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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Royal College of Surgeons,
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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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Aldershot, Hants.

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, lynoestrenol 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

of the liver cells except for those around the portal tracts, which appeared normal. The centrilobular zones were very haemorrhagic, the mid-zonal sinusoids were distended with blood, but the surviving outer zones were not congested.

Her oliguric renal failure continued and was treated with intracaval 20% mannitol at first and 40% dextrose. She continued to deteriorate, however, and became comatose. Despite peritoneal dialysis for her renal failure, supervised by Dr. H. M. Leather, she did not improve and died on 13 December 1967.

At necropsy, by Dr. M. R. Thomas, a smooth mottled thrombus was found projecting from the orifice of the hepatic vein and partially obstructing its lumen. Dissection into the main tributaries showed dark-red adherent thrombus occluding the veins. The liver weighed 2,600 g., and the left lobe was relatively enlarged compared with the right. The outer aspect was icteric and cyanosed, and the cut surface was similar to a "nutmeg liver," except that the red zones were brighter, larger, and more sharply demarcated than those seen in cardiac failure. The liver histology confirmed the microscopic appearances found in the biopsy specimen, and an occasional cluster of hepatic cells were undergoing calcification. An antemortem thrombus of a flat strap-like appearance was found in the inferior vena cava and right common iliac and femoral veins extending up to about 2 in. (5 cm.) below the thrombus in the hepatic vein from the site of insertion of the inferior caval catheter. There was oedema and congestion of the lungs, with a 6-cm. red infarct in the right lower lobe due to an embolus. The lower oesophagus, stomach, and upper jejunum were congested with a few small subserosal haemorrhages. The kidneys appeared normal, but histology showed moderate cloudy swelling of the tubular epithelium and some calcified concretions within the tubular lumen. The spleen was dark and firm, weighing 325 g.

As five previous cases of Budd-Chiari syndrome in young women taking oral contraceptives have been reported by Ecker *et al.*,¹ Drs. K. Sterup and J. Mosbech (16 December, p. 660), Dr. B. N. Somayaji and others (6 January, p. 53), Mr. R. L. Rothwell-Jackson, and Drs. A. W. Clubb and C. Giles (27 January, p. 252), we feel that the association may not be entirely fortuitous.—We are, etc.,

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M. C. T. REILLY.

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Plymouth, Devon.

REFERENCE

- ¹ Ecker, J. A., McKittrick, J. E., and Failing, R. M., *Amer. J. Gastroent.*, 1966, 45, 429.

Gallstone Disease

SIR,—Professor A. J. Harding Rains (3 February, p. 295) states in his interesting article, that "Foreign bodies also act as nuclei for stones." This brought to mind a patient who passed through my hands about 10 years ago.

She was a white woman of 33 (parity unknown) who was referred for an oral cholecystogram by her family doctor, who suspected that she was suffering from "gallstone disease." The resultant radiographs were such that a confident diagnosis of the presence of a foreign body (a pin) surrounded by cholesterol stones in the gall bladder could be made. The gall bladder itself

concentrated the contrast medium satisfactorily and contracted well after the stimulus of a fatty meal.

Subsequently the patient admitted that about five years previously she had swallowed a few pins deliberately. At operation several cholesterol gallstones were found, one of them encasing the pin. There were no adhesions between the biliary and alimentary tracts, and how the pin passed from one to the other must be conjectural.—I am, etc.,

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Colchester.

M. J. HARMAN.

Volvulus of Sigmoid Colon

SIR,—As a recent colleague of Mr. J. J. Shepherd I would congratulate him on his report of 425 cases of sigmoid volvulus in Africans (3 February, p. 280), but I must question him over his advice of conservative management by intubation for the non-gangrenous type of volvulus seen in Uganda. My own experience, gained over 21 months at Mulago Hospital (and Mr. Shepherd's paper), leads me to recommend primary resection as the treatment of choice.

The comparison of 89 patients treated conservatively without a death with 65 patients treated by resection with 16 deaths (25%) must be qualified, as patients exhibiting signs of shock and peritonitis will be preferentially subjected to laparotomy in the belief that gangrene exists, whereas those more healthy patients without such ominous signs will be more readily chosen for intubation. The highest mortality of all (33%) relates to three deaths in nine patients with viable bowel who were subjected to laparotomy after the failure of intubation.

There were eight deaths (16%) in 49 patients subjected to laparotomy with untwisting of viable bowel. These deaths should not be due to untwisting, as death does not follow intubation alone. In Uganda cardiopulmonary disease is very rare in surgical patients; hence the presumptive cause of death must be inappropriate surgery and anaesthesia in the face of dehydration and electrolyte imbalance resulting from the intestinal obstruction and any hookworm anaemia (which is common).

Primary resection in 35 patients with viable bowel had a lower mortality (14%) than mere untwisting.

The known recurrence rate of 18 out of 43 patients who failed to have further treatment following immediate intubation was 42%. If a similar overall mortality of 50:425 appertains to this group then four deaths could be expected. To these may be added the two deaths following anaesthesia for elective resection in 74 patients, to give a mortality of six (5%) out of 117 patients treated by immediate intubation alone in their first attack. My memory of discussions with Mr. Shepherd reminds me that the mortality in the second or subsequent attack of volvulus is higher than that of the first attack. Furthermore, the true recurrence rate must be higher than 42%.

The choice offered lies between immediate intubation followed by elective resection (with its inevitable opportunity for the patient to refuse operation or default from readmission for resection, and with a minimum mortality rate of 5% plus the dire consequences of laparotomy when intubation fails) and primary resection of patients with viable bowel, which carries a 14% mortality; in neither instance is the mortality due to the sigmoid resection. Mr. Shepherd states that the pelvic colon is thick-walled and vascular, and it is of interest that this holds good for the bowel distal to the twist. From my own experience I can add that the distension is almost totally gaseous; in fact, a more perfectly prepared bowel would be difficult to find. It therefore seems that primary resection is both theoretically and practically acceptable; the error rests in the selection and preparation of patients for resection. Surely the advice needed is to improve the surgery of primary resection and not to retreat to a policy of dubious value for the wrong reasons.—I am, etc.,

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Bristol.

SIR,—Mr. J. J. Shepherd's paper (3 February, p. 280) provides important confirmation of the value of conservative treatment of sigmoid volvulus by Bruusgaard's technique of sigmoidoscopy and intubation. It seems possible that an important factor in the success of this technique is the fact that the patient is usually placed in the knee-elbow position for sigmoidoscopy. Indeed, two of Mr. Shepherd's patients spontaneously passed gas when placed in this position prior to sigmoidoscopy.

Consideration of the anatomical and physical factors involved will indicate why this should be so (see Figure). In a colonic volvulus gas accumulates in the convexity of the distended loop, which rises upwards. The site of the torsion is fixed by the mesenteric attachment to the posterior abdominal wall. If the patient is supine, or even upright, the site of the torsion will usually be the lowest point. Any fluid in the loop will therefore collect at this site and form a seal which will