urea in the stomach to produce ammonia. We therefore postulate that ammonia increases the pH in the mucus layer that overlies the gastric antrum, resulting in impairment of the normal inhibition of gastrin release by intraluminal acid.

This is the first report of a fall in postprandial plasma gastrin responses after eradication of *H pylori* in patients with duodenal ulcers. This could explain the reduced likelihood of relapse of ulcers in patients treated with agents that suppress or eradicate *H pylori*.¹ The fall in postprandial plasma gastrin concentrations, which does not occur after treatment with H₂ antagonists⁵ and is therefore not simply a result of the ulcer healing, would be expected to result in a diminished postprandial secretory drive on the gastric parietal cells. Another probable long term result of the lower gastrin concentrations is a reduction in the number of parietal cells owing to a reduction in the trophic effect of gastrin on these cells. Peak acid output, which reflects the parietal cell mass, did not fall significantly in our patients after one month of

treatment to eradicate *H pylori* despite the lower gastrin concentrations. Thus a longer period of treatment may be needed before the number of parietal cells falls.

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Erythrocyte zinc concentrations in subacute (de Quervain's) thyroiditis

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Patients with subacute thyroiditis classically present with fever, neck pain, and a tender goitre. Functional changes in thyroid state include both hyperthyroidism and hypothyroidism. Constitutional symptoms may, however, be absent and patients may present with features of hyperthyroidism alone, thus making differentiation from other causes of hyperthyroidism difficult. Nevertheless, differentiation is important as the implications for treatment are different. In such cases measurement of the uptake of radioiodine by the thyroid gland is useful, but it may not be measured unless thyroiditis is suspected. Thus the disorder may be misdiagnosed and patients given inappropriate treatment.

As patients with subacute thyroiditis are hyperthyroid for a short time a marker that reflects the duration of their hyperthyroidism might be useful in differentiating their condition from established hyperthyroidism—for example, that due to Graves' disease. Erythrocyte zinc concentrations are decreased in patients with established hyperthyroidism.¹ Because, however, the circulating life span² of erythrocytes is long, the erythrocyte zinc concentration reflects the integrated functional state of the thyroid over a period of months. Thus the erythrocyte zinc concentration may be used to differentiate between transient hyperthyroidism and established hyperthyroidism lasting more than two to three months. We previously reported that the erythrocyte zinc concentration differentiated between transient hyperthyroxinaemia associated with hyperemesis gravidarum and pre-existing thyrotoxicosis,³ and we report the results in patients with transient hyperthyroidism associated with subacute thyroiditis.

Patients, methods, and results

We studied five patients (four women and one man ranging in age from 35 to 42) with clinically incontrovertible subacute thyroiditis. Hyperthyroidism was

suspected clinically in four of them and was confirmed in all five by measuring the concentrations of thyroid hormones. All five patients presented with fever, anterior neck pain, a tender goitre, and a raised erythrocyte sedimentation rate (mean 111 mm in the first hour, range 58-150); the uptake of radioactive iodine by the thyroid gland was subnormal in all five and none had thyroid antibodies.

Erythrocyte zinc concentrations were measured by atomic absorption spectrophotometry.⁴ The coefficient of variation between days was 5%.

Erythrocyte zinc concentrations in the five patients were compared with those in 30 patients with established thyrotoxicosis and 110 control patients who were euthyroid (figure). The mean erythrocyte zinc

Erythrocyte zinc concentrations in 30 patients with established thyrotoxicosis (▲) and five patients with subacute thyroiditis (●). Hatched area shows 2 SDs of mean in 110 euthyroid control patients

concentration in the patients with thyrotoxicosis (105 (SD 64) μmol/l red cells) was significantly lower than that in the euthyroid control patients (196 (50) μmol/l red cells, $p < 0.001$). The mean erythrocyte zinc concentration in the patients with subacute thyroiditis was, however, 255 μmol/l red cells (range 185.6-394.7) and not significantly different from that in the control group. One patient had an erythrocyte zinc concentration of 394.7 μmol/l red cells but he also had a thalassaemia trait with microcytosis.

