Correspondence

20 January 1968

BRITISH MEDICAL JOURNAL

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Malaria in Britain

Sir,—Three weeks ago a patient was admitted to our unit in a semiconscious condition and with high fever. Examination of the blood revealed massive infection with malignant tertian malaria (P. falciparum). She responded well to parental therapy and was eventually discharged cured of the infection but anemic. The diagnosis of malaria was not confirmed until her admission to our unit 10 days after the fever started. During this time she was examined by two general practitioners. On the first occasion she was apparently considered to have influenza, in common with many others in this area at the time. On the second occasion, the day of admission, the diagnosis of malaria was suspected. By this time her condition had deteriorated sharply and she was extremely ill with progressive drowsiness and confusion. She was lucky. In her case the diagnosis was made in time. In case of P. falciparum malaria which occurred in the Midlands shortly after this episode diagnosis was made too late and the patient died.

These cases illustrate the case with which a specific diagnosis like malaria. They are often mistreated for much commoner fevers such as influenza, especially at this time of the year. They also indicate the serious nature of such mistakes, which could be avoided (as we have not been overestimated) if the patient's geographical history was taken as a routine at the time of first examination. In our case the simple question: “Where have you been and when?” would have disclosed the following. The patient was admitted in late November 1967 from north-eastern Nigeria, where she had been living since February with her husband in a road-construction camp. Malaria was common in the area. She had not taken antimalarial suppressive drugs. Had this information been obtained at the first examination the possibility of malaria would have (or should have) occurred to the practitioner. Exotic infection should have been considered in any case in this area. Since, according to the husband, the yellow card issued by the Ministry of Health on arrival at London Airport was shown to the doctor at the time of the first examination. This card states: “Whilst abroad you may . . . have been in contact with some dangerous epidemic disease. . . . If during the next 21 days, wherever you may be, you or any person in the same house fall ill, in your own interest call in a doctor immediately and give him this card.”

The case of malaria we have reported here is only one example of imported infection which was missed or nearly missed in the course of general practice. We could quote many others. In fact in our experience general practitioners, with a few notable exceptions, still seem unaware of the likelihood of meeting imported diseases in practice, especially in air travellers, and of the serious consequences which may result from their being overlooked. We have tried hard to enlighten them in this respect not only here but in other sophisticated countries where similar problems arise.†

It looks as if more pressure is needed. Perhaps this is an area in which the Royal College of General Practitioners could exert some influence.—We are, etc.,

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Vitamin C and Gastroduodenal Disorders

Sir,—I have read with interest the original article by Dr. M. M. Cohen and Miss Anne M. Duncan (2 December, p. 516) describing their studies on ascorbic acid metabolism in patients with gastroduodenal disorders. Continuing our earlier studies (6 May, p. 375) we have now investigated 109 patients with duodenal ulceration before or after surgery.

**Buffy Layer Ascorbic Acid Levels in Gastroduodenal Disorders**

<table>
<thead>
<tr>
<th>Group</th>
<th>No. Studied</th>
<th>Buffy Layer Ascorbic Acid—mg/100 W.B.C.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>25</td>
<td>18.7±5.5</td>
</tr>
<tr>
<td>Duodenal ulcer</td>
<td>34</td>
<td>4.5±2.1</td>
</tr>
<tr>
<td>Previous gastric surgery</td>
<td>9</td>
<td>5.6±1.4</td>
</tr>
<tr>
<td>Previous gastric surgery with symptoms</td>
<td>39</td>
<td>7.3±3.0</td>
</tr>
<tr>
<td>Duodenal ulcer with stenosis</td>
<td>27</td>
<td>0.9±0.3</td>
</tr>
</tbody>
</table>

Compared with control patients our subjects with duodenal ulceration showed evidence of depletion of ascorbic acid, as did the patients who had duodenal stenosis secondary to ulceration. However, the depletion was more severe in those with stenosis. In patients who had previously undergone gastric surgery and who were in good health most were found to have normal levels but a few showed depletion. Despite an apparently normal dietary intake of ascorbic acid. These results contrast with those of a group of patients who had a variety of complications following gastric surgery, including afferent loop and dumping syndromes, recurrent ulceration, and malabsorption. In these subjects depletion was present in the majority.

We conclude from our survey that depletion of buffy layer ascorbic acid is commonly found in patients with duodenal ulcer, duodenal stenosis, and postgastrectomy syndromes. Some patients who have previously undergone gastric surgery, and are well, may also show depletion. We would agree with Dr. M. M. Cohen that dietary factors are the likely cause of the ascorbic acid depletion but suspect that, as suggested by Dr. J. M. Williamson and others (1 April, p. 23), there may be inadequate absorption or increased utilization in some patients. It has been suggested that antacids, particularly aluminum hydroxide, might be responsible, but Hoffman and Dyneiwicz found that alumina gel had no significant effect on the absorption of ascorbic acid. We agree with Dr. M. M. Cohen and his colleagues that supplementation of ascorbic acid may be required in these patients. A good response to therapy has been obtained in all patients.—We are, etc.,

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**Emotion and Ileitis**

Sir,—Perhaps out of anxiety that it be used by others for antipsychosomatic poliemics I should like to comment on the impressive study of regional ileitis by Dr. F. Feldman and his colleagues (23 December, p. 711) questioning the psychosomatic nature of the disorder. Although they interpret their results with caution, it should be pointed out that the crucial assessments of patients and controls were apparently not made “blindly” (thus exposing them to unconscious bias), and without knowing the composition of the gastroenterological group (whatever mixture of “organic” and “functional” disorders) it is not really possible to evaluate the figures given.

