of these hair dyes it will be important to take account of smoking habits in any such studies of smoking-related cancers, particularly in view of a recent report suggesting that beauticians have an increased risk of lung cancer.⁹

We thank the consultants who allowed us to interview patients under their care, and the Toilet Preparations Federation and the manufacturers of hair dyes, who helped us by providing information about hair care preparations. We also thank Mr Paul Humphreys, who helped in the analysis of this study, and Mr Peter Smith, Mr Julian Peto, and Sir Richard Doll for their helpful comments.

ADDENDUM—The Registrar General has informed us that the latest (unpublished) occupational mortality analysis for 1970-2 shows no appreciable excess of breast cancer in single female hairdressers aged under 65 years—eight observed, seven expected (OPCS, by permission).

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SHORT REPORTS

Superior vena caval obstruction complicating fiberoptic gastroscopy

Here I report a case of superior vena caval (SVC) obstruction after fiberoptic gastroscopy in a patient with an unsuspected bronchial carcinoma, who presented with an acute gastrointestinal bleed. To date there are no other reported cases of this unusual complication.

Case report

An 83-year-old Polish woman was admitted to hospital after a three-day history of melaena. She was known to have a hiatus hernia, which had been diagnosed at barium meal, when she presented with a haematemesis nine years previously. She had otherwise been well and symptom free until the current episode. She did, however, admit to a one stone (6.3 kg) weight loss over a six-month period and was noted to be a heavy smoker. On examination, apart from moderate obesity, she had no abnormal physical signs, but rectal examination confirmed melaena stools. There was no haemodynamic disturbance. The haemoglobin concentration on admission was 9.7 g/dl with normal red cell indices. The urea was 7.8 mmol/l (48 mg/100 ml) and electrolytes normal. The chest x-ray film showed appreciable unfolding of the aortic arch but was otherwise normal and she had a normal electrocardiogram. A blood transfusion was started and she was referred for endoscopy. A premedication of 7.5 mg diazepam was given intravenously.

The instrument, which was a forward viewing Olympus oesophagogastroduodenoscope, was passed without difficulty through a normal upper oesophagus. A large sliding hiatus hernia with moderate oesophagitis, not actively bleeding, was noted. The stomach contained a large pool of blood with multiple gastric erosions. At this stage the patient was noted to be having some respiratory difficulties and the endoscopy was stopped. She continued to have difficulty in breathing and after 15 minutes was considered to require ventilation. When intubated, no evidence of aspiration was found, but she was then noted to have the clinical appearance of SVC obstruction. This was supported by a high central venous pressure CVP reading (30 cm) from a catheter high in the SVC. Although the possibility that this could be due to a haematoma induced at the time of insertion of the subclavian CVP line was considered, it was felt to be more likely to be due to hitherto unsuspected bronchial carcinoma and treatment with parenteral steroids was instituted. It was possible to wean her off the ventilator after 24 hours, but her general condition remained poor and she died 48 hours later.

Necropsy showed a proximal bronchial carcinoma of the right upper main bronchus invading the mediastinum and pericardium and causing complete superior vena caval obstruction. Since there were no clinical signs of this at the initial clinical examination, it was assumed to have been precipitated by oedema caused by the trauma of endoscopy.

Discussion

The complication rate for routine upper gastrointestinal endoscopy is low, although it is recognised that emergency endoscopy is more hazardous. In a comprehensive review of complications in 23 563

cases, based on a questionnaire from the British Society for Digestive Endoscopy¹ and their own experience,² Schiller and Prout found no report of SVC obstruction. SVC obstruction is an uncommon complication of carcinoma of the bronchus and there is usually an antecedent history of neck enlargement³ or pain⁴ in addition to the more usual symptoms of the carcinoma. This patient had no such complaints.

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Microbial hazard of a bedside test

We wish to draw attention to the microbial risks associated with a commonly performed bedside test. Kerry and Anderson described the use of a commercially available reagent, Clinitest Tablets (Ames Manufacturing Co Inc), to detect reducing substances in the faeces of children with gastroenteritis.¹ A small outbreak of *Salmonella typhimurium* infection in a children's ward prompted us to investigate the possible microbiological hazards of this procedure.

Outbreak, investigations, and results

Case 1—A baby girl aged 2 months was admitted on 21 May 1976 for investigation of diarrhoea, and was barrier nursed. Stool culture taken on admission yielded a growth of *Salmonella typhimurium*, phage type DT32.

Case 2—A girl aged 6 months was admitted to a cubicle on 21 May 1976 with respiratory difficulties and a cyanotic attack. On 25 May she developed diarrhoea and was barrier nursed. *S typhimurium*, phage type DT32, was isolated from stool specimens.

Examination of stool specimens from staff yielded *S typhimurium*, phage types DT32, from the faeces of one nurse. It emerged that each stool from Case 1 had been examined for pH and reducing substances by the method of Kerry and Anderson, the colour reaction being noted, the contents dis-

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.

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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	—	—	—
<hr/>							
	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.

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

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