depletion is not convincingly supported by their data. The incidence of hypersegmented neutrophils in the blood can hardly be used as a criterion of folic-acid deficiency, since many of their patients with hypersegmented neutrophils had normoblastic marrows, and, conversely, only 10 of 15 cases with megaloblastosis showed an excess of hypersegmented neutrophils in the peripheral blood.

If iron deficiency did initiate or exacerbate folic-acid deficiency one would expect fewer and less severe cases of megaloblastic anaemia in pregnant women who have received prophylactic iron. In our experience, which extends over the past nine years, this is not the case. On the contrary, the most florid megaloblastic marrow picture is seen in women who have taken regular supplements of iron in pregnancy. Several of our patients who had received no iron prophylaxis up to about the 30th week of pregnancy were found to have an equivocal or frankly normoblastic marrow, but after two to three weeks' iron therapy marrow aspiration yielded a florid megaloblastic marrow.

It is not really surprising that many pregnant women should be both iron and folic-acid deficient when one inquires into the diet of these patients. There is also evidence that a state of malabsorption, which is probably reversible, develops in many patients in late pregnancy. This is reflected in the relatively high faecal fat output of women with megaloblastic anaemia in pregnancy.¹ Megaloblastic anaemia in pregnancy, therefore, resembles intestinal malabsorption rather than the post-gastrectomy syndrome.

Finally, we do not share Dr. Chanarin's disappointment with the diagnostic value of the serum-folate assay. Four years' experience of this test has taught us that, once the technique is mastered, folate assay can detect at least 75% of cases of megaloblastic anaemia in pregnant women. If the labile folate factor is estimated² a 96% correlation can be obtained with marrow findings in pregnant women.—We are, etc.,

C. GILES.
E. W. BALL.

Stoke-on-Trent.

REFERENCES

- ¹ Giles, C., and Burton, H., *Brit. med. J.*, 1960, 2, 636.
- ² Ball, E. W., and Giles, C., *J. clin. Path.*, 1964, 17, 165.

Reactions to Intravenous Iron Dextran

SIR,—Total dose intravenous infusion of iron dextran (Imferon) may be followed by a number of reactions varying from local phlebitis to the severe complications of shock, dyspnoea, or cyanosis described by Dr. Barbara Clay and her colleagues (2 January, p. 29). I have recently treated four patients by this method and two of them developed thrombosis of veins remote from the site of infusion.

The circumstances in the two cases were very similar. Both were African females, aged 39 and 47 years, and both presented with iron-deficiency anaemia caused by menorrhagia from fibroid uteri. The haemoglobin levels were about 9 g./100 ml., and iron-deficiency was evidenced by hypochromia of the red cells, low serum-iron values, and the absence of stainable iron on marrow biopsy. The patient aged 47 had varicose veins of the right calf. These were mild, had been present for "years," and had

never troubled her. The history and physical examination were otherwise negative in the two cases. Both were ambulant up to the time of the infusion, and neither had received parenteral iron previously.

One patient was given 35 ml. of iron dextran dissolved in 1 litre of normal saline at the rate of 40 drops per minute, and the other 40 ml. in 1 litre of saline at the rate of 15 drops per minute. In both cases a superficial arm vein was used for the infusion, during the course of which no local or systemic reactions were observed. The infusion was given during the afternoon and evening, and the patients were discharged the following morning.

Both patients presented with pain and swelling of one leg two days after the infusion. On examining the affected limb the calf was found to be warm and tender, and both the calf and foot were considerably oedematous. In the 47-year-old patient the deep-vein thrombosis involved the right leg. The condition was treated in the usual way and both cases responded satisfactorily.

I have no proof that the infusion of iron dextran caused the venous thrombosis in these patients, but the circumstances suggest that the association is more than coincidence. Its mechanism is obscure, but in one of the patients it is possible that the pre-existing venous abnormality acted as a predisposing factor. Perhaps some of your readers have encountered similar cases.—I am, etc.,

Non-European Hospital,
Johannesburg, S. Africa.

H. C. SEFTEL.

Management of Acute Iron Poisoning

SIR,—With reference to the commentary on desferrioxamine B mesylate in "To-day's Drugs" (6 February, p. 366), we should like to make some further observations on the management of acute iron poisoning.

