($P=0{\cdot}01$), and a small but insignificant fall in the phenformin group. The two drug groups, however, were no longer matched for OGTT sugar area with the diet group at the third test. Since the OGTT insulin area is related to the sugar area, a fourth group of patients also treated with diet alone for six months, was used for comparison. This group had a mean OGTT sugar area similar to those of the two drug-treated groups. The mean OGTT insulin areas of these three groups did not differ significantly (table III). The significance of this cross-sectional analysis, however, was weakened by the large variations in individual insulin areas. The rise in insulin area detected by the more sensitive longitudinal analysis suggests a definite β-cytotrophic effect of glibenclamide but does not exclude an additional extrapancreatic mechanism.

Sulphonylureas produce a steep rise in plasma insulin levels when given acutely.[1] There is doubt, however, about whether this effect is important in long-term treatment. Sheldon *et al*[14] carried out repeat glucose tolerance tests in diabetic patients treated with acetohexamide. While glucose tolerance improved progressively throughout the study, the area under the OGTT plasma insulin curve initially increased but later declined. Studies by Reaven and Dray[5] using chlorpropamide and Turtle[6] using tolazamide showed improved glucose tolerance and reduced insulin secretion during treatment. In patients treated with diet alone for two months we have shown that the change in OGTT insulin area is related to both the initial OGTT sugar area and the change in that area between the two tests.[8] Possibly the conflicting results of previous workers may have been due to differences in the severity of diabetes in the patients studied and in the improvement of glucose tolerance obtained.

There have been few studies of the effect of biguanides on the relation between glucose and insulin levels. Abramson and Arky[2] observed improved glucose tolerance in 11 patients after three weeks' treatment with phenformin. The mean OGTT plasma insulin curve was lower, as in our study, but the change was not statistically significant. The reduced OGTT sugar incremental area found in our phenformin-treated group suggests impaired gastrointestinal absorption of glucose.

Mean body weight fell significantly in all three groups during the first period of treatment with diet alone. The mean changes during the next four months in the diet and glibenclamide groups were not significant, but a further small reduction ($1{\cdot}6\pm2{\cdot}9$ kg; $P<0{\cdot}02$) was observed in the phenformin group. The influence of glibenclamide and phenformin treatment on body weight is unlikely to have much biological significance.

At a time when the long-term safety of treatment with oral antidiabetic drugs has been questioned it is important that their mechanism of action should be better understood. Further studies of the long-term effects of treatment with diet alone, glibenclamide, and phenformin are needed.

We thank Hoechst Pharmaceuticals and the Trent Regional Health Authority for financial support.

Requests for reprints should be addressed to JWHD.

References

1 Breidahl, H D, *et al*, *Drugs*, 1972, **3**, 79.
2 Abramson, E, and Arky, R A, *Metabolism*, 1967, **16**, 204.
3 Feldman, J M, and Lebowitz, H E, *Diabetes*, 1971, **20**, 745.
4 Duckworth, W C, *et al*, *Journal of Clinical Endocrinology*, 1972, **35**, 585.
5 Reaven, G, and Dray, J, *Diabetes*, 1967, **16**, 487.
6 Turtle, J R, *British Medical Journal*, 1970, **3**, 606.
7 Reaven, G, and Miller, R, *Diabetes*, 1968, **17**, 560.
8 Doar, J W H, *et al*, *Lancet*, 1975, **1**, 1263.
9 Wynn, V, and Doar, J W H, *Lancet*, 1966, **2**, 715.
10 Technicon method N-2b.
11 Albano, J D M, *et al*, *Acta Endocrinologica*, 1972, **70**, 487.
12 Documenta Geigy, *Statistical Tables*. Macclesfield, Geigy Pharmaceuticals, 1956.
13 Doar, J W H, unpublished observations.
14 Sheldon, J, Taylor, K W, and Anderson, J, *Metabolism*, 1966, **15**, 874.

