Two possible explanations for the appearances of oedema need to be considered. The first is that the low μ numbers reflect an increased fluid content of brain tissue—that is, true oedema—perhaps related to regional cerebral ischaemia due to vasospasm or to arteriovenous shunting with bypassing of the microcirculation.¹ Alternatively, a reduction in μ numbers and increased blackening on the Polaroid film might be due to a decreased content of blood per matrix cell if regional cerebral blood flow were decreased at the time of scanning. Further observations with both CAT and cerebral blood flow techniques in the prodromal and headache phases of migraine are required to throw further light on the significance of this finding.

The significance of the mild cerebral atrophy found in some patients is uncertain. In view of the lack of data from normal volunteers,^{2,3} the possibility that the mild widening of the subarachnoid space and ventricular system in these patients may in fact represent one end of the spectrum of normality rather than being truly indicative of early cerebral atrophy cannot be excluded. Nevertheless, in view of the fact that a greater degree of atrophy was found in two cases, it is tempting to consider the possibility that some degree of cerebral atrophy may result from the effects of recurrent episodes of regional cerebral ischaemia in migraine.

We thank our radiological, neurological, and neurosurgical colleagues for their co-operation.

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Pilonidal sinus of the prepuce

There have been many reports of pilonidal sinus at various sites since the first description of a case in 1847 by Anderson. We report here a case of pilonidal sinus of the prepuce, which has not previously been recorded.

Case report

A single 40-year-old bottle packer presented to the casualty department on 28 June 1974 complaining of swelling and soreness of the penis. On examination he was a fit man of White race with light brown hair. His penis was grossly swollen, particularly around the prepuce, so that it could not be retracted. On 12 July his penis was not painful, but there was some induration of the skin on the left side of the shaft. The prepuce was retracted and three groups of hairs were seen emerging from the inner surface of the prepuce, just distal to its junction with the shaft (see figure). One group of hairs came out very easily. There was no urethral discharge or lymphadenopathy.

Hairs arising from inner surface of prepuce.

He underwent circumcision on 23 September 1974, which showed multiple sinuses just proximal to the glans. The area of indurated tissue, impregnated with hair, was excised and the skin was closed with cat gut.

The macroscopic appearance of the hairs from the pilonidal sinus was different from that of the rest of the

hairs on his pubic region. They were therefore sent for electron microscopy, which showed them to be of human origin.

Discussion

Since Anderson first described pilonidal sinus, which commonly occurs in the upper part of the natal cleft, there have been many reports of similar pathology in other sites, including the supra-pubic region, clitoris, upper eyelid, left brow, intermammary region, cervical cutaneous subarachnoid region, ear, nipple, anal canal, scalp, perineum, and mons pubis.

Pilonidal sinus of the shaft of the penis was described independently by Bervar and Manojlovic¹ and Yates-Bell² in 1968. Bervar and Manojlovic in fact coined the name "pilonidal sinus penis" for this condition.

Since Patey and Scarff³ described pilonidal sinus of barber's hand in 1946 the opinion has swung towards the view that it has an acquired aetiology.⁴ Ghosh and Sanyal⁵ emphasise this theory by stating that pilonidal sinus is a peculiarity of occupation and poor personal hygiene; men are affected four times more than women, and the subjects are usually hirsutes.

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Carbenoxolone-induced hypokalaemia simulating Guillain-Barré syndrome

We report the cases of three patients who after carbenoxolone treatment developed weakness and areflexia mimicking a profound Guillain-Barré syndrome.

Case reports

Case 1—A man aged 67 had received, unknown to us, carbenoxolone 150 mg daily for eight weeks. During the 10 days before his admission he had noticed progressive weakness of his arms and legs, difficulty in swallowing, and headaches and constipation. Examination showed a symmetrical proximal weakness of the limbs with weak sternomastoids. Tendon reflexes were diminished or absent. Sensation was normal. The provisional diagnosis was Guillain-Barré syndrome and he was treated with steroids. The cerebrospinal fluid (CSF) protein was, however, normal, and the serum potassium concentration was 1.6 mmol (mEq)/l. ECG showed flat T and U waves. The steroids were discontinued and he was given potassium supplements 80 mmol (mEq) day. After 48 hours he walked normally with a serum potassium of only 2.1 mmol/l, and two weeks later his strength and serum potassium level were normal.

Case 2—A 54-year-old mechanic was admitted to hospital unable to stand after four weeks of progressive weakness and paraesthesiae of the limbs. Inquiries showed that he had been taking carbenoxolone 300 mg daily. His speech was slurred and he had weakness of the proximal arm and leg muscles with distal paraesthesiae and diminished reflexes. The CSF protein was raised at 0.92 g/l with normal cells. Serum potassium was 1.6 mmol/l and he was treated with oral and intravenous potassium. At 72 hours he was clinically normal. Although he was receiving 100 mmol potassium daily, it took nine days for his serum potassium to return to normal.

Case 3—A 50-year-old clerk was admitted to hospital after collapsing with a suspected haemorrhage from a known gastric ulcer for which he was taking carbenoxolone 150 mg daily. On admission he had weak muscles, paraesthesiae, and difficulty with speech, swallowing, and respiration. He required intubation and assisted respiration for hypoventilatory hypoxaemia. His

distended rectum was emptied manually. The limbs and jaw were hypotonic with absent jerks and normal sensation. The CSF protein was normal, and the serum potassium 1.8 mmol/l. Although 100 mmol intravenous and intra-gastric potassium was administered daily, it took 48 hours to raise the serum potassium to 2.5 mmol/l. He could breathe unaided after 36 hours. Recovery was uneventful.

