BRITISH MEDICAL JOURNAL

SATURDAY 29 JUNE 1968

LEADING ARTICLES

PAPERS AND ORIGINALS

PRELIMINARY COMMUNICATIONS

MEDICAL MEMORANDA

MIDDLE ARTICLES

BOOK REVIEWS 815

OBITUARY NOTICES 832

NEWS AND NOTES

CURRENT PRACTICE

CORRESPONDENCE 826

SUPPLEMENT

No. 5608 Brit. med. J., 1968, Vol. 2, 775–838 Weekly. Price 5s.
BRITISH MEDICAL ASSOCIATION, TAVISTOCK SQUARE, LONDON W.C.1 TEL: 01-387 4499

Correspondence

Letters to the Editor should not exceed 500 words.

A Puzzle

SIR,—The Clinicopathological Conference on a case of adult coeliac disease resistant to treatment (15 June, p. 678) interested me for a number of reasons. There was the puzzling nature of the disease and the vigour and erudition displayed by the experts, whose narrow views of the situation contrasted sharply with the broad common sense of the general practitioner. And yet it sounded to me as though the general practitioner had been dealt with cavalierly and told in effect that his observations did not contribute to the understanding of a purely organic problem.

I particularly noticed that, although skin lesions had been a part of the patient's illness, no evidence was presented of a dermatologist having been asked for his opinion, and no dermatologist spoke at the meeting. It did not appear as though any skin biopsy had been done with the object of demonstrating the histopathology of the skin changes, although a vasculitis was found incidentally in material taken for histochemical study. Had a dermatologist spoken he could have said that a vasculitis of skin and muscles had been recognized, and that many forms of vascular disease have cutaneous components that may aid their recognition,[1] and he could also have come to the aid of the general practitioner in affirming his familiarity with organic bodily changes that depend upon emotional causes.

Dermatologists may be ignorant in the sense that they share the lack of omniscience which characterizes humanity in general, but I do not believe that they know less than their colleagues in other disciplines, and it is worth while asking their opinion. Certainly the ignorance of dermatology generally displayed by Britain's physicians is profound. This might be of little consequence did it not lead them to believe that because they know little there is little to be known, and to act on this belief. The truth is quite otherwise. Although the subject is vast a great deal is known. But, as Professor P. J. Hare said,[2] dermatology lacks facilities, money, and workers skilled in specialized techniques. So please, if you have ill patients with skin lesions, ask for the help of a dermatologist; and if you are in a position to dispense scientific help, remember Cinderella in the skin department.—I am, etc.,

A. LYELL.

Department of Dermatology,
Royal Infirmary,
Glasgow.

REFERENCES

1 *Textbook of Dermatology*, ed. by A. Rook, D. S. Wilkinson, and F. J. G. Ebling, 1968. London and Edinburgh.
2 Hare, P. J., *Lancet*, 1968, **1**, 1306.

SIR,—The case reported at the Clinicopathological Conference (15 June, p. 678) puzzled the experts, including you (p. 640), so I venture to dash in where you feared to tread and to suggest that the man had deficiency of pyridoxine. This vitamin of the B complex is required mainly for the metabolism of amino-acids, and its dietary requirement is proportional to this (and therefore to metabolism of protein). The increased synthesis of protein in the intestinal mucosa, the loss into the intestine, and the "vast amount of parenteral protein" he was given must have increased his requirement, and there may have been decreased absorption of the vitamin from the intestine. We are not informed how, if at all, pyridoxine was given to him. The statement that he received "large doses of parenteral vitamins throughout his illness" (vitamins B_{12} and E and folic acid being specified) is not sufficiently helpful.

Deficiency of pyridoxine could account for most of his lesions. He had a "curious rash," thought to be like that of deficiency of essential fatty acids, since topical application of these was unsuccessfully tried (and it would be discourteous not to acknowledge that Dr. Neale telephoned me on 15 February 1967 about the possibility of such deficiency). But the skin lesion of this deficiency is superficially so similar to that of pyridoxine deficiency that Sherman[1] claimed the two to be identical—erroneously, as we showed.[2] The mental changes of acute pyridoxine deficiency and the peripheral neuropathy of chronic deficiency are very similar to those of thiamine deficiency, and I suggested a common biochemical basis of both.[3] He had mental changes when he had "emotional crises" (and therefore possibly defective diet), and his peripheral neuropathy, "beriberi-like," was not explained. He had low plasma taurine, which is found in pyridoxine deficiency, since cysteinesulphinic-acid-decarboxylase is very sensitive to such deficiency. Levels in plasma of other amino-acids vary with dietary protein, but in rats there is a striking increase in glycine and cystathionine and decrease in serine and cystine[4] (of these glycine and serine were measured in the patient; the former was almost the only amino-acid in the normal range and serine was low, thus supporting pyridoxine deficiency). Production of circulating antibodies is decreased in pyridoxine deficiency; the patient had septicaemia. Fatty liver and haemosiderosis, both of which he had, are very common in pyridoxine deficiency. Intolerance to ethanol, which he had, is alleged in the deficiency. Finally, since Paneth cells secrete protein, they might be expected to degenerate in deficiency of pyridoxine, since this vitamin is necessary for the synthesis of protein.

So I am puzzled by not being told the amount of pyridoxine he received, and why xanthurenic acid was not measured after a tryptophan load—I am, etc.,

Oxford. HUGH SINCLAIR.

REFERENCES

1 Sherman, H., *Vit. Horm.*, 1950, **8**, 55.
2 Ramalingaswami, V., and Sinclair, H. M., *Brit. J. Derm.*, 1953, **65**, 1.
3 Sinclair, H. M., in *Chemical Pathology of the Nervous System*, Proceedings of the Third International Neurochemical Symposium, edited by J. Folch-Pi, 1961, p. 98. New York.
4 Swendseid, M. E., Villalobos, J., and Friedrich, B., *J. Nutr.*, 1964, **82**, 206.

Publicizing of Adverse Drug Reactions

SIR,—It has been suggested that I was less than fair in stating in a recent address to the Pharmaceutical Group of the Royal Society of Health that "medical journals have some inhibitions in publicizing adverse reactions to drugs." I gladly agree that many of the first warnings of adverse effects of