SHORT REPORTS

Removal of impacted rectal foreign body with obstetric forceps

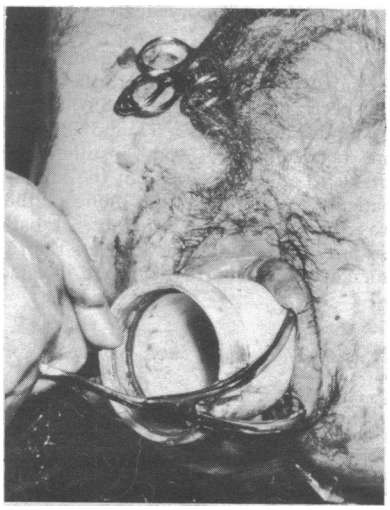
The removal of an impacted rectal foreign body using obstetric forceps is described. The technique is valuable in accident and emergency departments when other attempts at removal have failed.

Case report

A 52-year-old man presented at the accident department complaining of rectal pain and inability to open his bowels. Six hours previously he had inserted the polyethylene screw-on waste trap from the U-bend of a sink waste pipe into his rectum. On examination, the hollow cup was firmly impacted in the rectum, open end facing outwards. Under general anaesthesia, the open edge of the cup was felt hard against the ischial tuberosities and could not be drawn down past this obstruction. Short obstetric forceps were then applied, using a technique exactly similar to that in delivering an infant's head, and the cup slowly delivered (see figure). Sigmoidoscopy showed no sign of a rectal mucosal tear and the patient was discharged from hospital the next day.

Discussion

The large impacted rectal foreign body may tax the accident surgeon's ingenuity in attempting its removal. Various manipulations have been described, including plaster of Paris in glass tumblers and corkscrews for woody objects.[1] The slightly flexible nature of the cup in this case allowed it to slide into the rectum but the open end then jammed against the bony pelvis. Slow dilatation of the rectum allowed the application of well-lubricated obstetric forceps, which proved to be the only instrument that would encompass the cup without damaging the rectal mucosa. Removal was then simple.



Removal of cup using obstetric forceps.

Sigmoidoscopy should be performed shortly after removal of a foreign body to look for mucosal tears, but in patients where the rectum has been habitually overdistended rectal damage is not found.

[1] Bailey, H, and Love, J McN, in *A Short Textbook of Surgery*, ed A J H Rains and H D Ritchie, 16th edn, p 1013. London, Lewis, 1975.

Royal Portsmouth Hospital, Portsmouth
T N D PEET, MB, FRCS, surgical registrar (present address: Hammersmith Hospital, London W12 OHS)

Resolution of cutaneous manifestations of systemic sclerosis after haemodialysis

Acute renal failure, usually in association with accelerated hypertension, is a rare but well-described feature of systemic sclerosis.[1 2] It appears to develop relatively early in the disease, often without previous evidence of renal impairment, and carries a poor prognosis. Richardson[3] has reported a patient in whom haemodialysis and renal transplantation resulted in survival for 18 months, and resolution of cutaneous manifestations, arthralgia and Raynaud's phenomena. We report here a patient in whom dialysis and hypotensive therapy have been instituted for 13 months.

Case report

A 42-year-old woman presented to Dr K Keczkes in the dermatology department in July 1974. She had noticed tenderness, pain, and stiffness in the fingers of both hands for the previous 18 months; later she had developed Raynaud's phenomena and extensive thickening with tightness of the skin around her face, lips, and neck. She also had had considerable difficulty in opening her mouth. There was no family history of systemic sclerosis, although a sister had had vitiligo, both parents pernicious anaemia, and her mother rheumatoid arthritis.

She had waxy, yellowish, shiny thickening of the skin and subcutaneous tissues of her face, neck, hands, and around her mouth. She could not open her mouth more than 2 cm between the incisor teeth. There was considerable immobility of her fingers and she could not form a fist with either hand. The appearances were those of classical scleroderma. Her blood pressure was 150/100 mm Hg, but there were no other physical abnormalities. Serum urea was 3·5 mmol/l (21 mg/100 ml), electrolytes normal, and creatinine clearance 79 ml/min. Cold agglutinins were present in a titre of 1/4 at 4°C and urinary protein excretion less than 100 mg in 24 hours. A chest *x*-ray film showed interstitial fibrosis, and pulmonary function tests showed diminished diffusing capacity. The results of other extensive investigations were normal.

She was initially treated with azathioprine, 100 mg twice daily, and topical corticosteroids and emulsifying ointment. After two weeks she developed anorexia and vomiting. On her second admission to hospital her blood pressure was 240/120, while fundoscopy showed exudates and haemorrhages. She rapidly became oliguric and her creatinine clearance was found to be 4 ml/min. Renal biopsy showed appreciable endothelial proliferation of the arterioles with narrowing of the lumena.

