SHORT REPORTS

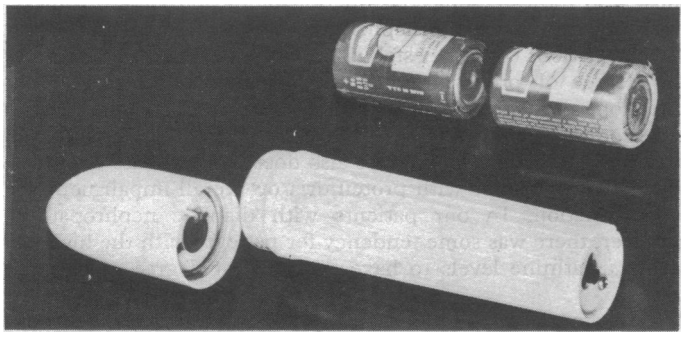
Vaginal vibrator lodged in rectum

Vaginal vibrators and other sexual surrogates are readily available in most metropolitan areas in the USA, often over the counter in pharmacies. Although usually not advertised as such, they are designed for use within the vagina, which, being fairly short, makes their retrieval comparatively easy. But increasingly these devices are being used to gratify anal erotism and may easily be inserted beyond the anal sphincters. These tend to close and to force a foreign object up the tract out of reach of the user, so that it has to be retrieved by surgery. We think these cases are increasing and that therefore the following reports are timely.

Case reports

A 40-year-old woman reported that the previous day, during sexual intercourse, her husband had inserted a battery-driven vibrator into her anus. In the excitement of the moment he had pushed the vibrator past the external sphincter and it was lost from his control. The patient was admitted to the hospital and under saddle-block anaesthesia the anus was dilated and the vibrator (fig 1) removed with forceps. Recovery was uneventful.

Another case was that of a 24-year-old woman who presented at the emergency room with a history that five days previously during intercourse her partner, at her request, had introduced into her rectum a vibrator similar to that in the first case, except that it was longer. The partner lost control of the vibrator, which disappeared beyond the anus. Attempts to retrieve it were unsuccessful. On examination the patient's anal mucosa was hyperaemic and superfiically lacerated. She was suffering from abdominal pain, nausea, and anorexia and her abdomen was slightly distended. The vibrator could not be seen within 17 cm at sigmoidoscopy, and *x*-ray examination showed it lodged in the sigmoid colon. She reported that the motor had continued to operate for about five hours.



Vaginal vibrator removed from rectum in case 1.

The patient was hospitalised and a nasogastric tube was passed to provide alternative proximal decompression. After decompression mineral oil as an emolient was introduced over a period of 24 hours. Then a mineral oil enema was given and a laparotomy was performed. No injury to the bowel was found. Since the *x*-ray examination the vibrator had moved up into the proximal descending colon, where it was held firmly by spasm of the bowel. It was carefully milked down the descending and sigmoid colon to the rectal ampulla. After this, with the patient placed in stirrups, the anal canal was dilated and, aided by manipulation from within the abdomen, the vibrator was withdrawn.

Discussion

The use of the foreign objects for anal stimulation is not new[1] nor is their removal by surgical means.[2] They have been mostly ordinary household utensils, bottles, sticks, or the like. Specially manufactured surrogate phalluses have been available since biblical times and before,[3] but never before have such a variety of sophisticated devices been so readily obtainable so inexpensively. The permissiveness engendered by today's so-called "sexual revolution" has also done much to lessen the social stigma attending some of the more unusual modes of sexual gratification. We would therefore expect the use of these devices to increase and, concomitantly, the number needing to be surgically removed from the rectum. We strongly recommend that doctors counselling patients should whenever possible emphasise the dangers of the rectal use of artificial phalluses designed for vaginal use. The law should require appropriate safeguards to be incorporated into the design to prevent their loss into the gastrointestinal tract, and they should be supplied with clear and unmistakable warnings about the dangers of improper use. Finally, the consequences of the misuse of vaginal devices might be a suitable subject for inclusion in appropriate programmes of sex education in schools and elsewhere.

