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

- Spencer, R. P., Purdy, S., Hoeldtke, R., Bow, T. M., and Markulis, M. A., *Gastroenterology*, 1963, 44, 768.
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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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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

(13 January, p. 109). I wanted and still want to warn the others. I have my suspicions, too, about you, Mr. Editor, as the result of your putting out a special supplement on the royal malady complete with vivid purple-coloured cover!—I am, etc.,

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REFERENCE

- ¹ Waldenström, J., and Vahlquist, B., *Acta med. scand.*, 1944, 117, 1.

Steroids and Nephrotic Syndrome

SIR,—Dr. I. G. Wickes (16 March, p. 706) claims that the regimen he uses will identify those children with nephrotic syndrome who are "likely to relapse." He instructs the mothers to test their child's urine with Esbach's reagent and to record the result. However, this does not predict anything; it merely records the relapse or the degree of proteinuria. We know of no way of predicting which nephrotic child will run a relapsing course; neither renal biopsy nor the selectivity of proteinuria will do this. Dr. Wickes then advocates that the mother should titrate the dose of steroid against the amount of proteinuria, maintaining that in this way the increasing proteinuria that may be associated with colds and other infections is kept in check. We do not doubt that the proteinuria goes during this regimen, but believe that it would often disappear without any therapy. Our experience accords with that of Arneil and Lam.¹ Very often the proteinuria goes as the child recovers from the respiratory infection, and therefore it is better to withhold steroids for one or two weeks, provided that oedema does not develop. Withholding therapy avoids needless steroids, and parents agree to it willingly, since the child does not necessarily feel ill at the time of the proteinuria.

As he suggests, a simple record of proteinuria kept by the parent can be very helpful for outpatient supervision. However, Esbach's test seems a rather cumbersome one to inflict on parents, particularly as it takes 24 hours to read. Albustix is a simple and reliable test² which a parent or child can use readily at home, recording the result in a few seconds. When demonstrating the method we warn them that trace and transient one-plus readings are unimportant.—We are, etc.,

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REFERENCES

- ¹ Arneil, G. C., and Lam, C. N., *Lancet*, 1966, 2, 819.
² Rennie, I. D. B., and Keen, H., *Lancet*, 1967, 2, 489.

Detection of Bacteriuria

SIR,—Dr. J. M. Bradley and others in their paper (16 December, p. 649) have misquoted the results we obtained in our assessment of the triphenyl tetrazolium chloride reduction test for the detection of significant bacteriuria.¹ Their statement that in our series "only 44% of infections in the 10⁴–10⁹ organism per ml. of urine range were

detected" referred only to the ability of the test to detect infections with Gram-positive organisms, and this was made clear in our paper. As we also made clear, when patients on antibiotic therapy were excluded, the triphenyl tetrazolium chloride (T.T.C.) test picked up 12 of 24 infections with Gram-positive organisms, and these included, besides a number involving *Streptococcus faecalis*, infections with diphtheroids, micrococci, and *Candida albicans*, infections which the method described by Dr. Bradley and colleagues, depending as it does on the use of one MacConkey plate only, must inevitably miss.

When all urines with significant bacteriuria (10⁵ or more bacteria per ml.) encountered in a general hospital are considered the detection rate for the T.T.C. test was 182 of 258 specimens or 70.5%. Our paper further pointed out that in a selected population, such as in obstetrical patients, where 79% of all infections are due to *E. coli*, the detection rate of the T.T.C. test can be bettered, and can detect as many as 90% of cases of significant bacteriuria.

