the unpredictable and variable course of acute mountain sickness.

This study has shown that dexamethasone may be used not only prophylactically, as shown by Johnson *et al*,[6] but also to treat fully established acute mountain sickness. Johnson *et al* observed a reduction in the diameter of retinal arteries and suggested that dexamethasone reduced cerebral oedema, and though we did not find a significant reduction in the diameter of retinal vessels, the pronounced reduction in cerebral symptoms, such as headache, suggests that such an effect is probable.

The reduction in symptoms in the dexamethasone group coincided with an increase in arterial oxygen saturation and a small improvement in spirometric variables. As the minute ventilation did not change the improvement in arterial oxygen saturation might have been due to a reduction in postulated interstitial pulmonary oedema. The weight loss in patients who received dexamethasone supports this theory and might be the result of a more general mobilisation of oedema fluid accumulated during the development of acute mountain sickness.

Though the potential long term effects of dexamethasone may be neglected during short term administration, this treatment should be reserved for emergencies to facilitate safe descent. Patients who improve with treatment should be discouraged from ascending further.[10,11] Severe acute mountain sickness may be avoided in most cases by slow ascent and by taking rest days when early symptoms occur or, if these progress, by descent.[7,12]

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SHORT REPORTS

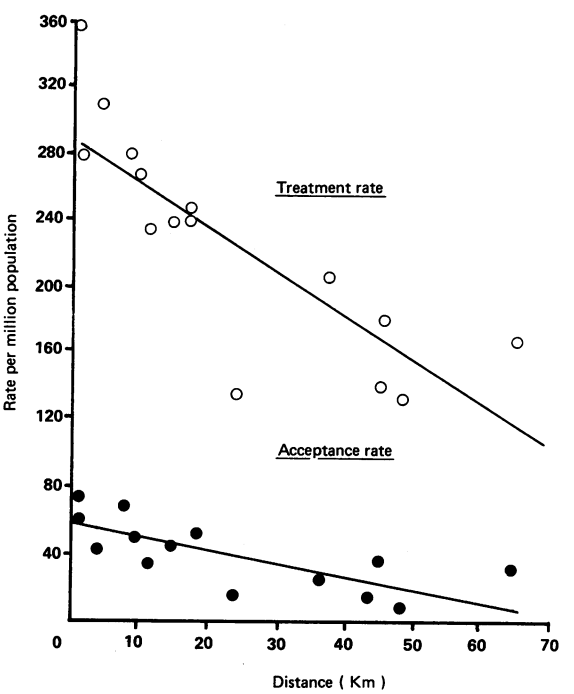
Intraregional variation in treatment of end stage renal failure

Though much has been written on the international and national variations of acceptance and treatment rates for end stage renal failure,[1,2] more local data are not routinely available because the European Dialysis and Transplant Association Registry does not collect information by the area of residence. We report the results of a survey carried out by one region.

Patients, methods, and results

In 1984 information was obtained on the mode of treatment and area of residence of all patients living in the North West Thames region who were admitted to treatment for end stage renal failure during 1983 and were still alive and having treatment at 31 December 1983. Information was collected from the units within the region and from units within neighbouring regions. The district specific acceptance and treatment rates adjusted for age and sex were calculated by using the 1983 final population estimates published by the Office of Populations, Censuses, and Surveys. The distance in kilometres (as the crow flies) of all electoral wards was weighted by population to give the average distance for each health authority from the nearest unit. Spearman rank correlation tests were carried out to investigate the relation between the distance from the provision of service and treatment and acceptance rates.

The district specific acceptance rate ranged from 8 to 73 per million population and there was a similar differential pattern for the district specific treatment rate, from 134 to 357 per million population (figure). Significant rank correlations were found between the distance from a renal unit and the age/sex adjusted acceptance ($r=0{\cdot}68$, $p<0{\cdot}01$) and treatment ($r=0{\cdot}89$, $p<0{\cdot}002$) rates for end stage renal failure.



Treatment and acceptance rates for end stage renal failure by district health authority with distance from nearest renal unit.

Comment

Treatment for end stage renal failure is traditionally provided in teaching hospitals in Britain. This may result in patients having to travel considerable distances. The distance as the crow flies was used as the measure of service access; evidence suggests there is little to choose between it and alternative measures.[3] The survey did not include the private sector; nevertheless, few British residents receive treatment for end stage renal failure outside the National Health Service. Possibly there is a higher incidence of the disease in West Indian and Asian people.[4]

The numbers involved in the acceptance rate calculation are small; the pattern is very similar, however, for the rate of patients alive with end stage renal failure per million population on 31 December 1983. This suggests similar differential acceptance rates over a period of time. Our results suggest that the further one lives from a dialysis centre the less likelihood there is of receiving lifesaving treatment. These findings have not been noted before.

One reason for these differentials may be different referral practices by general practitioners and hospital consultants.[5] Possibly the further hospital consultants and general practitioners are from a dialysis centre the less likely they will be to have up to date information about the methods of treatment, such as the use of continuous ambulatory peritoneal dialysis and the move

towards treating end stage renal failure in more elderly people and those with underlying diseases such as diabetes, and the less skilled they will be in diagnosing chronic renal failure. These findings suggest that intraregional variation should be examined more closely, as the findings may have implications for the organisation of treatment for end stage renal failure in the National Health Service.

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Loperamide toxicity in a child after a single dose

Loperamide has been used in Britain for diarrhoea since 1975. Respiratory depression and coma may occur after overdosage[1] and long term therapeutic use[2] and have been shown to be reversible by injection of naloxone.[1] So far as we know the following is the first report of opioid toxicity after a single therapeutic dose.