Discussion

Carbenoxolone is widely used, and, although some reported side effects are neuromuscular,¹⁻³ such a profound Guillain-Barré-like weakness is unusual. Our second patient even had a raised CSF protein. The side effects of carbenoxolone treatment seem to be related to the electrolyte disturbance.³ Sodium and fluid retention with potassium loss occur in 60% of patients receiving a daily dose of 300 mg.⁴ Our three patients were not receiving diuretic or other treatment and did not have hepatic cirrhosis or primary aldosteronism. They rapidly responded to potassium replacement and remained well at follow-up when not receiving supplements.

It is clearly important to consider the possibility of carbenoxolone treatment and to check the serum potassium concentration in a patient with weakness, myopathy, or paraesthesiae.¹⁻⁵ Our experience shows that the weakness may simulate a life-threatening Guillain-Barré syndrome; a wrong diagnosis may result in hypokalaemia worsened by steroid treatment.

We thank Dr A B Herring for help with the first case, and Dr B A Gwynne-Jenkins for details of the second. Mrs F Oates prepared the script.

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Glucagon and beta-blocker toxicity

This report illustrates the effectiveness of glucagon in a case of oxprenolol overdosage.

Case report

In June 1975 a 62-year-old man was admitted to the casualty department having collapsed. He was given external cardiac massage by the ambulance crew and on arrival was pulseless with cold, blue peripheries. The pupils were 4 mm in diameter and unreactive to light, and the ECG showed asystole. After 35 minutes of resuscitative measures, including intravenous isoprenaline infusion, a 3-ml bolus of intravenous adrenaline (1/1000 strength), 20 ml calcium gluconate, and 100 ml sodium bicarbonate, a slow sinus rhythm of 32 beats/min was established. This increased to 68 beats/min after 0.6 mg intravenous atropine. Respiration also returned and the pupils contracted to 3 mm in diameter and responded to light. Systolic blood pressure did not increase above 30 mm Hg, and further adrenaline by continuous infusion had no effect on heart rate or blood pressure over one and a quarter hours. Glucagon 10 mg intravenously produced an increase in blood pressure to 150 mm Hg within 60 seconds, and shortly afterwards an improvement in peripheral circulation occurred. As the cardiovascular state improved severe bronchospasm developed, which was treated successfully with intravenous terbutaline. Urinary output increased from 30 to 150 ml/hour, and central venous pressure fell from 15 to 3 cm of water. A continuous infusion of glucagon 2 mg hourly was given over the next five hours, and improvement continued after its withdrawal. The patient was eventually discharged after an uncomplicated recovery.

The response to glucagon suggested the possibility of beta-blocker toxicity. It was subsequently found that the patient had ingested large amounts of

oxprenolol and diazepam. The serum oxprenolol level was 11.7 µmol/l (3100 ng/ml). The manufacturers state that a range of 1.3-1.9 µmol/l (350-500 ng/ml) would be expected one hour after an oral dose of 80 mg.

Comment

The positive inotropic effects of glucagon were first described in 1960,¹ and since then much interest has been taken in the use of glucagon in heart failure. Its inotropic effects have been noted in man,² and are more evident in acute failure than in chronic heart failure.³ They were investigated in isolated cat atria and intact dog hearts by Glick *et al*,⁴ who showed their persistence after beta-blockade with propranolol. Similarly, in man glucagon has been reported as effective in increasing cardiac output when the myocardium is severely depressed by beta-blockade.² We know of only one previously reported case in which glucagon was used effectively to treat myocardial depression after self-poisoning with beta-blocking agents.⁵ Our patient showed the effect of glucagon in the presence of overwhelming beta-blockade unresponsive to intravenous catecholamines.

We thank Dr R N Maini for permission to report this case.

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Development of RhD antibodies after kidney transplantation

We wish to report a case in which RhD antibodies developed apparently as a result of sensitisation to a kidney graft.

Case report

An A, RhD-(cde/cdE) man entered the London Hospital dialysis and transplantation programme early in 1975 as a consequence of analgesic nephropathy. RhD antibodies were detected for the first time at a level of 0.1 mg anti-D/l during a routine screening for atypical antibodies before a second kidney transplant. The table shows the relevant blood transfusion and clinical history. All serum samples indicated were screened by the conventional techniques for detecting blood group antibodies: (a) saline agglutination test at 17°C; (b) agglutination of papain-modified cells at 37°C; and (c) indirect antiglobulin test at 37°C. The RhD- cde/cdE state of each donor of the 19 units of transfused blood was rechecked at the regional transfusion centre on several occasions.

Blood transfusion and clinical history

Date	Events	Serum samples tested for:	
		Anti-RhD	Anti-HLA-A, B, C
1972	15 Units whole blood (RhD-)	-	Not tested
1973	2 Units nitrogen-frozen blood (RhD-)	-	-
1975	Haemodialysis started	-	-
3 Feb		-	-
15 Aug	Cadaver kidney from RhD + donor	-	-
2 Sept		-	-
3 Sept	Donor nephrectomy, 2 units of nitrogen-frozen blood (RhD-)	-	-
25 Nov	2nd transplant (RhD-)	+	-
1976		+	-
(25 Feb)		+	-