dysentery was observed in German prisoners of war in the Middle East by Spillane and Scott (1945). Similar cases have recently been seen in India in British and Indian troops returned from captivity.

**Heat Stroke**

In India heat stroke was one of the principal medical causes of death amongst British other ranks. Improved methods of treatment have led to a lowering of the mortality rate, but as might be expected, some of the men who survived showed permanent sequelæ. The effects of heat stroke on the brain are at first generalized. On recovery from coma a variety of signs may be present, including severe mental confusion with incontinence, aphasia, pyramidal and cerebellar signs. As recovery proceeds the patient becomes orientated, ceases to confabulate, shows progressive improvement in memory, and is no longer incontinent. In short, the mental picture is very similar to that seen after a severe head injury. In a small percentage of cases this favourable course is not followed, a gross memory defect, both for recent and past events, persists and is accompanied by lack of interest, childish behaviour and incontinence. These signs of dementia vary in their severity and in the extent to which they eventually clear up. Inability to talk may be due to aphasia or to a gross dysarthria; both of these defects may be found in the same patient. Signs of a unilateral or bilateral hemiplegia clear up as a rule, although incompletely in some cases.

The most interesting sequel to heat stroke is a cerebellar picture. In July 1942 I examined three men in the Middle East who had recovered from heat stroke; all three were dysarthric and showed ataxia in their limbs, and unsteadiness in walking. None showed nystagmus. In two of these cases the signs cleared up in the course of a few weeks, but in the remaining case they persisted in association with signs of a gross dementia. In India I saw several similar cases, of which the following is an example:

Sergt. P., aged 40, on June 17, 1944, travelled 100 miles in a lorry west of Calcutta. He arrived in his destination in coma and was at once admitted to hospital where his rectal temperature was found to be 110°. Blood slides were negative for malarial parasites. He received energetic treatment for hyperpyrexia. On the evening of June 19 he recovered consciousness. He was moderately confused and incontinent. His speech was grossly dysarthric. He continued to improve slowly. On July 24 he appeared euphoric and showed involuntary laughter. He was well orientated and his memory was only slightly impaired for recent events. There was a rotary nystagmus, especially to the right. Gross ataxia was present in both upper and lower limbs and he was unable to stand unsupported. The jaw-jerk was increased and there were signs of mild double hemiplegia. The picture he presented was very similar to that seen in advanced disseminated sclerosis. Two months later his condition was virtually unaltered.

Similar cases have been reported by Weisenberg (1912), R. M. Stewart (1918), and recently by Freeman and Dimoff (1944). Stewart postulated a cortical cerebellar lesion in his case and Freeman and Dimoff reported a case fatal on the third day in which changes in the cerebellum were maximal in the region of the Purkinje cells. Although in the case I have cited nystagmus was present, as a rule it is absent. Confirmation of a cortical cerebellar atrophy in these cases has been found by Major L. Krainer, R.A.M.C., a neuropathologist working in India.

**REFERENCES**

McALPINE, D. (1945) Lancet (ii), 139.  

**An Unusual Form of Epidemic Food-Poisoning with Neurological Symptoms**

By Denis Brinton, F.R.C.P.

For thirteen months from October 1942 the population of Aden had been intermittently affected by epidemics of this food-poisoning. Perhaps 450 persons in all had suffered; and from the first (although the authorities did not learn this till much later) the natives had correctly blamed the Abyssinian wheat which had been shipped to Aden from the earliest harvest after the fall of the Italian East African territories, to save the better-quality Dominion-supplies, then very short for a number of reasons. According to the sufferers, this Abyssinian wheat was not only of poor quality, but contained a poisonous weed-seed, called in the local arabic miscara, literally to be translated as "tipsy", which you will see is a first-rate description of the effects it produces in man.
The natives insisted that this miscara was a common weed in every wheat-field of the Aden-peninsula also; and that, though they had never heard of any case of poisoning from the local crop, the greatest care had always been exercised by the farmers to remove the weed and its seeds from the wheat, at all stages from harvest to milling. In samples of Abyssinian wheat, they showed me how to recognize miscara, and in the hilum of many of these grains pointed to a black discoloration which under a hand-lens presented a mouldy appearance. Several samples of newly wharfed Ethiopian grain were collected and counted, and found to contain miscara-seeds in quantities up to 10%.

