be a suitable subject for the Royal College of General Practitioners to investigate.—I am, etc.,

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J. CARSON.

Fatal Embolus During "Pill" Administration

SIR,—I wish to record a case of sudden death in a young woman of 23 who had been taking an oral contraceptive containing mestranol and ethynodiol diacetate for three months. I might add that this was not prescribed by me.

Post-mortem examination revealed a large pulmonary embolus, and loosely adherent antemortem thrombus was found in both iliac veins and their tributaries. There was no other lesion present which might have predisposed to thrombosis.

The facts have been reported to the Dunlop Committee.—I am, etc.,

Cleethorpes, Lincs.

R. G. ALLEN.

Degos's Syndrome

SIR,—Mr. R. E. May's well-documented medical memorandum (20 January, p. 161) describes a case under the title "Degos's Syndrome" (malignant atrophic papulosis). The photographs illustrating the case, and the description of the skin lesions, do not substantiate a diagnosis of this rare syndrome. Degos,¹ in a private communication, maintains that the cutaneous lesions are so characteristic that the diagnosis is easily made from a photograph. Case reports such as those of Nomland and Layton,² Naylor *et al.*,³

and Winkelmann *et al.*⁴ are illustrated with photographs which show typical lesions of malignant atrophic papulosis. These are well shown in the accompanying photograph of the first reported British case.⁵

The early lesion is a pale rose papule of 2-5 mm. diameter which soon becomes umbilicated and porcelain-white in colour, though some lesions may show a yellowish scaly centre; a striking feature is the sharply defined and elevated enclosing pink halo. Ulceration of the skin is rare. The eruption occurs in crops at intervals of days or weeks and the development of a characteristic lesion with a porcelain-white centre may occur over a period of 7-10 days. The black centre and tenderness of the lesions in the case described would make most dermatologists query the diagnosis; also, in Degos's syndrome the patient is almost invariably asymptomatic in the early cutaneous phase of the disease, which may precede the onset of abdominal symptoms by months and sometimes years.

A diagnosis of Degos's syndrome cannot be upheld on the basis of a histopathology

which shows a non-specific vasculitis; in our present state of knowledge probably "allergic vasculitis" might serve as a convenient label for this most interesting case.—I am, etc.,

Hove, Sussex.

PATRICK HALL-SMITH.

REFERENCES

- ¹ Degos, R., personal communication, 1964.
- ² Nomland, R., and Layton, J. M., *Arch. Derm.*, 1960, 81, 181.
- ³ Naylor, D., Mullins, J. F., and Gilmore, J. F., *Arch. Derm.*, 1960, 81, 189.
- ⁴ Winkelmann, R. K., Howard, F. M., jun., Perry, H. O., and Miller, R. H., *Arch. Derm.*, 1963, 87, 54.
- ⁵ Hall-Smith, S. P., *Proc. roy. Soc. Med.*, 1964, 57, 519.

Budd-Chiari Syndrome after Oral Contraceptives

SIR,—At the time of the report by Drs. K. Sterup and J. Mosbech (16 December, p. 660) we were treating a very similar patient who had been taking the same oral contraceptive, lynestrenol 5 mg. and mestranol 0.15 mg. (Lyndiol) for six months, and for the previous six months another oral contraceptive, Gynovlar 21 (norethisterone acetate ethinyloestradiol).

A 37-year-old housewife with two children had been perfectly well until a few weeks before admission on 3 December 1967, when she had developed a slight sensation of distension in the upper abdomen. About 24 hours before admission she had vague epigastric pain, which became severe within a few hours, radiating to the left scapular region and right hypochondrium and associated with vomiting of bile and a little altered blood. The pain continued and was accompanied by the passage of several greenish loose stools.

On admission the patient was shocked and dehydrated, with a pulse rate of 134/min. and a systolic blood pressure of 70 mm. Hg, the diastolic pressure being unobtainable. There was possibly slight icterus, and the urine contained a trace of bile and albumin. There was marked tenderness in the epigastrium and right hypochondrium, and a smooth mass could be palpated in the epigastrium. There was on admission no evidence of ascites, and bowel sounds were normal. The possibilities of a posterior perforation of peptic ulcer into the lesser sac or acute pancreatitis were suspected. The dehydration and shock were treated with intravenous fluids, and repeated gastric aspiration was performed. The serum amylase was normal (160 units/100 ml.), and the plasma electrolytes were normal except for a raised potassium of 5.2 mEq/l. and an alkali reserve of 12 mEq/l. The blood urea was 47 mg./100 ml., the haemoglobin 16.5 g./100 ml., P.C.V. 57.5%, and white count 23,000/cu. mm. with a polymorphonuclear leucocytosis. After intravenous fluids and ampicillin the patient's general condition improved over the next 36 hours, though her daily output of urine was never more than 100 ml. Two days after admission she deteriorated again, with hypotension and drowsiness, but no fetor hepaticus or liver flap. The blood urea had risen to 120 mg./100 ml. By this time she had developed a large ascites and was slightly icteric. The liver-function tests were reported subsequently as bilirubin 2.5 mg./100 ml., S.G.O.T. 2,000 R.F. units/ml., and alkaline phosphatase 18 K.A. units/100 ml. With the rapid development of ascites a diagnosis of Budd-Chiari syndrome was made, but, as the possibility of generalized peritonitis could not be excluded, a laparotomy was performed which revealed a large amount of clear ascitic fluid, a grossly enlarged, engorged purple liver, and a moderately enlarged spleen. No other organs were obviously diseased. A wedge liver biopsy was taken, which showed necrosis of most