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Correspondence

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LITHIUM PROPHYLAXIS OF PERIODIC HYPERSOMNIA

DEAR SIR,

For the treatment of periodic hypersomnia, with or without polyphagia, stimulants such as amphetamine or methylphenidate are commonly prescribed. However, the response is not always favourable, and side effects may be significant. Female patients with periodic hypersomnia appearing in the depressive phases of an affective disorder have been successfully treated with lithium (1–3). Recently Ogura *et al* have reported the successful prevention with lithium of periodic hypersomnia without polyphagia in a 17-year-old boy, who showed hypomania of only slight degree immediately following hypersomniac phases (4). Lithium abolished hypersomniac episodes which had come periodically once a month for the previous four months. Discontinuation of lithium was followed by a recurrence of another period of hypersomnia.

The present report is about a patient with periodic hypersomnia unaccompanied by disturbances of mood or appetite, in which recurrence of the episodes was successfully prevented with lithium.

The patient is a 15-year-old male, son of a senior civil servant. His mother's maternal uncle was once admitted to a mental hospital in middle age with a diagnosis of depression.

Early in March 1975, the patient passed an entrance examination to a high school for which he had been studying hard during the preceding semester. No other significant events or 'flue-like' symptoms were noted in the three months preceding the first onset of hypersomnia. On 25 May he slept until the afternoon and thereafter, for the following 10 days, he slept 15–20 hours a day, and on awakening in the afternoon ate only one meal a day and slept again. The amount of food consumed was about the same as usually eaten at supper, and there was no increase in appetite. The sleep in these days was just like his normal sleep and he could be awakened but soon fell back into sleep. There was no bladder or sphincter disturbance. There was only transient drowsiness when awake, and no depressive symptoms or irritability were noted. After this period of hypersomnia his daily behaviour was back to his usual standard, and there were no hypomanic manifestations either. Further episodes of hypersomnia recurred, as shown in Fig 1.

On examination, he was a slightly built, cooperative, alert and well organized boy. There were no significant neurological finding. EEG, ophthalmoscopy, plain skull X-ray and renal, liver and thyroid function tests, and serum electrolytes revealed no abnormalities. His IQ was 127. Orthostatic albuminuria was the only significant finding.

From 18 September, lithium carbonate 600 mg daily was administered but hypersomnia recurred in the beginning of October. Since the serum lithium level was 0·32–0·4 meq/L before this episode, the dosage was increased to 800 mg daily from 14 October. From 27 October to 3 November the patient noted feelings of depersonalization and asthenia, and his father reported slight under-responsiveness. As this episode was considered a milder manifestation of his recurrent process, the dosage was increased to 1,000 mg daily from 4 November; it was back to 800 mg daily from the middle of December. No further episodes of hypersomnia or depersonalization occurred until the end of March 1976, ten days after the discontinuation of lithium.

Since the discontinuation of lithium was followed each time (in March and July 1976) by relapse of hypersomnia, lithium was considered effective in preventing hypersomniac episodes in this patient, and thereafter the serum lithium level was kept at about 0·6 meq/L by 800 mg a day regimen.

This patient's usual behaviour (as a diligent student) was in such contrast to the episodes during which he slept most of the day, that the effect of lithium was evident to all his family. The placebo effect is an unlikely explanation, as he had been given various drugs without effect before he was referred to us, and as lithium was also ineffective at the beginning of the treatment when the serum level was below 0·4 meq/L. The clear effect of lithium in this patient, together with a previous report by Ogura *et al* (4) suggest that lithium is also effective in preventing hypersomniac episodes unaccompanied by manic-depressive symptoms. In view of a previous

report of the effectiveness of lithium in a case of hypersomnia accompanied with polyphagia occurring in an adolescent female (2, 3), I would expect the drug to be effective in typical Kleine-Levin syndrome, but this is yet to be confirmed.

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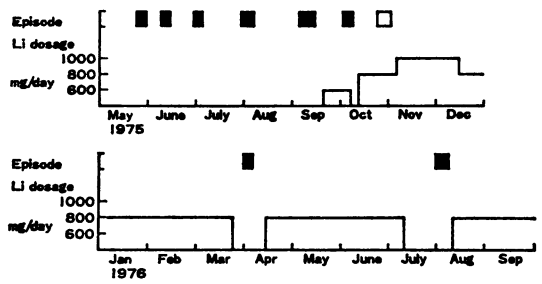

Fig 1—Hypersomniac episodes (shaded boxes) and lithium dosage. The unshaded box at the end of October 1975 refers to an episode of depersonalization.

PRENATAL PROGESTERONE AND EDUCATIONAL ATTAINMENTS

DEAR SIR,

The paper of Katherina Dalton (*Journal*, Nov. 1976, 129, 438–42) has received so much advance publicity, and presents such unexpected findings, that it deserves the closest scrutiny.

It essentially makes two claims: one that the effects of toxaemia in the mother and the intelligence of the child can be reversed by progesterone; the second, even more remarkable, that progesterone will increase the intelligence of the subsequent child above normal. Her results, however, do not warrant these conclusions.

The first claim would require that the mothers treated for toxaemia of pregnancy by progesterone were compared with an identical group treated with placebo injections. These conditions appear to have been satisfied with the first study reported in the *Journal* in 1968 ('Antenatal progesterone and intelligence', 114, 1377–82), but the only statistically significant result ($P = <\cdot05$) was that the progesterone children were more frequently walking at six months. The current study, however, gives insufficient information to support the first claim, let alone the second, largely because insufficient information is given about the control group. Progesterone mothers are likely to have been an unusual group to have opted for progesterone injections in the 1950s, 11 of them within the first trimester. It is not altogether fanciful to assume that they were both more open-minded and more concerned about the future health of their children than the controls, who were picked at random from obstetric wards or the General Practice Register. Not only is no evidence cited for the equivalent intensity of toxaemia for the toxaemic controls, but no comparison is made about any of the controls and the progesterone mothers, except to say that they belonged to classes 3–5.

Having, however, selected what one must hope are comparable controls, Dr Dalton uses the chi-squared test which distinguishes the groups qualitatively rather than quantitatively, but she does not quote the cut-off point used to divide the groups. More importantly, she assesses the three groups together, thus allowing the generally greater difference between the toxaemic controls and the progesterone group to obscure the significance, or lack of it, between the normal controls and the progesterone mothers. (To be fair, the only statistics where figures are provided 'Entrance to university'—actually distinguishes the progesterone group from the normals more significantly, $\chi^2 = 9\cdot53$ and $P = <\cdot01$). Under these circumstances, the presence of two more controls than can be accounted for by the double matching of four is merely a quibble.

It is all too easy to destroy exciting findings by over-zealous criticism. Nevertheless, it is sad that such an interesting paper should have been published in its present form, and we hope Dr Dalton will furnish us with sufficient details to confirm her remarkable claims.

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DEAR SIR,

In her article, Dr Katharina Dalton states that 'progesterone given to the mother (antenatally) not

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