charged down the sink, and the test tube rinsed under the tap. This test was, on almost every occasion, performed by the nursing staff.

Twenty-two specimens of faeces were examined. Three contained intestinal pathogens—*S. montevideo*, *Shigella sonnei*, and enteropathogenic *Escherichia coli*. The faeces were tested by the method described by Kerry and Anderson. After the effervescence had subsided, the contents were plated on to blood agar and MacConkey's medium and inoculated into double strength MacConkey broth and Robertson's cooked meat broth. After overnight incubation the broths were plated on to solid media. Faecal organisms—that is, *S. montevideo*, *Klebsiella* sp., *E. coli*, *Proteus* sp., *Clostridium*, *Streptococcus faecalis* and *Bacteroides* were recovered from five (22.7%) stools by direct culture, and from 18 (81%) after incubation in broth media.

0.5 g of each of seven specimens of faeces was emulsified in 5-ml aliquots of polyvalent poliovirus vaccine containing 10^6 pfu/ml of virus. The emulsions were tested as before. Identical volumes of emulsion were used as controls. After the reaction the test and control samples were dialysed against distilled water and Hanks's solution, to remove copper ions, and then inoculated on to monolayers of Vero cells. Surviving virus was detected by plaque formation. Virus was recovered from all the controls and from three of seven of the faecal suspensions to which "Clintest" tablets had been added.

The heat produced during the test was recorded using a recording thermocouple. The temperature before the addition of a Clintest tablet was 25°C. The maximum temperature of 80°C was reached after 22 seconds. Immediately the temperature began to fall and had reached 60°C after a further 1 min 18 s. It had reached 40°C 4 min 30 s later.

Discussion

Nearly all copper compounds are strongly bacteriostatic and to some extent bactericidal but have weak virucidal properties.² There is evidence that for certain organisms the antimicrobial action of copper compounds is increased by heating. It may be assumed unwisely that the exothermic chemical reactions produced during solution of a Clintest tablet destroy the microbial population of urine or faeces. Nevertheless, the effervescence resulting from the reaction produces large bubbles, which are lifted away from the hottest part of the tube. Organisms trapped in the bubble film will not be subjected to the same conditions as those at the bottom of the tube. Collapse of these bubbles will also generate aerosols, which may also contribute to the contamination of the environment in addition to more obvious contamination due to overflowing.

We suspect that insufficient thought has been given to the microbial hazards of the test and of the subsequent disposal of the reactants and tubes. We strongly urge our colleagues that this test, although of undoubted diagnostic value, is not devoid of microbial hazards and encourage them to take the necessary steps to minimise the risk of infection.

¹ Kerry, K R, and Anderson, C M, *Lancet*, 1964, **2**, 981.

² Sykes, G, *Disinfection and Sterilization*, 2nd ed, p 422. E & E N Spon Ltd, 1965.

(Accepted 6 April 1977)

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Diabetes insipidus and panhypopituitarism after pituitary infarction in a case of acromegaly

Although panhypopituitarism is not an uncommon sequel of acromegaly the development of diabetes insipidus is rare. Postpartum haemorrhage is probably the commonest cause of pituitary infarction and occasionally it is accompanied by diabetes insipidus.¹ We have found only one report of diabetes insipidus after spontaneous infarction of a pituitary tumour.² We describe an acromegalic patient who had a pituitary infarct and subsequently developed impairment of both anterior and posterior pituitary function.

Case report

A 34-year-old man presented with a five-year history of increasing hand and foot size, weight gain, and excessive sweating. Three months before admission he developed a severe headache of sudden onset, photophobia, and neck stiffness which lasted for three days. During the subsequent months he lost weight and had decreased sweating, cold intolerance, constipation, dry skin, and diminished libido. He also complained of mild polyuria and polydipsia.