Comment

The normal erythrocyte zinc concentrations in our patients show that their hyperthyroidism was transient and therefore provides additional evidence to support

the diagnosis of subacute thyroiditis. These findings and those in our previous report of normal erythrocyte zinc concentrations in pregnant women with hyperemesis gravidarum² show that the measurement of erythrocyte zinc concentration may be useful in distinguishing transient hyperthyroid states from established hyperthyroidism and might therefore be a valuable addition to the current range of thyroid function tests. Measurement of erythrocyte zinc concentration is fairly simple and provides information that cannot be obtained by conventional thyroid

function tests. It may alert the clinician to the correct diagnosis, particularly in patients with "silent" thyroiditis with associated thyrotoxicosis, and therefore avoid inappropriate treatment.

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Renal replacement treatment in patients with spina bifida or spinal cord injury

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Number of new patients with chronic pyelonephritis secondary to neurogenic bladder who started dialysis in west of Scotland and United Kingdom per million population, 1982-8

	West of Scotland	United Kingdom
1982	0.36	0.22
1983	0.73	0.31
1984	0.36	0.38
1985	0	0.33
1986	1.09	0.18
1987	2.18	0.16
1988	0.73	*

*Data not available.

Before 1980 few patients with spina bifida or traumatic paraplegia started dialysis in British renal units, probably because doctors had an unsubstantiated belief that their prognosis was poor¹ and because of a lack of resources to fund renal replacement treatment.² Since then, however, successful treatment of the elderly and diabetics with renal failure, two other groups of patients expected to have a poor prognosis, has been described.^{3,4} We report our experience of treating patients with renal failure due to spina bifida or spinal cord injury.

Patients, methods, and results

Fifteen of 883 adults starting dialysis in the west of Scotland from January 1982 to December 1988 had renal failure due to spina bifida or spinal cord injury. Data from the European Dialysis and Transplant Association's registry show the number of patients classified as having chronic pyelonephritis secondary to neurogenic bladder who started dialysis in the United Kingdom during this period (table). We studied the patients until the end of February 1989, a follow up of four to 78 months, and calculated cumulative survival by using actuarial analysis.

The patients (10 men, five women) were of mean age 27 (range 16-50) years at the time of first referral to a nephrologist. Twelve had spina bifida, two traumatic paraplegia, and one traumatic tetraplegia. Fourteen had been referred because of renal failure (12 with a serum creatinine concentration >500 µmol/l), and eight of these patients started dialysis within six months. In all cases renal failure was attributed to neurogenic bladder and recurrent urinary tract infections, but severe hypertension (seven patients), amyloidosis (two), shunt nephritis (one), and renal calculi (one) were identified as additional contributory factors.

Eleven patients were treated by haemodialysis (six at home) for a mean of 20 (range 2-53) months and four patients by continuous ambulatory peritoneal dialysis for 27 (3 to 55) months. Thirteen patients were considered suitable for transplantation after further urological assessment. Three of these patients received

transplants 31-58 months after starting dialysis; all grafts were functioning at follow up eight to 35 months after transplantation, serum creatinine concentrations being 61-124 µmol/l. Three patients died 12-35 months after starting dialysis; all had been treated by haemodialysis in hospital, and all deaths were related to infection. Cumulative survival rates were 93%, 93%, and 62% at one, two, and three years respectively.

Comment

This study shows that the outlook for patients with renal failure due to spina bifida or spinal cord injury is good in the short and medium term. Survival is better than that for diabetics with end stage renal disease in the region. Moreover, many of the patients can be treated by dialysis at home, and most are suitable candidates for renal transplantation. Previous reports of treating such patients are scarce, but our experience compares favourably with that in California, where survival rates at one and two years were 60% and 52% respectively.⁵

The data from the registry of the European Dialysis and Transplant Association suggest that a higher proportion of patients with spina bifida or spinal cord injury receive renal replacement treatment in the west of Scotland than in the rest of the United Kingdom. This suggests that non-nephrologists in our region are more aware than those in the rest of the United Kingdom of the value of treating renal failure in patients with paraplegia. Regrettably, these patients were often referred in advanced renal failure, and this has been observed in other groups of patients expected to have a poor prognosis. Our results indicate that all patients with neurogenic bladder should be referred to a nephrologist as soon as their blood pressure or serum creatinine concentration is raised, as we have not found any such patient to be unsuitable for renal replacement treatment.

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