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Cluster headache and melatonin

Sir—J N Blau and H O Engel (Sept 18, p 1001)[1] describe a new cluster-headache precipitant—increased body heat, from the environment, a hot bath, or central heating in 52 (26%) of 200 patients, and from exercise in 23 (12%) patients (three by sexual intercourse).

The causes of cluster headache are still unknown; the temporal pattern of the cluster periods suggest the involvement of central structures in particular the hypothalamus, which regulates circadian rhythms. The pineal gland through melatonin secretion plays a central part in the circadian organisation of biological rhythms. Evidence obtained in animals suggests that the pineal gland and melatonin may be related to the regulation of core body temperature. Dependent on the species considered, melatonin has a part in the generation of seasonal rhythms of daily torpor and hibernation, in heat stress tolerance, and in setting the core body temperature set point. In human beings, the circadian rhythm of melatonin is closely associated with that of core body temperature, the nocturnal decline of this temperature being inversely related to the rise of melatonin.[2]

Chazot and colleagues[3] reported lower melatonin concentrations in cluster-headache patients than in controls. Waldenlind and colleagues[4] also show lower concentrations in the cluster period than remission.

Increased body heat might precipitate cluster-headache attacks by alteration of melatonin concentrations, leading to hypothalamic dysregulation and chronobiological dysfunction. These findings also support a therapeutic option of melatonin in the prophylaxis of cluster headache.[5]

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Folate supplementation and neural-tube defects

Sir—L Abramsky and colleagues (Sept 18, p 998)[1] report a high rate of neural-tube defects (NTDs) in the UK population in 1996–98, despite a national campaign to encourage supplementation with folic acid in the prenatal period. Our data from a population at risk over the same period suggest that poor compliance with preconceptual or antenatal folate supplementation, in conjunction with a low folate intake, remains a major hurdle.

Mothers in the west of London have a high rate of NTDs, in particular those in the Indian or Pakistani population, in whom serum concentrations of folate are low.[2] A direct-questioning survey of 1357 women in a west London antenatal clinic from January to May, 1998, showed that 380 (28%) had not taken folic acid supplements before attendance at the clinic. 285 of these women were of Indian or Pakistani origin and 323 were primiparous. None of the women was aware of the benefits of the vitamin supplement. In 1998 there were no live deliveries of NTDs from the clinic, since all were diagnosed antenatally by ultrasonography. There were six terminations for NTDs; four from primiparous pregnancies, five in women of Indian or Pakistani origin (three with English as a second language). Only three women who had a termination had taken some antenatal folic acid. This lack of compliance with antenatal folic acid is not surprising because a UK study has shown that if English is a mother's second language, the idea of antenatal care is not aways understood.[3]

There are no other obvious factors other than folate deficiency that might increase the rate of NTDs in the west London population. None of the families with an NTD in 1998 was consanguineous. There was no indication of a poor zinc intake, and the rate of mutations in the methylenetetrahydrofolate reductase gene are thought to be similar to that in Caucasian populations. Folate concentrations have proved lower by other investigators in London in the Indian or Pakistani ethnic group, suggesting that this should be the main target for treatment.[3]

How might this situation be improved? Intervention with food fortification with folic acid was effective in raising serum folate and reducing serum homocysteine in an American study.[4] This approach overcomes the complications of local dietary custom and periconceptual planning, but it may be difficult to ensure an adequate intake of folic acid in all mothers. We propose implementing a local strategy with both food supplementation of specific foods and an educational campaign. A lack of behavioural change after a recent folate promotion campaign has been documented in Virginia, USA, so the difficulties in London are not unique.[5] The public-health approach to recent folate supplementation is important as a cardioprotective measure and a method to reduce NTDs; it merits careful consideration and more debate.

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