placed intramuscular injections injuring one or other of these nerves. There are probably very many cases that are not reported in the literature, since this is now a recognized hazard of injection at these sites.

The lateral aspect of the thighs has many advantages for intramuscular injections. Six areas may be used before an injection has to be repeated in the same site. A large volume of injectate can be accommodated. There are no important nerves in the neighbourhood. The skin should be cleaner than the buttock. The patient does not have to sit on the injection site. There is usually less discomfort in the thigh than in the buttock or shoulder. The only disadvantage common to all sites is the presence of blood vessels.

I would therefore advocate that all intramuscular injections should be given in the lateral aspect of the thighs, and that the deltoid and buttock be abandoned on account of the dangers that may accompany the not-so-rarely misplaced injection.—I am, etc.,

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IgD Myeloma

SIR,—Since the first description by Rowe and Fahey,¹ several cases of IgD myeloma have been reported.²⁻⁴ In 1966 one case was also described in Italy.⁵ This variety of plasmocytoma is however uncommon; thus we think it could be useful to report here a case that we have recently observed.

The patient was a 71-year-old man in good clinical condition who had been complaining of cervical and lumbar pain since August 1967, and was seen in October in the rheumatological centre of this hospital. X-rays of cervical, thoracic, and lumbar spine revealed diffuse osteoporosis with signs of spondyloarthrosis. Because of an increased E.S.R. and of a dysproteinaemia the patient was transferred to this institute.

The clinical examination was negative. E.S.R.=135 mm./hour. Serum electrophoresis showed total proteins 8.5 g./100 ml. and gamma-globulins 3.65 g./100 ml. A marked M band was present in the fast- λ region (see Fig.). There was marked proteinuria and a narrow band of Bence Jones protein was detected by electrophoresis. Bone marrow was difficult to obtain,

but its appearance was typical with many myeloma cells present. X-rays of skull revealed widespread osteolytic lesions that were not present in the spine, ribs, or pelvis. The immunoelectrophoresis showed a reduction of IgG, IgA, and IgM. With an antiserum against the heavy chains of IgD (kindly provided by Professor C. Ricci) the paraprotein gave a typical thick bowed arc, and the light chains of the myeloma protein and the Bence Jones protein were λ type. Albumin and other proteins were also observed in the urine, but no clinical symptoms of renal failure were present.

This case was not different, so far as the haematological and clinical findings are concerned, from other myeloma types. It is interesting that the light chains are of λ type: this occurs in one-third of myelomata, but is much more frequent in IgD plasmocytoma.³—We are, etc.,

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REFERENCES

- Rowe, D. S., and Fahey, J. L., *J. exp. Med.*, 1965, 121, 171.
- Hansson, U. B., Laurell, C. B., and Bachmann, R., *Acta med. scand.*, Suppl. No. 445, 1966, 89.
- Hobbs, J. R., et al., *Lancet*, 1966, 2, 614.
- Zawadzki, Z. A., and Rubini, J. R., *Arch. intern. Med.*, 1967, 119, 387.
- Ventruto, V., and Quattrin, N., *Haematologica*, 1966, 51, 545.

Long-term Administration of the Pill

SIR,—Mr. P. C. Steptoe (30 March, p. 836) asserts that "the long-term use of these [oral contraceptive] pills carries with it the definite danger of producing irreversible sterility." This is contrary to the conclusions reached by the W.H.O. Scientific Study Group on Oral Gestogens,¹ which was that "there is no evidence that the fertility of women is impaired after the long-term use of oral contraceptives (five or more years)." Mr. Steptoe's findings in ovarian biopsies in long-term users are not original, and his implication that they represent ovarian damage is not borne out by his own statement that many of the patients with secondary amenorrhoea can be induced to ovulate with clomiphene.

It seems probable that the secondary amenorrhoea which may follow cessation of oral contraceptive use in a small and at present undetermined proportion of women is due to disturbance of hypothalamic gonadotrophin-releasing factor production, a condition which arises spontaneously often enough. As yet we have no means of knowing whether the prevalence is greater in women giving up oral contraception than in comparable women who have never used it. Other unconnected situations may also occur: one of my patients, referred because of secondary amenorrhoea on stopping oral contraception, was found on investigation to have adrenal hyperplasia. I am quite sure she had this before she started oral contraception and she responded in the expected manner to corticosteroid therapy, with conception soon afterwards.

To involve the thalidomide tragedy in this context, as Mr. Steptoe does, seems irrelevant and even irresponsible.—I am, etc.,

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REFERENCE

- Scientific Study Group on Oral Gestogens, *Wld Hlth Org. techn. Rep. Ser.*, 1966, 326.

Gastrectomy and Vagotomy

SIR,—I am grateful to Mr. H. Taylor and Mr. D. G. A. Eadie (23 March, p. 766) for pointing out that the Polya gastrectomy referred to in my recent paper (3 February, p. 288) was carried out using either a retrocolic or antecolic loop of jejunum anastomosed to the gastric remnant. I should, of course, have made this clear.

