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Disorders of the nervous system in malnutrition

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Disorders of the nervous system resulting from malnutrition have been recognized since the last century. Their occurrence during the recent war, among prisoners and populations subsisting on inadequate diets, stimulated at that time a great deal of interest, and the important monographs of Denny-Brown (1947) and Spillane (1947) contain a large number of clinical observations. There are, however, still very many problems to be solved in this field, even in the definition of the clinical syndromes which result from particular dietary deficiencies.

I think that today the important aspects of the subject to consider are those conditions which do occur in this country and which in the elderly might easily remain undiagnosed if the possibility of nutritional disorder were not entertained.

I would like to take first the abnormal neurological state thought to be due to nicotinamide deficiency, because it is the one which in the elderly, and in this country, could give rise to the greatest difficulty in diagnosis. It is probably uncommon here but is apparently seen much more frequently in the large cities of the United States among those addicted to alcohol.

It was first described by Jolliffe, Bowman, Rosenblum & Fein (1940) in New York, who defined a condition which would respond to treatment with large doses of nicotinic acid given parenterally while the patient was maintained on a diet low in vitamins of the B complex. The condition was named nicotinic-acid deficiency encephalopathy.

The onset of the illness is insidious and the nervous manifestations protean in character. The earliest symptoms are often psychological. Fatigue, irritability, depression or nervousness are all noted and it is easy to imagine how difficult the diagnosis can be at this stage. Later an impairment of intellectual function supervenes followed by stupor and coma. Many of the patients described in the literature were stuporose by the time they were admitted to hospital.

Associated with this clouding of consciousness there are other neurological signs. Rigidity of the limbs (of the sort seen in Parkinsonism) is found, and grasp and sucking reflexes are often present. The plantar responses may be extensor and the

tendon reflexes exaggerated, although an accompanying polyneuritis may complicate the picture. There may also be evidence of spinal-cord lesions, but at this stage it may be impossible to be certain of their presence, since the signs may well be overshadowed by those of the cerebral disturbance.

There are certainly changes found in the spinal cord at autopsy, when the posterior and lateral columns show degenerative changes. In the cerebrum there are changes in the cells of the cerebral cortex, most easily seen in the Betz cells. Signs of classical pellagra may be seen; a scaling erythematous skin rash, glossitis or diarrhoea.

When, as in this country, the condition is rare the diagnosis must be made so infrequently that the possibility is hardly ever considered. In the one severe case which I have seen, a diagnosis of multiple metastatic deposits in the nervous system was originally made. The patient was stuporose and the possibility of nicotinic acid deficiency was entertained because of an associated persistent diarrhoea, glossitis and a very mild scaling skin rash. She responded well to large doses of B-complex vitamins parenterally but I thought that an important feature of her case was the slowness in the improvement in her neurological state. In fact one was encouraged in persisting with vitamin B therapy only because of the dramatic and rapid cessation of the diarrhoea, which up to that time had been resistant to all forms of treatment. In this instance the deficiency had arisen in an elderly woman who had developed a severe depression after the death of her husband. After she had recovered she was able to say that she had eaten little but bread and butter, and had drunk only tea for over a year. The practical difficulties that may stand in the way of the correct diagnosis are well brought out by the fact that her daughters' account of the patient's diet, when the possibility of a deficiency disease was first considered, was quite inaccurate.

As I said earlier the first symptoms of this illness may be of a psychological sort. For this reason some psychiatrists ensure that all patients who present with depression, or in a state of confusion, are given large doses of B-complex vitamins. The relationship between such symptoms and vitamin deficiency may of course be very difficult to assess since, as in the case just mentioned, the psychological illness may lead to malnutrition which may then in its turn produce the signs of vitamin deficiency.

The second disorder which I would like to say something about is polyneuritis. There is no doubt that a lack of vitamins of the B group is the cause although there is still a good deal of doubt as to exactly which deficiency is responsible. Spillane (1947, p. 59) concludes, in his review of the literature on beriberi, that the outstanding deficiency is certainly of thiamine and many believe that this is true also of alcoholic polyneuritis. The objection has been raised, however, (Walshe, 1945) that the rate of recovery from polyneuritis has not apparently been accelerated by large doses of thiamine. Spillane himself was disappointed with the results of thiamine administration in the wartime cases that he studied. However, his criteria for assessing the effect of treatment were the length of stay in bed and the length of stay in hospital. He adds, however—and my experience on a very much smaller scale is similar—that the administration of thiamine led to the early relief of the pain

and muscular tenderness which is often a prominent feature of the condition. I think that it could well be that this is the important point. Large doses of thiamine may well initiate recovery rapidly, and the disappearance of pain and tenderness may be evidence that recovery has begun. The regeneration of the damaged nerve fibres once it has begun may well be independent of further treatment, and depend solely on the normal reparative processes of nerve regeneration which cannot be hurried by any means that we know.

From the clinical point of view I would be quite sure that the mild case of nutritional neuropathy could easily be overlooked. The early symptoms may be vague pains in the limbs and mild paraesthesiae, and the presence of slight degrees of muscular weakness might easily be missed by one not accustomed to testing power in a critical way. Absent ankle jerks which may be an early sign will of course be a helpful finding. Polyneuritic symptoms even of a mild degree, however, will be enough to cause a considerable amount of distress. What is more they may well be enough to immobilize a patient who is in addition elderly and frail.

Wernicke's encephalopathy, which is due certainly to a severe deprivation of thiamine, is uncommon in this country, and my experience of it here is confined to patients who are alcoholics or who are suffering from malignant disease with repeated vomiting. The clinical picture is a striking one with ocular palsies, nystagmus, ataxia and vomiting. There is mental confusion and in the severe untreated case clouding of consciousness. If the possibility is considered, the diagnosis can hardly be missed and the response to a dose of parenteral thiamine is very striking. The response is very rapid indeed as far as the ocular movements are concerned, which may become normal within 12 h. The mental symptoms are unfortunately much more resistant to treatment.

The last condition I would like to mention is nutritional amblyopia or nutritional retrobulbar neuritis as it is also called. My own experience is limited to three cases in this country, two of whom were alcoholics. The presenting feature is a failure of visual acuity which is slowly progressive over weeks or months. The optic discs may be normal but are also recorded as showing either pallor or hyperaemia. Field testing on the Bjerrum screen reveals scotomae in the central or paracentral region which increase in size as the condition gets worse. In some cases the response to treatment with B-complex vitamins is striking, and within a month there may be a remarkable improvement in vision. It was true in the cases I have observed, but they were all treated soon after the onset of their symptoms. In the cases which occurred in prisoners of war, however, in whom treatment was impossible or inadequate, there was often much residual disability. There are many much commoner causes of failure of central vision in the elderly and unless borne in mind this disease might well pass unrecognized.

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