Reconstructing the clinical aspects of this food-poisoning by questioning both those who had suffered and the doctors who had had charge of them when ill (for the Governor had rightly frozen all stocks of the poisoned wheat a few days before I arrived, and the epidemics had come to an abrupt end), I learnt that, from one-quarter of an hour to two hours after taking food made of this grain, a man would become dizzy, and be smitten with headache, marked generalized tremors, lassitude, slurred speech, and a staggering gait. Sometimes, there had been vomiting and diarrhoea, and less commonly nausea and abdominal pain. Subsequent events seemed to vary directly with the amount of poison taken. If the subject had fed well, he paid for it by quickly subsiding into stupor or even a coma from which it might be impossible to rouse him for so long as ten hours; but, if he had taken only a little, his earliest symptoms would not noticeably increase and he might be fully recovered in three or four hours. There were no deaths, and within seventy-two hours even the most severely affected were well enough to resume their usual occupations.

Specimens of miscara sent home for botanical and chemical analysis revealed it to be a weed called the flax-darnel (*Lolium liniculum* or *temulentum*); and suggested that the poisonous element was in the mould to be seen on nearly every weed-seed, and was probably a pyridine-base of incompletely established chemical composition, known as temuline.

There were, however, certain paradoxes, necessarily slurred over in this bald statement of fact. First, it had to be established why Abyssinian wheat had proved poisonous to the inhabitants of Aden, though it had long been used in East Africa by the Italians, and more recently by us, without ill-effects. Secondly, I wished to find out how the natives of Aden came to have such an exact knowledge of miscara, although none could recall a case of poisoning from the local wheat, or indeed until the imports from Ethiopia began in 1942.

As to the first problem, a visit to a standard Italian mill in Eritrea provided the answer. Here, Ethiopian wheat, not less filthy, not less charged with miscara-seeds, was put through a complicated system of specially designed mechanical sieves before the wheat was milled. The resulting flour, long fed to the people of Eritrea and Abyssinia, was of course harmless; but Aden, without any sieving devices for cleaning the contaminated grain, had milled miscara with wheat and become poisoned.

As to the second paradox, the only explanation was the traditional usages of the small farmers of the Aden protectorates. No case of miscara-poisoning from locally grown wheat could be recalled, merely because from time immemorial scrupulous attention had been paid to the removal of the weed and its seeds from the harvested wheat. The miscara-plant, they said, is easily identified from the time when the crop has grown to a height of about one foot; it is a darker green than the wheat, and splits a short distance above the ground into five or six heads. Before the crop is cut, which in these extremely primitive communities is done by grasping a stool of stalks and severing them near their base with a curved knife, as much miscara as possible is weeded out; but some is unavoidably cut with the wheat, and is spread out with it in a clean place to dry in the sun. When these products of the harvest are dry, most of the grain is removed by a heavy stone drawn by oxen. Afterwards, what remains attached is hand-picked from the stalks, and all the free grain is then winnowed and housed. As wheat is required for food, the women take the grain from store and once more spread it in the sun. After it is warm, it is tossed up again and again in a straw-basket, so that light impurities such as husks are blown away. The same process flicks most of the miscara-seeds to the edge of the basket, whence they are readily removed. The residue is finally hand-picked, grain by grain, until all dirt, miscara-seeds, and other foreign bodies are separated. With these precautions, clearly applicable only to small holdings, local wheat can always be eaten with safety.

So, in essence, these epidemics of food-poisoning arose from the wartime marriage of primitive methods of agriculture to modern methods of distribution. If the Abyssinian harvest had been put through modern threshing machines, with their ordered winnowing arrangements, no seeds smaller than mature wheat-grains would have been exported to Aden, and the seeds of the flax-darnel would have been excluded. If the project could have been on a small enough scale to allow the hand-picking just described, clean wheat
Nutritional Disorders of the Nervous System in the Middle East

By J. D. Spillane, M.D.