Mr. Taylor is a strong and persuasive advocate of the "no-loop" gastrectomy and would no doubt maintain that this particular technique should have been used in the trial I reported. However, Mr. Taylor¹ finds it necessary to warn his patients that "they must not eat too much, too quickly," and reported that "several patients had changed their jobs to get more favourable conditions for meals during the day." These do not impress me as the attributes of a good operation, even if it is free of other sequelae such as bilious vomiting or recurrent ulcer. It is difficult to believe that these sequelae are completely eliminated by the "no-loop" technique and it would be helpful to have some comparative facts and figures derived from a properly planned prospective trial.

It is difficult to design a perfect clinical trial but I believe that my paper provided a useful comparison of symptoms after two gastric operations as they are commonly performed.—I am, etc.,

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REFERENCE

- Taylor, H., and Eadie, D. G. A., *Brit. med. J.*, 1967, 1, 15.

Dealing with Attempted Suicide

SIR,—Not infrequently would-be suicides refuse any form of medical attention. In most such cases doctors are confronted with an individual who allegedly has ingested an overdose of drugs and subsequently refuses a stomach wash-out or other appropriate measure. On such occasions general practitioners, casualty officers, or mental welfare officers contact the psychiatrist by telephone—more often than not in the middle of night—and ask for further directions. Though some of the suicide attempters may be acutely disturbed or under the influence of alcohol, others need not necessarily display any gross psychological abnormalities. A proportion of these cases are animated by exhibitionism, and their refusal of a stomach wash-out is due to their secret knowledge of the futility of such a procedure. They have either not ingested any drugs at all or only small quantities of an innocuous kind. However, it is always prudent to give them the benefit of a doubt, as this is the surest way of avoiding tragedy.

The problem arises: Is the psychiatrist or any doctor justified—on moral, medical, or psychiatric grounds—to enforce treatment by putting such an individual on a compulsory order? I believe that he is. Some have remarked that doctors had no authority to enforce a blood transfusion if a person objected on religious grounds. They think the situation is analogous with suicide attempters. This analogy is not quite correct, for the individual refusing blood transfusion on religious grounds does not object to other forms of treatment, nor does he necessarily

A is normal serum, B is diluted patient's serum, C is patient's urine.

wish to die. In suicidal attempts there is an implied intention to kill oneself and therefore an emergency compulsory order (Section 29, Mental Health Act, 1959) seems indicated, even in the absence of other psychological abnormalities.

The second problem is one of disposal. Suicide attempters ought not to be admitted directly to a mental hospital since their immediate needs are medical or surgical in nature. Some regional hospital boards emphasize this point. Mental hospitals have not the same facilities to deal with emergencies; besides their usually remote location implies the loss of valuable time, which in emergencies can make all the difference.

I believe that such patients, after being put on an emergency three-day order, ought to be detained in a general hospital. This procedure is in accordance with the abolition of "designated hospitals" for the treatment of mental illnesses of the Mental Health Act, 1959. After preliminary first aid and short observation in the general hospital the patient is seen by the psychiatrist and transferred to a mental hospital if necessary.

Though this procedure seems a logical one some general hospitals refuse admission of patients on a compulsory order. In view of the controversial nature of this topic, comments from the Ministry of Health may help to clarify this matter.—I am, etc.,

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Vitamin C and Gastroduodenal Disorders

SIR,—We were interested to read the reports by Dr. J. M. Williamson and others (1 April 1967, p. 23), by Dr. M. M. Cohen and Miss Anne M. Duncan (2 December 1967, p. 516), and the letter by Dr. I. W. Dymock and others (20 January, p. 179) on the estimation of leucocyte ascorbic acid levels in patients with malabsorption or gastroduodenal disorders. Using the method adopted by these authors, we have also found a marked depletion of ascorbic acid in subjects with duodenal ulcer. The mean leucocyte ascorbic acid level in 16 patients was $10.6 \mu\text{g./}10^6 \text{ W.B.C. (S.D.} \pm 4.9)$, while that in the controls was $22.1 \mu\text{g./}10^6 \text{ W.B.C. (S.D.} \pm 6.4)$.

We have attempted to assess if ascorbic acid deficiency was correlated with secretory capacity on maximal histamine stimulation. Low levels of leucocyte ascorbic acid were found both in subjects with hypersecretion and hyperchlorhydria, and in patients with normal secretory results. These findings suggest that depletion of vitamin C in such disease is independent from the degree of gastric acid concentration. We have also studied ascorbic acid metabolism in three cases of diaphragmatic hernia, in four cases of ulcerative colitis, and in 12 cases of advanced liver cirrhosis, all of whom were known to have an intake of ascorbic acid of at least 30 mg./day. In the first two conditions the leucocyte ascorbic acid test showed a significantly lower level than in the control group; in patients with liver cirrhosis the mean buffy layer ascorbic acid level was $13.1 \mu\text{g./}10^6 \text{ W.B.C. (S.D.} \pm 5.1)$.

