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 present 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 then in the psychiatrist's care 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 controversy surrounding this topic, reference from the Ministry of Health may help to clarify this matter.—I am, etc.,

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Vitamin C and Gastrointestinal 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. Dynnuck and others (20 January, p. 179) on the estimation of leucocyte ascorbic acid levels in patients with malabsorption or gastrointestinal disorders. Using the method adopted by these authors, we have also found a marked correlation between ascorbic acid in subjects with duodenal ulcer. The mean leucocyte ascorbic acid level in 16 patients was 10.6 μg/10 W.B.C. (S.D. ± 4.9), while that in the controls was 22.1 μg/10 W.B.C. (S.D. ± 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 in 8 of 15 patients with ulcer and hypertrophy, 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 the control group; in patients with liver cirrhosis the mean buffy layer ascorbic acid level was 13.1 μg/10 W.B.C. (S.D. ± 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 gastrointestinal 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, 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


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. for cyclohexylamine in the rat had been shown to be in the region of 300 mg./kg. body weight. This contrasts with that for sodium cyclamate, which had been shown to be 6,000–12,000 mg./kg. body weight. 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. 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.

I am, etc.,

ALASTAIR FRASER.

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REFERENCES


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? It is 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 I am in the wrong on this point. The diagnosis 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 say 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 form a valid opinion. Our colleagues in 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 clinical status of this phenomenon is not yet 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 am now trying to do is this: if, as Dr. Ida Macalpine and her colleagues suggested, the variegate porphyria gene passed directly down the royal line from Queen Victoria of Scotland to George IV, and is, through nine generations—then the gene must have had an uncanny knock, 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 mundane 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 read that one historian had swallowed the story hook, line, and sinker.