Nutritional polyneuritis.—In the Middle East during 1942-43 Polish troops and refugees filled some of the camps and hospitals of Palestine and Israel during their evacuation from Russia. Most of them had escaped into or had been transported to Russia in the winter of 1939-40. For two years they endured considerable hardships there and after the German invasion many of them were transported across the Caucasus to Iran, Iraq and Palestine. I had the opportunity of seeing many hundreds of these unfortunate people. Dysentery, malaria and typhoid were very common among them. Many were tuberculous. Malnutrition was widespread. Especially in the officer class one observed considerable loss of flesh; glossitis, oedema of the ankles, chronic diarrhoea were very common. A few had scurvy. Many of them had symptoms and signs of polyneuritis. Two types of case were encountered.

In the first and largest group symptoms dated back some six or twelve months. Difficulty in walking was the earliest complaint. Sharp pains in the soles of the feet and in the calf muscles would arrest progress and force the patient to rest—as in claudication. Affected individuals found difficulty in keeping their feet warm, painful dysesthesiae distressed them, especially at night. Muscle tenderness, wasting, weakness of the extensors of the toes and feet, sensory loss and reflex disturbances subsequently made their appearance in many cases. In the second group the onset of paralysis was quite rapid and almost always occurred during or shortly after an acute infective illness such as malaria, dysentery or typhoid. And yet I can say that I never once saw, during three years, a case of polyneuritis in a British soldier during such infections. Post-diphtheritic and acute infective polyneuritis were readily excluded in these Polish patients. A few fatal cases had terminal signs of brain-stem involvement—diplopia, optical neuritis, paresis of the tongue musculature—and at post-mortem there were typical vascular lesions of the Wernicke type. But these haemorrhagic lesions in the brain-stem were sometimes found where no localizing signs had been demonstrated ante-mortem (in some cases after careful observation).

On one occasion brief examination of the nervous system of all patients in one hospital ward revealed that about 25% had reduced or absent knee and/or ankle jerks. The majority were suffering from the acute infections of the type mentioned. I came to the conclusion that the neurological disturbance was one of nutritional polyneuritis. Many of them were already on vitamin-B therapy and one was able to watch the progress of nearly all of them (60 cases) during the following six to nine months. Thiamine when given intramuscularly seemed to improve appetite, relieve the sharp pains in the legs and feet and possibly increased the sense of well-being of some of the patients. But it had no effect whatever on the period of incapacity. Over and over again initial improvement of a subjective kind was thought to herald definite objective cure. Occasionally one observed some shrinkage of the area of sensory loss, but restoration of muscular power and reflex activity were never hastened. Yet these are the very signs which denote morphological alteration in the peripheral nerves and if consistently reversed would indicate some specific healing processes in the vitamin.

Since then I have had similar experiences with native and European prisoners of war and internees, in the Middle East and in the United Kingdom, and observation of over 200 cases of polyneuritis in these malnourished individuals has provided ample data on which the general effects of thiamine therapy may be judged. I have no strictly controlled series of cases to substantiate my conclusions—but they were never necessary. Routine clinical observation of cases which received adequate treatment with thiamine, by all channels, or who were given courses of treatment lasting one or two weeks (20 to 100 mg. daily), showed quite clearly that they fared no better than cases who received no additional supplements of vitamin.

On this problem of the “antineuritic vitamin” it seems probable that there is a large body of unpublished experience which indicates the inertness of vitamin B, in the treatment of polyneuritis. If general experience in the last ten years had established the value of thiamine we should not now have to debate the problem. The acceptance of liver and insulin therapy was never so long delayed, and there is no reason to suppose that a cure for polyneuritis will not be recognized when it appears. I do not think my clinical experience is necessarily in disagreement with the demonstration by the bio-