From such preliminary findings it would appear that ascorbic acid deficiency is found also in states—for example, advanced liver

cirrhosis, in which dysfunctions of gastro-intestinal motility and absorption are often present. It is not yet clear if ascorbic acid is absorbed through the intestinal mucosa by diffusion or by active transport process,¹ but it may be suggested that inadequate absorption of the vitamin in such conditions is the most important factor causing its deficiency. Reduction of the dietary intake or increased utilization of the vitamin can obviously concomitate, and in some cases they could play the prevalent pathogenetic role.—We are, etc.,

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REFERENCES

- 1 Spencer, R. P., Purdy, S., Hoeldtke, R., Bow, T. M., and Markulis, M. A., *Gastroenterology*, 1963, 44, 768.
- 2 Stewart, J. S., and Booth, C. C., *Clin. Sci.*, 1964, 27, 15.

Cyclamates

SIR,—Your leading article on "Cyclamates" (9 March, p. 594) contained the following statement: "Such evidence as existed suggested that toxicity of cyclohexylamine itself was low." This statement could be misleading. In fact the oral L.D.₅₀ for cyclohexylamine in the rat had been shown to be in the region of 300 mg./kg. body weight.¹ This contrasts with that for sodium cyclamate, which had been shown to be 6,000–12,000 mg./kg. body weight.² Furthermore, cyclohexylamine had been shown to cause changes in the liver and kidneys of rats at a dosage level of 60 mg./kg. body weight.³ It is hoped that the further studies that have been requested will show that cyclohexylamine does not present any hazard to man at the levels that are likely to occur after consuming cyclamate-containing foods. However, it is idle to suggest that conversion of cyclamate to cyclohexylamine does not materially alter the potential hazard.—I am, etc.,

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REFERENCES

- 1 Flinn, F. B., *Ind. Eng. Chem.*, 1937, 29, 1247.
- 2 Richards, R. K., Taylor, J. D., O'Brien, J. L., and Duescher, H. O., *J. Amer. pharm. Ass.*, 1951, 40, 1.
- 3 Pliss, G. B., *Probl. Oncol. (N.Y.)*, 1958, 4, No. 6, 22.

Royal Malady

SIR,—The proponents of the theory that George III and others suffered from variegate porphyria have had a good run recently in your correspondence columns. May I try to sum up? We seem to be divided still on two fundamental issues—namely, is variegate porphyria a really serious disease in its own right when not provoked by modern drugs, and is it sufficiently similar to acute intermittent porphyria to allow experience of the latter to be applied usefully to patients with the former? My position here is that only one clinician has really adequate and reliable knowledge of variegate porphyria as such, and he (Dr. G. Dean,

17 February, p. 443) has clearly stated that the case for the alleged royal malady is not proved. This was mainly on the grounds that variegate porphyria in the past was not a serious disease, but also from his knowledge of hundreds of living patients who are kept off certain drugs. The most I can allow is that in time, when we have in Great Britain collected sufficient cases of proved variegate porphyria to allow our having proper opinions of our own, it may be that we could find that our so-called variegate porphyria is not the same as the South African variety either genetically, biochemically, or clinically. Current findings make this seem rather unlikely, although in the world of biochemical genetics it is very common to find that an alleged single disease, for instance phenylketonuria, is later found to be more than one disease.

May I take a firm stand about the visible urinary changes? I insist still that the urine in an acute attack of either form of porphyria is usually of normal colour when freshly passed. The essential and characteristic feature, not present in the descriptions of the royal malady, is the development of reddish or brownish coloration on standing. This still occurs even in those rarer cases when the urine is passed already slightly coloured. The reference quoted by Professor Rimington (24 February, p. 510) from Drs. Waldenström and Vahlquist is unfortunately incorrectly described. These authors state that if the urine is passed strongly acid the darkening on standing occurs more quickly than if alkaline. They did not state that acid urine is passed already coloured, nor that it does not then further darken on standing. Their colour plates of this phenomenon should, by the way, be inspected by those who may still feel that the colours in question bear any relation to what we usually call purple.

I am sorry my genetic thoughts have not got across to Professor A. Goldberg (24 February, p. 509), who somehow relates them to "mathematical pyrotechnics." May I try again with a briefer family tree and leaving out all figures and horrible calculations? What I am now trying to point out is that if, as Dr. Ida Macalpine and her colleagues suggested, the variegate porphyria gene passed directly down the royal line from Mary Queen of Scots to George IV—that is, through nine generations—then the gene must have had an uncanny knack, defying, I think, scientific explanation, for picking out the subjects in the direct line of succession: first son when available, otherwise whoever comes next according to the particular rules in this complicated game. Perhaps he will think again about the relation of these man-made laws of inheritance to those put forward by Mendel, bearing in mind the now generally accepted rules for the inheritance of an autosomal dominant gene. Till he has explained the alleged royal inheritance to me I will continue to tell the students here that the reassortment of genes before each conception is "just a blinking lottery" and that this occurs among royalty just as in the common herd.

My object in raising these matters originally was to tell historians that there is considerable doubt among some clinicians interested in the porphyrias about the story of the royal malady. I was most distressed to note that one historian seemed to have swallowed the story hook, line, and sinker