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Non-ketotic diabetic precoma associated with high-dose frusemide therapy

Diabetes mellitus is a complication of thiazide diuretic treatment[1] and glucose intolerance has been associated with frusemide therapy.[2] Thiazide diuretics may cause non-ketotic diabetic coma[1] but only four cases due to moderate doses (40-80 mg/day) of frusemide have been reported.[3 4] We report a case of non-ketotic diabetic precoma in a patient receiving a high dose of frusemide for salt and water retention secondary to chronic respiratory failure.

Case report

A 61-year-old man presented in 1972 with a six-year history of progressive dyspnoea. He had gross salt and water retention secondary to chronic bronchitis. There was no glycosuria and a glucose tolerance test was normal. He was treated with digitalis, warfarin, frusemide (250 mg/day), a low salt diet (44 mmol (44 mEq) sodium), and continuous oxygen therapy. His condition improved and remained stable. From November 1974 his weight gradually increased by 8 kg, and his frusemide dosage was increased. He was taking 750 mg/day on 1 June 1975 when he was admitted to hospital confused and disorientated with blurring of vision, an extensive monilial rash in both groins, and a weight loss of 7·5 kg. There was heavy glycosuria with no ketonuria. The results of investigations were as follows: blood glucose 64 mmol/l (1150 mg/100 ml); plasma osmolality 360 mmol/kg (360 mosmol/kg); pH 7·39; plasma insulin 17·5 mU/l (normal range 4-25 mU/l); plasma sodium 120 mmol/l (120 mEq/l); plasma potassium 6·0 mmol/l (6·0 mEq/l); blood urea 23 mmol/l (140 mg/100 ml); haemoglobin 13·2 g/dl; packed cell volume 44·3%; and a total white blood cell count of $15{\cdot}3\times10^9$/l (15 300/mm^3). Chest *x*-ray examination showed a decrease in transverse cardiac diameter (17·5 cm to 14·0 cm).

The non-ketotic diabetic precoma was treated with intravenous fluids, insulin, and potassium supplements. Nine litres of half-strength normal saline was given in the first 16 hours and 7 litres of normal saline with 52 mmol of potassium during the next 36 hours. Soluble insulin 36 U was given in the first 16 hours to reduce the blood sugar to 15·5 mmol/l (280 mg/100 ml), which was subsequently maintained at 6·7 mmol/l (120 mg/100 ml) with 112 U soluble insulin/day and a 135-g carbohydrate, 5·9-MJ (1400-kcal), 44-mmol sodium diet. One week after admission the patient developed increasing salt and water retention and treatment with frusemide was restarted. When discharged six weeks later he was taking frusemide 500 mg/

day, which did not increase his insulin requirements. He was fully orientated and vision had returned to normal. The results of investigations on discharge were as follows: plasma sodium 139 mmol/l; plasma potassium 3·8 mmol/l; blood urea 3·8 mmol/l (23 mg/100 ml); plasma bicarbonate 35 mmol/l (35 mEq/l); haemoglobin 11·4 g/dl; packed cell volume 35·7%; and white blood cell count $9·8 \times 10^9$/l (9800/mm³).

Discussion

The clinical picture in this case has the typical features of non-ketotic diabetic precoma.[4] The hyperosmolar syndrome has been associated with chronic cardiovascular and renal disease.[5] Although our patient had severe chronic respiratory disease there is no evidence that this alone was the cause of the glucose intolerance. His glucose tolerance was normal three years before he presented in non-ketotic precoma. He had normal renal function and had not been taking any drugs known to be associated with non-ketotic hyperosmolar coma. Therefore frusemide, which is now a recognised factor in this biochemical abnormality, was probably the responsible agent. It should be noted that the patient's illness was precipitated by a vast increase in dosage of frusemide.

This case emphasises that the high doses of frusemide now used in cases of "resistant" oedema associated with chronic cardiac, respiratory, and renal disease may precipitate glucose intolerance and non-ketotic diabetic precoma.

Requests for reprints to Dr P R W Tasker.

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Long-term treatment with 0·01% fluclorolone acetonide in children

The use of long-term, full-strength fluorinated steroids in children is deprecated because of well-documented side effects of collagen atrophy and interference with adrenal function.[1-5] I thought it worth while, therefore, to study the effect of low concentration fluclorolone acetonide (0·01%) used regularly for 12 months in children suffering from unremitting chronic atopic dermatitis.

