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 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.

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 4-14 years) who had had glucocorticoid-sensitive idiopathic nephrotic syndrome for up to 10 years were studied after their parents had given informed consent. The clinical definitions and prednisone regimens have been described elsewhere.3 A two-hour ACTH test4 was performed 1-12 days after treatment. Children with subnormal responses5 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 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 Albusit) 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 ($p^2=4.0$). 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.

Comment

The results of this double-blind cross-over study indicate that partial cortisol substitution prevents half the relapses prior to 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-adrenal 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.

We thank the Sigrid Juselius Foundation for generous support and AB Medica OY, Helsinki, for the drug preparations.

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 L2-3, 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.
Dengue shock syndrome in Jamaica

In South-east Asia and the western Pacific severe dengue characterised by shock and haemorrhage has become a major cause of death.\(^1\) In a current epidemic in Jamaican dengue shock syndrome and dengue haemorrhagic fever have been seen for the first time.

<table>
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<th>Case No</th>
<th>C1q</th>
<th>C4</th>
<th>B</th>
<th>C3</th>
<th>Cd1</th>
<th>C5</th>
<th>CH50 Units</th>
<th>Transferin (μg/L)</th>
<th>IgG (μg/L)</th>
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<td>58</td>
<td>98</td>
<td>86</td>
<td>66</td>
<td>12-0</td>
<td>2-5</td>
<td>3-3</td>
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<td>12-0</td>
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<tr>
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<td>58</td>
<td>48</td>
<td>74</td>
<td>66-124</td>
<td>2-5</td>
<td>3-3</td>
<td>11-0-180</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(^{*}\)Normal values for our laboratory. \(^{\dagger}\)C3d was measured in EDTA plasma by immunoelectrophoresis.  

Serum protein concentrations (radial immunodiffusion assay) in cases 1 and 2. Concentrations of complement components C1q to C5 are expressed as % normal human pooled serum

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**Comment**

**Case 1**

A 42-year-old woman was admitted to hospital with a three-day history of fever, rigors, headache, retro-ocular, chest, and back pain, vomiting, and fainting. She was confused and restless, her temperature subnormal (35.5°C), and the periphery cold and clammy. Her pulse was 112/min and weak, blood pressure 90/0 mm Hg, and respiratory rate 40/min and shallow. Central venous pressure (CVP) was 0.2 cm H\(_2\)O.

An electrocardiogram showed low voltages, S-T depression and T-wave inversion. Haemoglobin was 15.7 g/dl (subsequently 11.3-3 g/dl) and blood urea 13 mmol/l (78 mg/100 ml) (subsequently 3-6 mmol/l (22 mg/100 ml)) suggesting considerable haemoconcentration. White blood cell count (WBC) was 15.4 x 10\(^9\)/l (neutrophils 83%); platelets, prothrombin time, partial thromboplastin time were normal; aspartate aminotransferase (SGOT) was 971 U/l and alanine aminotransferase (SGPT) 40 IU/l and alkaline phosphatase normal. There was proteinuria (0.05 g/24 h). Sputum, urine, and blood cultures were negative. Dengue haemagglutination inhibition (HAI) was 1/160 on day 5 and 1/640 on day 19. The table shows the complement profiles.

With intravenous fluid therapy the blood pressure and temperature rose to 110/70 mm Hg and 37.9-38.5°C respectively after three days. On day 6 a desquamating rash appeared on the face and neck and pulmonary oedema developed, requiring treatment with frusemide. Further progress was uneventful.

**Case 2**

A 30-year-old woman was admitted with a 10-day history of fever, headache, retro-orbital pain, generalised myalgia, and bone pain. Transient improvement after seven days was followed by episodes of fainting, vomiting, and a spinal fluid pressure rising to 84 mm H\(_2\)O.

When she was seen in circulatory failure, the temperature 36°C, pulse 100/min, blood pressure 70/60 mm Hg, respiratory rate 28/min, and CVP 0 mm H\(_2\)O. She was jaundiced and her abdomen distended, with tenderness in both upper quadrants, rebound tenderness, and shifting dullness. There was tender hepatomegaly of 3 cm.

Haemoglobin was 14.7 g/dl, falling to 9.5 g/dl in five days; WBC 17.9 x 10\(^9\)/l (neutrophils 76%); platelets 146 x 10\(^9\)/l; prothrombin time 22 s (control 12.5 s); partial thromboplastin time 48 s (control 39 s); urea 7.2 mmol/l (43 mg/100 ml), falling to 4.0 mmol/l (124 mg/100 ml) over five days; bilirubin 86\(\mu\)mol/l (5 mg/100 ml); alkaline phosphatase normal; SGPT 120 IU/l, urinary amylase 869 IU/24 h, serum protein 64 g/l, albumin 29 g/l, globulin 35 g/l, ascitic fluid protein 39 g/l, albumin 20 g/l, and globulin 19 g/l. Urine and blood cultures and leptospira agglutination titres were negative. Dengue H1 was 1/640 at day 10 and on day 22. Complement profiles are shown in the table.

She was treated with intravenous fluids, plasma, metoclopramide, and pethidine. Nasogastric aspiration yielded "coffee-ground" fluid. Her temperature rose to 39-35°C on the day after admission and remained for five days. The abdominal tenderness and ascites resolved over seven days, but upper abdominal pain continued for two weeks. She had diarrhoea on the second and third days in hospital. Weakness and faintness also continued and her blood pressure was subnormal until the third week.

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**References**


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