Nevertheless, possible alternative explanations exist. The higher incidence of “psychosomatic signs” in the control group could indicate that the latter are predominantly “psychosomatic,” or that the regional ileitis group are “abnormally normal.” Applying the x² test for two independent samples† to two of their criteria suggests that these possibilities should be considered. Using a 1-tail test (predicting that regional ileitis patients are more “normal”) the higher incidence of “normal personalities” is of borderline significance (0.1>P>0.05), and the lower incidence of preceding “general, conscious stress” is significant (0.05>P>0.025).

The difference in the reporting of stress may be a function of the passage of time, as the average onset was nearer nine than three years before the regional ileitis patients were seen by the authors. However, Dr. Feldman and his colleagues might have data that explains these differences in terms of their
personalities. The high incidence (17 out of 19) of "normal passive" and "normal aggressive" personalities suggests a link with the passive personalities and aggressive conflicts said to be important in "psychosomatic" illnesses, including ulcerative colitis. Further, the possibility exists that defensiveness (the use of such mechanisms as projection, denial, and intellectualization) and a reduced capacity for the non-somatic expression of emotional disturbances are playing a part in the disease. This might put regional ileitis alongside such "low neuroticism" groups as patients with lung cancer, coronary thrombosis, and perhaps the type of eczema patient that I have called "super-stable". A crucial point in psychosomatic research might be, as the authors point out, the difficulty of assessing deeper problems and defenses with brief interviews (and, one might add, questionnaires or other means).

The psychosomatic criteria used by Dr. F. Bendien (ibid., 6, 1967) and his colleagues are clear and an excellent basis for work in this controversial field. But, as presented, their data only hint at their conclusions. The importance of their paper is increased by the fact that they provide us with enough information to stimulate our doubts and stimulate us to ask for more.—I am, etc.

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REFERENCES

WATER VAPOUR LOSS OF PSORIASIC SKIN

Sir,—The report of Drs. Katherine A. Grice and F. R. Betteley (28 October 1967, p. 195) reveals very interesting data of trans-epidermal water loss. For that very reason we regret that they state: "The mean trans-epidermal water loss in unaffected skin areas in 16 cases of psoriasis . . . was not significantly different from normals," as it may suggest that no difference exists. However, different values of the water vapour loss at unaffected skin can be determined during certain periods of the disease. An example is the Figure below, representing a series of measurements at the volar aspect of the forearm skin of a 16-year-old patient (psoriasis inversa). At the unaffected skin at time 0 a somewhat increased water vapour loss of 1.2 mg./sq. cm./hr. was found, decreasing in about four days to a normal value of 0.6 mg./sq. cm./hr. (dotted curve P.I.), but rising again coincidentally with a clinical exacerbation elsewhere. A psoriatic patch (P.s.) at time 0 having a higher water vapour loss than unaffected skin showed a steadily decreasing water vapour loss comparable with normal healing of stripped skin in normal volunteers and unaffected by the clinical exacerbation when it started. Unaffected (N) and psoriatic skin (P.s. str.) of the patient was stripped with adhesive tape in such a way that the tape covered unaffected as well as psoriatic skin in the very same

SIR,-The article by F. Bendien (ibid., 1967, 6, 123) revealed the data of trans-epidermal water loss of unaffected skin areas in 16 cases of psoriasis. The authors state: "The mean trans-epidermal water loss in unaffected skin areas was not significantly different from normals," suggesting that no difference exists. However, different values of the water vapour loss at unaffected skin can be determined during certain periods of the disease. An example is the Figure below, representing a series of measurements at the volar aspect of the forearm skin of a 16-year-old patient with psoriasis inversa. At the unaffected skin at time 0 there was a somewhat increased water vapour loss of 1.2 mg./sq. cm./hr., which decreased in about four days to a normal value of 0.6 mg./sq. cm./hr. (dotted curve P.I.), but rose again coincidentally with a clinical exacerbation elsewhere. A psoriatic patch (P.s.) at time 0 having a higher water vapour loss than unaffected skin showed a steadily decreasing water vapour loss comparable with normal healing of stripped skin in normal volunteers and unaffected by the clinical exacerbation when it started. Unaffected (N) and psoriatic skin (P.s. str.) of the patient were stripped with adhesive tape in such a way that the tape covered unaffected as well as psoriatic skin in the very same