Correspondence

British Medical Journal

13 April 1968

Vitamin C and Gastrointestinal Disorders

SIR,—We were interested to read the report 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. Dynmock 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 of ascorbic acid in subjects with duodenal ulcer. The mean leucocyte ascorbic acid level in 16 patients was 10.6 μg./10 cm.3 W.B.C. (S.D. ± 4.9), while that in the controls was 22.1 μg./10 cm.3 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 both duodenal ulcer 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 lowered result. In the control group: in patients with liver cirrhosis the mean ascorbic acid level was 13.1 μg./10 cm.3 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

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. My 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? Secondly, 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 this is the alleged royal malady that 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 add 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 the 'royal malady' we speak of 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 is not present in acute attacks 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. Their urine does not pass strongly acid and the urinary colouring on standing occurs more quickly than if alkaline. They did not state that acid urine is passed already coloured, nor that it does not further darken on standing. Their claims of this phenomenon are not 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 brief family tree and leaving out all figures and horrible calculations? When I am now trying to do is 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 III (or IV), 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 manic depressive 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 find that one historian has swallowed the story hook, line, and sinker.