0.96 mmol/l (2.9 mg/100 ml), alkaline phosphatase 150 IU/l, and urea 3.8 mmol/l (23 mg/100 ml). Serum immunoassayable parathyroid hormone (iPTH) concentration was 13.1 µg/l (normal 0.8 µg/l). Skeletal radiographs showed subperiosteal erosions of the phalanges and a "rugger-jersey" spine. These findings indicated primary hyperparathyroidism. In March 1977 his neck was explored. A large, encapsulated, tan-coloured tumour (2.5 cm × 2 cm × 2 cm) was found in the left lower position. Three other normal-sized nodes were thought to be recognised clinically, but biopsies were not done. The tumour was composed of chief cells and, although the cells had a regular appearance, occasional mitoses were seen, suggesting the possibility of parathyroid carcinoma. He made an uneventful recovery, and the serum calcium and iPTH concentrations returned to normal.

He remained well until June 1978, when he again developed backache. The serum calcium concentration was now raised (3.8 mmol/l), as was the serum iPTH (5.4 µg/l). He was readmitted. The hypercalcaemia was confirmed but while an inpatient his serum calcium concentration fell spontaneously to normal. This change was accompanied by decreases in the serum iPTH and in the urinary nephrogenic cyclic AMP¹ and by an increase in the renal tubular reabsorption of phosphate (figure). This remission persisted for

Serum concentrations in case of spontaneous biochemical remission in parathyroid carcinoma (---) shows upper limit of normal range.

Conversion: SI to traditional units—Calcium: 1 mmol/l ≈ 4 mg/100 ml.

three weeks. In September 1978, after the return of hypercalcaemia, his neck was again explored and also the mediastinum. A biopsy specimen of tissue taken from the left upper and right lower poles of the thyroid gland showed parathyroid tissue surrounded by adipose tissue and separated by dense fibrous bands. The histology of tissue from sites in the thymic region showed further nests of parathyroid tissue insinuated between thymic lobules. There was a clump of parathyroid cells in a vein. In one of the nests of parathyroid tissue there was an area of granulation tissue containing haemosiderin pigment. The appearances were those of parathyroid carcinoma with possible tumour infarction.

He made a good postoperative recovery and the serum calcium and iPTH concentrations fell to normal. In March 1980 hypercalcaemia recurred (serum calcium 3.15 mmol/l, serum iPTH 1.3 µg/l) and has persisted.

Comment

The clinical, operative, and histological findings confirmed that this patient had a parathyroid carcinoma. Spontaneous relapse of hyperparathyroidism was to be expected. In primary hyperparathyroidism associated with adenoma the serum calcium concentration occasionally fluctuates² and may return to normal. Much less common is spontaneous infarction of the affected gland, which may cause a prolonged biochemical remission and sometimes profound hypocalcaemia.^{3,4} The granulation tissue and haemosiderin pigment found in one of the nests of parathyroid cells suggests localised infarction of the tumour. This might explain the temporary biochemical remission. Hypomagnesaemia, which impairs parathyroid hormone secretion, was not present at any stage.⁵

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Risks from cannulae used to maintain intravenous access

Over five million intravenous plastic cannulae are used each year in Britain. Most are the Teflon Venflon type with a valved side injection port permitting convenient intermittent intravenous access. This design, however, reportedly increases the possibility of infection,¹ though evidence is lacking. We have therefore studied prospectively the risks of using the Venflon cannula for intermittent vascular access.

Materials, methods, and results

During January to March 1980 we studied 102 consecutive Venflon cannulae inserted for at least 24 hours for intermittent or emergency intravenous access in patients in two medical wards. Each entry site was examined for local inflammation (defined as the presence of two or more of noticeable tenderness, redness, swelling, and phlebitis), a skin swab taken, and the site sprayed with 0.5% chlorhexidine in spirit. The cannula was then carefully removed, the intravenous portion cut off aseptically into a dry sterile container, and a blood specimen taken for culture. The cannula tip was cultured both quantitatively by a roll-plate technique² and qualitatively in nutrient broth. Fifteen cannulae produced definite bacterial growth² on roll-plate culture, and these and an additional eight were positive on broth culture.

Cannula infection was significantly associated with positive skin-swab cultures (see table), the same micro-organism (mainly *Staphylococcus epidermidis*) always being isolated from both sites. Phage typing, biotyping, and antibiograms suggested that out of 12 paired isolates from positive cannula and skin cultures, seven were identical. One patient had *Staph aureus* grown from all specimens, including blood, and died of septicaemia and acute endocarditis. Cannula infection was not associated with local inflammation or duration of cannulation, though the second two were significantly related (table). Type of medication given through the side port

Relation between cannula infection, skin infection, local inflammation at site of cannulation, and duration of cannulation

	No of cases (total = 102)	No of positive skin cultures	Duration of cannulation (hours)	
			Mean ± SD	Range
Cannulae with positive roll-plate culture	15	13*	92 ± 59	24-243†
Cannulae with negative roll-plate culture	87	17*	83 ± 53	24-312†
Local inflammation present	35	12†	112 ± 63	39-312‡
Local inflammation absent	67	18†	68 ± 40	24-243‡

*Significant difference ($p < 0.005$; χ^2 test).