Case report

A 15 month old girl weighing 8 kg was admitted to hospital after accidental scalding, superficial burns covering 35% of the body area. She was rehydrated with intravenous fluids, treated with flucloxacillin and penicillin, and on day 5 transferred to a plastic surgery unit for assessment. At the time she was taking fluids and had copious green watery diarrhoea. Clinical examination showed no other abnormality and she was well hydrated. Results of investigations were: haemoglobin concentration 94 g/l, urea and electrolyte values normal, stools negative for reducing sugars and culture, urine culture negative. The diagnosis was diarrhoea as a stress response to burns.

On day 9 she was still having the diarrhoea, and at 1330 she was prescribed an initial 1 mg oral dose of loperamide. Fifty minutes later she was collapsed, pale, and unresponsive to pain; pulse was 120/min and respiratory rate 14/min. She had not vomited or convulsed. She was resuscitated with oxygen by Ambu bag and given 0·3 mg naloxone intravenously. By two minutes conscious level was improved and respiratory rate 30/min. Next day she was still drowsy. Blood values were: haemoglobin 87 g/l, urea and electrolytes normal, alanine aminotransferase activity 327 U/l, total protein 36 g/l, albumin 11 g/l. She was transfused 200 ml whole blood and 100 ml plasma protein fraction. Conscious level was normal on day 11. Serum alanine aminotransferase activity was 76 U/l, total protein concentration 48 g/l, and albumin concentration 18 g/l. The diagnosis was changed to cows' milk protein intolerance, as the diarrhoea resolved on withdrawal of milk.

Apart from antibiotics she received papaveretum 1·5 mg intravenously on eight occasions on days 1-3 and five doses of morphine elixir 2·5 mg on days 3-5 without ill effects. No opioids had been given within four days of the reaction to loperamide. At that time she was receiving up to 360 mg paracetamol syrup daily and had had a single dose of 200 mg chloral hydrate the previous evening.

Comment

The 1 mg dose used for this child (0·125 mg/kg) may have been greater than necessary, as the manufacturer's data sheet recommends the dose for children aged 4-8 as 1 mg every six hours until diarrhoea settles, and *Martindale* states that loperamide should not be used in the very young.[3] Doses in clinical trials have ranged from 0·045 to 4·0 mg/kg/day with few side effects, though convulsions occurred in a 4 month old infant treated for 11 weeks, the dose being 4 mg/kg/day over the previous week,[4] and possible ileus after a single dose of 0·045 mg/kg in a 1 year old.[5]

The reason for toxicity in this case is unclear. The low serum protein concentration may have been contributory (loperamide is 97% protein bound) and absorption may have been increased by damage to the gut wall. The raised serum alanine aminotransferase activity suggests that a temporary hepatic disturbance might have impaired handling of the drug.

As loperamide is available over the counter, doctors should be alert to the possible hazards of accidental ingestion of this drug by small children and the specific treatment that may be required. Naloxone appears to be an effective antidote. Reports to the National Poisons Unit suggest that though this drug causes symptoms in most cases of accidental overdosage, serious toxicity is rare.

We thank Dr N R C Roberton and Mr R C Campbell, of Addenbrooke's Hospital, Cambridge, for permission to report this case.

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Insulinoma unmasked by the Cambridge diet

Hypoglycaemia is not usually a feature of adherence to very low energy diets. We describe a patient who, for the first time, developed symptoms attributable to hypoglycaemia within three days of starting the Cambridge diet and was subsequently found to have an insulinoma.

Case report

A previously fit 46 year old man (height 179 cm, weight 82 kg, body mass index 25·6) was referred to the neurology department by his general practitioner because of a history of intermittent unsteady gait, slurred speech, and intellectual impairment which began three days after he started a self prescribed very low energy diet (the Cambridge diet; 1·38 MJ/day. His wife described how, while out walking, he began to stagger and his speech became slurred. The patient described feelings of unsteadiness, weakness, and intoxication. The symptoms completely resolved within 30 minutes of eating a light meal. Symptoms recurred two days later while he was still taking the Cambridge diet and again resolved after a light meal. Two days later he awoke confused and disorientated, wide eyed, and smiling inanely. On attempting to rise he staggered about the room. His symptoms resolved on eating breakfast. Physical examination showed no abnormality and investigations for spontaneous hypoglycaemia were initiated.

A random plasma glucose estimation while the patient was symptom free was 7·1 mmol/l. Next morning, after a 15 hour fast, the plasma glucose concentration was 2·6 mmol/l but unaccompanied by symptoms. The fast was continued with exercise. After 22 hours without food his responses to intellectual testing were slow and an electroencephalogram showed slight slowing of the α rhythm to 9 Hz during overbreathing. Intravenous injection of saline given as a control produced no change but 25 g glucose given similarly restored his mental state and returned the α rhythm to 10 Hz. The preinjection plasma glucose concentration was 1·6 mmol/l, β-hydroxybutyrate concentration less than 0·02 mmol/l, immunoreactive insulin concentration 55 mU/l, and C peptide concentration 3·6 μg/l. The finding of inappropriately high plasma insulin and C peptide values and suppressed β-hydroxybutyrate concentration in the presence of hypoglycaemia was highly suggestive of insulinoma. Computed tomography showed nothing abnormal.

At laparotomy a well encapsulated tumour 1 cm in diameter was removed from the posterior aspect of the head of the pancreas. Immunohistologically the tumour contained many insulin and a few somatostatin containing cells. Recovery was uneventful. Five days postoperatively the overnight fasting plasma glucose value was 7·1 mmol/l. Immunoreactive insulin (6·7 mU/l), C peptide (3·3 μg/l), and β-hydroxybutyrate (0·12 mmol/l) concentrations were all appropriate for the