were just significantly higher than those obtained at 30 minutes after either an eight-hour or a 16-hour fast. Nevertheless, the subsequent values obtained after the four-hour fast were similar to those after the other periods of fasting. Thus in the biological sense there was no impairment of carbohydrate tolerance after the four-hour fast.

We think that waking the patients at 0500 in the study of Walsh *et al*[1] may have influenced the results they obtained. We suggest that in further studies hormone concentrations, particularly those of plasma cortisol, should be measured in addition to blood glucose concentrations. We think that our results indicate that by itself the duration of the pretest fast does not affect tolerance of oral glucose.

[1] Walsh, C H, O'Regan, J, and O'Sullivan, D J, *British Medical Journal*, 1973, **2**, 691.
[2] Jarrett, R J, and Keen, H, *British Medical Journal*, 1969, **2**, 341.

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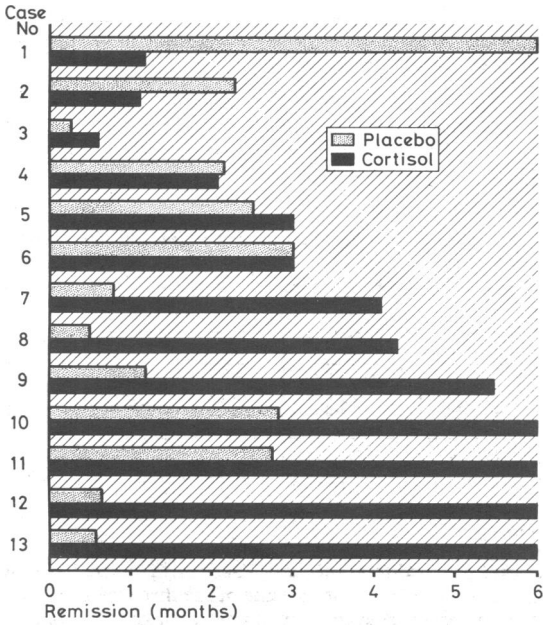
Idiopathic nephrotic syndrome: prevention of early relapse

In children with the idiopathic nephrotic syndrome early relapses are often due to adrenocortical suppression after prednisone.[1 2] We have shown that such early relapses can be avoided by partial cortisol substitution.

Patients, methods, and results

Eight boys and five girls (aged 4·7-14·6 years) who had had glucocorticoid-sensitive idiopathic nephrotic syndrome for up to 10·3 years were studied after their parents had given informed consent. The clinical definitions and prednisone regimen have been described elsewhere.[3] A two-hour ACTH test[4] was performed 1-12 days after treatment. Children with subnormal responses[4] were allotted to group 1 (drug sequence A-B) or group 2 (sequence B-A). Drugs A and B were tablets of identical appearance and taste, A containing 5·0 mg cortisol and B without the active agent. Their identity was not known to anyone concerned in the study. Children weighing 30 kg or more took two tablets at 7 am and one tablet at 2 pm; smaller children were given half the dose. When infection or detectable proteinuria (urine was checked at home every morning with Albustix) occurred the daily dose was doubled and given in equal parts every six hours over the next three days or until the symptoms had disappeared. The first drug was continued for six months or until relapse. As soon as the child again had post-prednisone adrenocortical suppression after a relapse, the second drug of the sequence was started. In six children the ACTH test was performed on completion of each drug period.

Twenty-one of the 26 drug periods had to be interrupted because of relapse (see figure). At three months eight children remained in remission during the cortisol period only, one child in the placebo period only, and one child during both periods. The difference was significant at the 5% level ($\chi^2 = 4·0$). At six months five remissions continued, four of them during the cortisol period. Three of these children were followed for a full year and all relapsed. The fifth six-month remission was observed in case 1 during a placebo period; subsequent 2·4-year follow-up indicated that the idiopathic nephrotic syndrome had been cured. The following variables were compared between the cortisol and placebo periods with Student's paired *t* test: the extent of pre-prednisone proteinuria; pre- and post-prednisone plasma concentrations of albumin, protein, cholesterol, and urea nitrogen; length of prednisone treatment; and post-prednisone and post-substitution ACTH test responses. No differences were significant. The effectiveness of cortisol substitution was independent of age and sex.



Length of remissions in 13 nephrotic children with post-prednisone adrenocortical suppression. Periods of partial cortisol substitution and placebo medication are compared.

Comment

The results of this double-blind cross-over study indicate that partial cortisol substitution prevented half the relapses predictable within three months of remission. This supports the hypothesis that post-prednisone hypocortisolism is responsible for many of the early relapses in the idiopathic nephrotic syndrome.[1 2] Many frequent relapsers are in a continuous cycle of relapse-prednisone treatment-adrenocortical suppression-relapse.[2] Children with adrenocortical suppression should be identified and temporarily provided with partial cortisol substitution. This has to be partial; otherwise the suppression will persist. Our present regimen seems to be appropriate.

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Persistent orthostatic hypotension after epidural analgesia

Some degree of hypotension normally occurs during epidural blockade, but is transient. In the case reported here, however, severe orthostatic hypotension persisted for many weeks. This complication of epidural analgesia has not been reported.

Case report

A 26-year-old primipara was admitted ten days after the expected date of delivery. She had been well throughout pregnancy, with normal blood pressure, no postural symptoms, and no evidence of gestational diabetes. Labour was induced by artificial rupture of the membranes, and soon after the patient requested epidural analgesia. A catheter was inserted through a Tuohy needle at the level of L 2-3, the epidural space being identified by loss of resistance. The anaesthetist was experienced in the technique, and the dura was not punctured. A test dose of 2 ml lignocaine (1·5%) was administered with the patient in the left lateral position, followed by a further 10 ml.