states that his skin eruption was in every respect similar to that of the younger brother, and showed, as did the other, marked seasonal variations. The elder boy died early in 1911, and no *post-mortem* examination was obtained. It is hoped that a complete report of the first-mentioned case, together with the pathological findings, may be published in due course."

We believe that the information so far collected is sufficient to prove that for an undetermined but long time pellagra has endemically prevailed in parts of the United Kingdom, and that, notwithstanding the recognition and publication of two grave typical cases, the disease has been generally overlooked in its milder and more obscure forms.

The presence of pellagra in the British Islands is of geographic interest, because hitherto the disease was believed to be limited northwards by the forty-fifth parallel. Etiologically, its presence in the United Kingdom is of importance, because here its causation cannot be reasonably explained by the consumption of either sound or bad maize, and its topographic distribution shows that here, as elsewhere, it is linked to the swift-flowing, Simulium-infected streams. Sociologically, the disease calls for the most urgent attention. Pellagra is an insidious disease, either rapidly fatal or of long intermittent course, leading to insanity. The pellagrous psychical disturbances are as many-sided and as obscure as the somatic manifestations of the disease, but the salient feature is an intermittent and progressive amentia, often assuming a semblance of melancholia and exhibiting from time to time regular outbursts of maniacal excitement. In some cases there may be all the appearances of progressive paralysis. Dementia is the invariable termination, unless the patient be cured, or carried off sooner by some intercurrent disease or mere exhaustion of the vital powers.

A careful reading of the old case-records of our lunatic asylums will no doubt bring to light many cases of overlooked pellagra; but—and this is far more important—we trust that henceforth physicians will be on the look-out for this disease, and that they will make a thorough search for the milder forms which, as our experience shows, very easily escape detection.

However dormant it may be at the present time, a fatal, insanity-causing disease, such as pellagra is, cannot be allowed to continue unchecked in its progress, and it behoves us to take the matter very seriously in hand.

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MITRAL STENOSIS: PERIPHERAL EMBOLI CAUSING PARTIAL MONOPLEGIA WITH SYMPTOMS SIMULATING CEREBRAL EMBOLISM.

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THIS case is of interest because on both occasions on which embolism occurred in a limb artery the attendant symptoms accurately simulated those associated with cerebral embolism. Thus on each occasion the patient was the subject of partial monoplegia, with attendant nausea and giddiness.

Additional interest is afforded by the fact that death was absolutely sudden, and was found to be due to the presence of a ball thrombus in the right auricle.

The patient, a married woman aged 42, had had mitral disease, following an attack of rheumatic fever six years previously.

The first embolus affected the left upper limb. It occurred eighteen days before admission to hospital. The patient while stooping suddenly felt sick and giddy, and the left forearm and hand fell powerless to the side. She retched but did not vomit, and she saw dark spots in front of the eyes. The second embolus, affecting the left lower limb, occurred three days later. "Without warning" she again felt sick and giddy, again saw dark spots before the eyes, and simultaneously "lost power" in the left leg and foot.

She was recommended as a case of cerebral embolism to University College Hospital, where she was admitted under the care of Sir John Rose Bradford, to whom I am indebted for permission to publish this case.

Considerable trophic changes in the affected limbs caused some doubt of this diagnosis, and the real state of affairs was revealed on noticing that the left radial pulse was absent.

Condition on Admission.

The facial muscles showed no paralysis or weakness. The skin of the left forearm and hand was cold, dry, and shining, and blebs containing serous fluid were present on the tip of each finger. Movements at the left shoulder and elbow were weaker than on the right. Pronation was only possible through an angle of 45°; supination was weak, but complete. Flexion and extension at the wrist and of the fingers were both markedly limited. No pulsation could be felt below the first part of the axillary artery. In the left lower limb also marked trophic changes were present, and pulsation was absent from some of the main arteries. The skin below the knee was white, shining, and oedematous. It was colder than on the right side. Over the front of the leg was an oval erythematous area, 8 in. long, in the centre of which was a smaller oval area, 3 in. in length, where the skin was blue. Over all this area the skin was hyperaesthetic. There was a small patch of anaesthesia on the dorsum of the left foot.

There was no paralysis on the left lower limb. Movements were weaker than on the right side, but the impairment was due rather to pain evoked than to any actual loss of power. Both knee-jerks were present; there was no ankle clonus, and the plantar responses were flexor. Sphincter control was normal. Examination of the heart showed well-marked mitral disease.

Death.

The patient died suddenly four days after admission. She was leaning over the bed, when she quickly raised her head and shoulders, became intensely cyanosed, made a few stertorous efforts at respiration and then stopped breathing. She had spoken to a nurse a few minutes before and had then appeared perfectly well.

Necropsy.

The *post-mortem* examination confirmed the opinions formed as to the nature and origin of the lesions in the upper and lower limbs and also supplied the explanation of the sudden death.

There were no cerebral lesions to account for the loss of power in the limbs.

As the right auricle was being cut open, there fell out an oval *ante-mortem* clot 2½ in. in its longer diameter. The endocardium of the walls and valves was smooth and free from adherent clot, so that the thrombus lay quite free within the auricle. The mitral orifice was buttonhole in shape and only admitted the tip of one finger. There were no vegetations to suggest malignant endocarditis.

The upper part of the left brachial artery was blocked by a reddish-grey clot evidently of recent formation. Another thrombus filled the lower half of the left femoral and the whole of the popliteal arteries below. This thrombus was white and organized. Hence, seeing that the leg lesion was the more recent, there must have been an earlier embolus in the femoral artery which had caused no symptoms, and either an extension of this or a second embolus must have been responsible for the symptoms and signs affecting the leg as described above. Whatever may be the correct explanation of the second series of symptoms, the additional interference with the blood supply brought about the subsequent commencing gangrene of the skin covering the tibia.

Thus the symptoms of nausea and giddiness accompanying a partial monoplegia were on both occasions misleading. No lesion to account for these could afterwards be found in the central nervous system. The loss of power in both instances was due to the local anaemia. The nausea and giddiness are more difficult to explain. They may only have been expressions of a general circulatory disturbance consequent upon the occurrence of a sudden peripheral embolism, or possibly they were due to reflexes originated from the walls of the plugged vessel. In this case the patient would be expected to have noticed some pain in the limb at the time of the embolism. Pain, however, was absent on both occasions. Hence the exact cause of the nausea and giddiness must be left open to conjecture.