During the third phase evidence of hepatic insufficiency tends to appear. This has been well documented in the past,^{1,2} and we have recent experience of one such child who died in liver failure and at necropsy showed massive hepatic necrosis. It would seem that in this context great caution should be applied in the use of the sedatives phenobarbitone, paraldehyde, and chlorpromazine which you mention.

There may be potential danger in advocating very large doses of chelating agent by mouth (e.g., 5–10 g. is suggested). Where the mucosa is damaged can the absorption of soluble iron chelate in fact increase the overall accumulation of iron in the body? In severe poisoning the patient may become oliguric,³ and our fatal case passed only 20 ml. of urine. In this circumstance the question of possible tissue toxicity from the ferrioxamine complex itself arises. Where excretion is minimal, chelation should perhaps be combined with other means of removal, such as exchange transfusion.

In addition to the fatal case mentioned above we have three further patients who have been followed with respect to serum and urinary iron, and an account of these is in preparation for publication. In the case of a girl aged 2½ years who was mildly affected as judged by her clinical state and an initial serum iron of 500 µg./100 ml., uninterrupted recovery followed the use of a considerably reduced chelating regimen. We should agree, therefore, with the comment stressing the tentative nature of any dosage schedule for desferrioxamine at present, and the need

for further well-documented case reports.—We are, etc.,

Royal Hospital for Sick Children,
D. G. D. BARR.
D. K. B. FRASER.
Western General Hospital,
Edinburgh.

REFERENCES

- ¹ Aldrich, R. A., in *Iron in Clinical Medicine*, edited by R. O. Wallerstein and S. R. Mettler, University of Southern California Press, Berkeley.
- ² Luongo, M. A., and Bjornson, S. S., *New Engl. J. Med.*, 1954, 251, 995.
- ³ Dugdale, A. E., and Powell, L. W., *Med. J. Aust.*, 1964, 2, 990.
- ⁴ Gokulanathan, K. S., Baird, R. L., Reichelderfer, T. E., and Rubin, M., *Georgetown med. Bull.*, 1963, 16, 238.

Disinfectants in Hospitals

SIR,—I refer to the article "Disinfectants in Hospitals" (13 February, p. 408) and to statements about ampholytic surface-active agents.

It is stated that the compounds are not sporicidal. We have ample independent evidence to show that these compounds will kill the spores of *Bacillus globigii*, *B. anthracis*, *Clostridium sporogenes*, and *Cl. welchii* at room temperature with a contact time of 30 minutes. It is also stated that the compounds are expensive. On a price-per-gallon basis this may appear to be so at first sight, but the cost of disinfecting 1 sq. yd. (0.28 sq. metre) of surface is 0.06 pence. In fact, the rational use of these compounds has turned out to be less expensive than the indiscriminate and sometimes unnecessary use of compounds the price per gallon of which may be somewhat less.—I am, etc.,

COLIN HARRIS.

Tego Division,
Hough, Hosason & Company Ltd.,
Manchester 19.

SIR,—The article entitled "Disinfectants in Hospitals" (13 February, p. 408) gave much useful guidance on the use of disinfectants in hospitals. However, the difficulties in choosing effective disinfectants within their class still remain.

The general disinfectant in use in this hospital was stated by the suppliers to be such that "bactericidal activity" was "maintained in the presence of blood and other organic matter." It was on this written claim that the preparation was retained. However, when the suppliers were asked for definite bacteriological data to support their claim they were unable to provide it immediately. It was only after a delay of two months, and after a further letter to them, that they were able to supply the required details. Their letter revealed a Chick Martin coefficient of 0.2 and repudiated their former claim. This clearly shows that this product is unsuitable for the purpose for which we require it, and we are discontinuing its use.

It may be that other hospitals are also using disinfectants which are equally unsuitable and for which the suppliers make equally unjustified claims.—I am, etc.,

Battersea General Hospital, D. P. MORGAN.
London S.W.11.

Medical Survey of Tristan da Cunha

SIR,—In 1963 we reported the results of a medical survey of the entire population