She was established on intermittent haemodialysis twice weekly, and eventually her hypertension was successfully controlled with oxprenolol, 240 mg; diazoxide, 200 mg; and tolbutamide, 1 g daily. Azathioprine was discontinued because of leucopenia and prednisolone 5 mg daily maintained. Over 13 months she has developed deep pigmentation and hirsutism (attributed to diazoxide therapy), but she has been generally well, and increasingly active. Her scleroderma has become much less prominent, while repeated examination and investigations have shown no obvious evolving disease of the respiratory or gastrointestinal systems.

Discussion

The cause of acute renal failure in systemic sclerosis remains unexplained, and there is no evidence that any treatment will halt the progression of renal damage once it has begun. The survival of this patient would seem to be attributable to the early and adequate combination of powerful antihypertensive therapy and intermittent haemodialysis. The unexpected remission of the subcutaneous manifestations is inexplicable. Possibly a spontaneous remission[4] has taken place. Treatment with azathioprine or the low dosage of prednisolone was probably not responsible. Conceivably chronic uraemia suppresses fibroblastic function and thereby reduces excessive collagen formation.

Evidence suggests that haemodialysis or renal transplantation in systemic sclerosis associated with acute renal failure may partially reverse the progress of the disease and allow a more optimistic approach to management.

[1] Moore, H C, and Sheehan, H L, *Lancet*, 1952, **1**, 68.
[2] Rodnan, G P, Schreiner, G E, and Black, R L, *American Journal of Medicine*, 1957, **23**, 455.
[3] Richardson, J A, *Arthritis and Rheumatism*, 1973, **16**, 265.
[4] Tuffanelli, D L, and Winkelmann, R K, *Archives of Dermatology*, 1961, **84**, 359.

Department of Nephrology, Hull Royal Infirmary, Hull, North Humberside
D J BARKER, MB, MRCP, senior house officer
M J FARR, MB, MRCP, consultant physician

C3 activator and hypocomplementaemia in a "healthy" woman

Hypocomplementaemia and the presence of C3 activator (C3 nephritic factor) have been described in the following syndromes: mesangiocapillary nephritis[1 2]; partial lipodystrophy and recurrent infections[3]; and partial lipodystrophy in combination with mesangiocapillary nephritis.[4] Hypocomplementaemia induced by C3 activation has not been described in healthy persons, and we describe such a case here.

Case report

A 36-year-old woman was seen in February 1974, complaining of pain and stiffness in her thumbs, though these were neither red nor swollen and they moved easily. Her symptoms had started with tonsillitis in December 1972; One week later she had developed a swollen and red right Achilles tendon, followed by migrating pains and stiffness in different joints. She had improved after treatment with salicylates and oxyphenbutazone, but had since then occasionally suffered from arthralgia in her fingers. During the acute illness repeated examinations had shown neither haematuria nor proteinuria; the sedimentation rate (1 hour) was less than 10 mm, and no rise in antistreptolysin O titre had been detected.

Investigations showed ESR 4 mm; Hb 12·7 g/dl, white blood count $5{\cdot}7 \times 10^9$/l (5700/mm^3); serum albumin 43 g/l, urea 1·7 mmol/l (10 mg/100 ml), creatinine clearence 91 ml per min. Urine deposit normal and no proteinuria. Tests for antinuclear factor were negative and antistreptolysin O titre normal. Several blood samples during 1974 showed the presence of very low C3 (3·5 mg/100 ml) and low total hemolytic complement (CH_{50} less than 25 units per ml). C3 nephritic factor was present, as indicated by C3 breakdown in normal serum in vitro. The levels of Clq, Cls, C4, C3b inactivator (KAF), and properdin were all normal.

Discussion

Low C3 concentration induced by C3 activator was initially found in two-thirds of patients with mesangiocapillary nephritis. The role of hypocomplementaemia in this form of nephritis is obscure, since there is a poor correlation between the activity of glomerular disease and serum complement concentration.[5] This type of hypocomplementaemia is, however, regarded as a predisposing factor in nephritis. Later C3 activation was described in a woman with partial lipodystrophy and increased susceptibility to infections but with normal renal function.[3] Our patient with low concentration of C3 and C3 activator