Patients, methods, and results

Ten children with mild or moderate atopic dermatitis aged from 6 to 12 years were treated with 0·01% fluclorolone acetonide in fatty acid and propylene glycol (FAPG) for one year. The patients were assessed clinically at one to two and two to three monthly intervals, and urine was collected for estimation of 17-hydroxycorticosteroids (17-OHCS) and 17-ketogenic steroids (17-KGS) as a check on adrenal function. Levels of plasma cortisol and stress tests of the hypothalamic-pituitary axis would have given useful information but multiple venepunctures would have been rather traumatic for the children. Throughout the study fluclorolone acetonide was applied continuously; the minimum quantity used was 15 g/week and the maximum 22·5 g/week.

The skin disease was kept under adequate control in all patients though never completely cleared. There was no evidence of skin atrophy in any case. The table shows that there was very little decrease in the 17-OHCS and 17-KGS values with this treatment. Only the 17-OHCS value at 12 months was significantly different from the baseline value ($P<0·01$), and this difference was an increase rather than a decrease.

Variations in 17-OHCS and 17-KGS values. Values are means ± SD

	Baseline value	Value at two months	Value at four months	Value at six months	Value at 12 months
17-OHCS (μmol/24 h)	9·0 ± 1·8	8·8 ± 1·4	9·7 ± 0·42	9·6 ± 1·1	10·5 ± 2·2
17-KGS (μmol/24 h)	13·7 ± 1·7	14·1 ± 1·5	13·5 ± 1·0	13·8 ± 2·7	13·2 ± 2·2

Conversion: SI to traditional units—17-OCHS and 17-KGS: 1 μmol/24 h ≈ 0·28 mg/24 h.

Discussion

I do not suggest that topical steroids should exclusively be the treatment of atopic dermatitis for which tar preparations and emollients have an important place. Nevertheless, topical steroids are widely used in treating these patients, and atrophy of the skin collagen and suppression of hypothalamic-pituitary-adrenal axis can be a problem.[5] This report suggests that, if it was necessary, maintenance treatment with low concentration fluclorolone acetonide (0·01% in FAPG base) is suitable for children with mild to moderate atopic dermatitis.

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Improvement in paraplegia in vertebral Paget's disease treated with calcitonin

Calcitonin is now accepted as an effective treatment for the bone pain of Paget's disease. We report a case of vertebral Paget's disease with spinal cord compression in which the neurological condition improved dramatically during treatment with porcine calcitonin.

Case report

The patient, a 54-year-old man, was first seen in 1972. He had been treated for hypertension for 10 years and gout for 9. Paget's disease had been diagnosed three years previously. For two years he had had pain in the lower lumbar region extending to both buttocks and thighs, which was worse on walking. His legs were also weak and for one year had felt cold and numb. He had retired from work as a clerk. On examination he was greatly disabled by pain and weakness, could not walk more than 50 yards (46 m), and used two sticks. Frontal bossing was present, gouty tophi were seen on the hands and elbows, the blood pressure was raised, and a right carotid bruit was heard. Neurological examination of the cranial nerves and arms was normal but he had weakness of the legs with increased tone, brisk tendon reflexes, and extensor plantar reflexes. Vibration sense and proprioception were impaired, Romberg's test was positive, but pain and touch were felt normally. *X*-ray examination showed Paget's disease of the skull, thoracic vertebrae 7, 8, 10, 11, and 12, lumbar vertebrae 3 and 4, the pelvis, and right femur. Myelography showed partial blocks at T7, T12, and L3 at the anterior aspect of the spinal canal opposite the vertebral bodies.

Because of the multiple blocks and the patient's opposition to surgery decompression was not performed. He was referred back one year later with a history of six months' severe lumbar pain, unrelieved by analgesics, and spasms in the thighs for some months. He was confined to a wheelchair. At this time he had a sensory level at T6, with spastic legs in which all sensation was reduced. Urinary hydroxyproline was 4·7 mmol/24 h (620 mg/24 h) and serum alkaline phosphatase was 3310 IU/l. Biopsy of bone from the iliac crest showed active Paget's disease. Treatment with porcine calcitonin 80 U intramuscularly daily was begun in October 1973. After four weeks pain and the spasms were less and he was walking with two sticks again, but the neurological signs had not changed. After three months' treatment he had little pain but the neurological signs were still present.