Examination revealed signs of acromegaly and hypothyroidism. Previous photographs showed a progressive increase in size of his jaw and hands. Visual fields were unimpaired and his blood pressure was 110/70 mm Hg. Radiographs showed an enlarged pituitary fossa with erosion of the base of the sella. There was tufting of the terminal phalanges, and heel-pad thickness was increased to 30 mm. Endocrine investigations confirmed hypothyroidism (serum thyroxine 41 nmol/l (3.2 µg/100 ml), T3 uptake test 113%) and also showed hypogonadism (total serum androgens 4.2 nmol/l; 1.2 ng/ml) and hypoadrenalism (morning serum cortisol <30-330 nmol/l (<1.1-11.9 µg/100 ml)). Pituitary stimulation by insulin-induced hypoglycaemia, TRH, and LH-RH showed that all the deficiencies were secondary to pituitary failure. Despite adequate hypoglycaemia there was no growth hormone response (table). After thyroxine and cortisol replacement therapy a water deprivation test indicated impaired posterior pituitary function together with a failure of the normal rise in plasma arginine vasopressin.³

Results of anterior and posterior pituitary stimulation tests

	Time (min)	Glucose (mmol/l)	GH (mU/l)	Cortisol (nmol/l)	LH (U/l)	FSH (U/l)	TSH (mU/l)
Anterior pituitary	0*	4.6	1	330	3	<1	3
	30	1.6	1	—	3	—	5
	60	1.9	<1	401	2	—	4
	90	2.5	<1	347	—	—	—
	120	3.5	<1	308	—	—	—
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	Time (h)	Urine Os (mmol/kg)	Plasma Os (mmol/kg)	Plasma AVP+ (pmol/l)	Wt (kg)		
Posterior pituitary	0	335	298	1.1	94.1		
	2	317	—	—	93.4		
	4	—	301	1.0	92.9		
	7	197	303	—	92.1		
	8	215	304	1.2	91.7		

*100 µg LHRH, 200 µg TRH, and 13 U soluble insulin given at time 0.

†Plasma arginine vasopressin measured by radioimmunoassay.³

Conversion: SI to traditional units—Blood glucose: 1 mmol/l ≈ 18 mg/100 ml. Cortisol: 1 nmol/l ≈ 0.39 µg/100 ml. AVP: 1 pmol/l ≈ 1.1 pg/ml.

Comment

Clinically there was no doubt that this patient had developed acromegaly associated with an enlarged pituitary fossa. Endocrine investigations, however, indicated both anterior and posterior pituitary impairment. From the history, the hypopituitarism and diabetes insipidus seem probably to be the consequence of a pituitary infarction three months before presentation. With cortisol and thyroxine replacement therapy the symptoms of diabetes insipidus became more pronounced and necessitated DDAVP treatment.

Diabetes insipidus is an unusual consequence of pituitary infarction. Sheehan's syndrome accounts for most pituitary infarctions, but according to a recent report¹ only a few patients develop diabetes insipidus after postpartum haemorrhage. The rareness of the clinical disorder contrasts with the finding of minor degrees of posterior pituitary and hypothalamic scarring in 90% of patients suffering from Sheehan's syndrome.⁴ Diabetes insipidus has been reported after spontaneous infarction of a pituitary tumour² and in a patient with the "empty sella syndrome,"⁵ but neither had evidence of acromegaly. The combination of anterior and posterior pituitary impairment is an uncommon consequence of pituitary infarction and our patient seems to be most unusual in that these deficiencies have followed acromegaly.

¹ Merber, E, and Futterweit, W, *American Journal of Medicine*, 1974, **56**, 555.

² Gebel, P, *Military Medicine*, 1962, **127**, 753.

³ Baylis, P H, and Heath, D A, *Clinical Endocrinology*, 1977, in press.

⁴ Sheehan, H L, and Whitehead, R, *Journal of Pathology*, 1963, **85**, 145.

⁵ Matisonn, R, and Pimstone, B, *Postgraduate Medical Journal*, 1973, **49**, 274.

(Accepted 31 March 1977)

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