hopelessness and helplessness, accompanied by frequent crying spells. In the other four patients, unbearable insomnia was followed after a few months by frequent severe cephalgia, and later by feelings of hopelessness, worthlessness, and crying spells. At times most of them, due to their migraine, were forced to lie in bed in a dark silent room, as every little noise increased their discomfort. None of them had in the past suffered from depression or anxiety, and they had always been capable of carrying out their usual duties. Prior to their first visit to our department, they had been treated with analgesic or hypnotic drugs or both, with little or no relief.

Amitriptyline (25 mg, t.i.d.) and 5-hydroxytryptophan (100 mg, t.i.d.) was prescribed to all of these patients. The latter was preferred to L-tryptophan, as it crosses the brain/blood barrier more easily (Wurtman & Fernstrom, 1976). Amitriptyline was chosen for its known mild sedative effect. Each patient was checked on a weekly basis for the following three months, and in eight cases a mild improvement of symptoms was reported after four weeks of treatment. This became more marked at the end of the eighth week. Both migraine (or cephalgia) and insomnia had progressively decreased in frequency and intensity without the use of any analgesia or hypnotic drugs, and consequently, the patients' mood had also improved so that they could more easily carry out their usual duties.

At the end of the 12th week of treatment these eight patients claimed to be symptom-free. In the other two patients some amelioration of symptoms occurred, but they still needed to use analgesics or hypnotics from time to time, although less frequently than before.

In those cases where a complete recovery took place, the drug regimen was gradually reduced and then totally discontinued after six months of therapy. They were followed-up for a while at random, and no relapses were found. The other two cases followed the same treatment for over one year, but no further improvement was reported.

This drug regimen was implemented on the assumption that migraine and insomnia – and the subsequent depressed mood – could be due to a brain serotonin deficiency. The role played by serotonin in the genesis of migraine was first shown by Sicuteri *et al* (1961). Both Jouvet (1969) and Jouvet & Pujorl (1974) demonstrated the influence of serotonin on the sleep cycle, whereas Van Praag (1981) showed the usefulness of 5-hydroxytryptophan for the treatment of a depressed mood.

In all the cases reported, the symptom complex began either as a severe migraine or as troublesome insomnia. The depressed mood which occurred some time later was reactive to the discomfort provoked by the other two aforementioned symptoms, which are very likely due to a brain serotonin deficiency.

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A HUNDRED YEARS AGO

Tea Drinking and Nervous Disorders

Tea has a powerful action on the nervous system of some individuals. Dr Bullard of Boston believes that it may produce a chronic poisoning of the nervous centres, shown in increased excitability, due partly to direct action of the alkaloid on the nervous matter, and also indirectly by the production of gastric derangement. Taken, therefore, too frequently, even in moderate doses, it places the nervous system in a condition of greater vulnerability to slight external influences, and favours the development of functional neuroses, or helps to render them permanent. Whilst there is no evidence to show that tea causes organic changes in the nervous tissues, yet, if such exist, tea may readily aggravate some of the symptoms. Tea may act as an important factor in the causation of neuralgia, hysteria, and allied affections. When taken constantly in large doses, dyspepsia usually supervenes before irreparable harm is done to the nervous system. In hemicrania, and possibly some other functional neuroses, there is probably a craving for some stimulant, and tea is better than other equally accessible articles, and so it happens that many sufferers from megrim are great tea-drinkers.

Reference

The Lancet, 14 January 1988, 86.