No chemical test, of course, can replace accurate quantitative culture of urine, but, as Dr. Bradley and colleagues point out, there is a real need for a simpler, less expensive screening method. We found, and reported, the T.T.C. test to be a much better screening method than the quotation of our paper would suggest. A direct comparison between the T.T.C. test and the method of Dr. Bradley *et al.* is not possible. Their graph (Fig. 2) shows a reasonable correlation between the number of colonies counted by their method, and a true quantitative culture in 20 specimens, none of which apparently had more than 10⁵ bacteria per ml. The graph (Fig. 4) showing the correlation between counts obtained with a standard loop and their screening method also shows a correlation, but in fact their paper does not state how many of a given number of positive urines can be screened out, nor how many false-positives are obtained, and the comparative values of the T.T.C. method (which is simpler to perform) and their screening method remain to be assessed.—I am, etc.,

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REFERENCE

- ¹ Hinton, N. A., and van der Hoeven, E., *Canad. Med. Ass. J.*, 1965, 93, 639.

Acute Iron Poisoning in Children

SIR,—Drs. D. G. D. Barr and D. K. B. Fraser (23 March, p. 737) report further tragedies from swallowing ferrous sulphate. This is the salt of a weak base and strong acid. Its solution has a pH of 3.7 and readily damages any mucosa with which it is long in undiluted contact. Many neutral salts are now available which small children may swallow by the dozen without harm. Often these are damned because they cost 2d. a week more than ferrous sulphate, but with increasing use of desferrioxamine even this miserable economic argument may fail. Is it not time to abandon and to ban the use of ferrous sulphate, which is a lethal poison?—I am, etc.,

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Q Fever

SIR,—The account of Q fever in Northern Ireland by Dr. J. H. Connolly (2 March, p. 547) prompts us to report our own experience in the period 1960–7. During these eight years we have made a laboratory diagnosis of Q fever by serological tests or by isolation of *Rickettsia burneti* from 24 patients (and confirmed it in two additional cases from other laboratories). They resembled closely those reported by Dr. Connolly in age and sex (see Table) and in seasonal distribution.

Age and Sex Distribution

Age Group	Male	Female	Total
10–29	4	1	5
30–49	6	4	10
50–69	5	2	7
70+	2	0	2
All ages	17	7	24

The acute illness of two patients began in the first quarter of the year, of 10 in the second, one in the third quarter, and six in the fourth. The types of illness were: pneumonia, 10 cases (one chronic with probable endocarditis); fever, eight cases (one severe with vomiting, dehydration, and rash; one post-partum); subacute endocarditis, four fatal cases, all male; pericarditis, two cases, one with pleuropericarditis, one with aortitis and syphilitic aneurysm. The last patient, the four with subacute endocarditis, and the patient with pneumonia and probable endocarditis all had high complement fixing antibodies to both phase 1 and phase 2 antigens, as had also an additional case of endocarditis in which we made confirmatory tests for another laboratory. Serious cardiovascular diseases have thus been more prominent in our series than in Dr. Connolly's, but pericarditis was found in both series. Tests for Q fever as well as for Coxsackie virus infection therefore seem appropriate in cases of acute pericarditis.

Contact with farm animals (mostly bovine) or their products was traceable in most cases. Six were farmers, three had driven trucks loaded with dung, one had cleared dung from cattle wagons, and three used dung as gardeners; three had consumed unpasteurized milk and one of these lived beside a farm; one office worker frequently visited a byre; an electrician developed pneumonia with serological evidence of both Q fever and a psittacosis-group infection after working in an abattoir¹; another case of pneumonia developed after working in fields near lambing sheep. No contact was traced in three Glasgow patients or three from the counties of Renfrew, Dumfries, and Kirkcudbright; in each of these counties Q fever is known to infect cattle. Infection of sheep and cattle in the west of Scotland has been recognized for more than a decade² and continuing surveillance tests in collaboration with Dr. T. F. Elias-Jones continue to reveal *R. burneti* in samples of unpasteurized milk. We agree, however, with Dr. Connolly that inhalation of particles derived from farm animals or their products seems a more important source of infection than ingestion of infected milk.

The finding of false positive complement fixation antibody responses to Q fever in some children with infections due to adenoviruses types 7 and 21 was reported at the meeting of the Society for General Microbiology, Janu-