†No significant difference.

‡Significant difference ($p < 0.005$; Student's t test).

did not influence infection or inflammation, and 39 cannulae had been used once or not at all. No medication had been given through four of the 15 infected cannulae, including the septicaemic case. Significantly more of the infected than non-infected cannulae, however, had been used for blood transfusion (four out of 15 compared with four out of 87: $p < 0.002$; χ^2 test with Yates's correction).

We studied a second group of 20 patients to determine the risks of using the Venflon injection port for giving intravenous medication during operations. The cannulae were in situ for up to two hours and no cannula infection or local inflammation occurred.

Comment

In this study complication rates for phlebitis (35%), cannula-tip infection (15%), and septicaemia (1%) were no higher than those reported for continuous-flow cannulae used for fluid³ or heparin infusion.⁴ Broth cultures yielded a higher incidence of cannula infection, but that technique probably overestimates important cannula infection.²

Isolation of identical *Staph epidermidis* strains from both skin and cannula tip suggests that bacteria originated from the patients' skin. Absence of infection in cannulae in situ for under two hours suggests that micro-organisms migrate along the cannula after insertion, and argues for careful local toilet.³ The one fatal case of septicaemia emphasises the potential seriousness of cannula infection. Blood transfusion may increase the risk of infection by increasing fibrin sheathing on cannulae, a factor implicated in cannula infection.⁵ We suggest that cannulae should be replaced after blood transfusion if continued intravenous access is necessary.

A quarter of our inpatients had cannulae inserted, yet less than two-thirds of these were used more than once. We do not regard our insertion rate as unusual. Hence, considering the risks, we question the necessity of inserting cannulae so often as a purely precautionary measure.

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Requests for reprints should be sent to Dr J T Macfarlane.

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Effects of high dietary sugar

The inclusion of sugar (sucrose) in the diet of laboratory animals or an increase in the amount of sucrose in the diet of healthy volunteers produces several changes in the body. Some of these resemble the abnormalities seen in coronary heart disease¹ and others those seen in maturity-onset diabetes. Examples of the changes that resemble abnormalities in diabetes are impaired glucose tolerance, retinopathy, nephropathy,² and reduced insulin sensitivity of the tissues.³ We have recently concluded a further experiment in which 14 young men increased for three weeks their average daily intake of sucrose from 115 g to 260 g. This may be compared with the average intake in this

country of about 125 g a day. Many people take 300 g or more. We can now report two further observations that add to the evidence that dietary sucrose is a likely cause of human disease.

Subjects and results

A high sugar diet of 260 g daily was given to 14 young men. At the end of three weeks, there was in 10 of the 14 subjects a fall in the fasted blood concentrations of high density lipoprotein (HDL) cholesterol (table), which reverted to its original value after the subjects had returned to their habitual diet for two weeks. The four who did not respond to sucrose had low initial HDL cholesterol concentrations. The total cholesterol concentration did not change throughout the experiment. It is now widely accepted that a low concentration of HDL cholesterol is an excellent indication of "coronary risk."⁴

We also measured the activity in the urine of the enzyme n-acetylglucosaminidase (NAG), an increase of which is considered to be an early indication of kidney damage.⁵ The activity of NAG had significantly risen at the end of three weeks on the high sugar diet. The apparent fall two weeks later did not reach statistical significance.

Effect of high sucrose diets on mean ($\pm$ SEM) serum HDL cholesterol concentrations and urinary N-acetylglucosaminidase (NAG) activity in young men (t test for related samples and significance of differences)

	Usual diet	High sucrose diet (3 weeks)	Usual diet (after 2 weeks)
Serum HDL cholesterol (mmol/l)	1.27 $\pm$ 0.06	1.07 $\pm$ 0.06*†	1.42 $\pm$ 0.08
Total serum cholesterol (mmol/l)	4.15 $\pm$ 0.30	4.12 $\pm$ 0.29	4.25 $\pm$ 0.39
Urinary NAG (units excreted in 24 h)	31 $\pm$ 6	51 $\pm$ 6‡	36 $\pm$ 4

* ($p < 0.01$) Compares values on initial diet (col 1) with those on high sucrose diet.

† Indicates statistical difference ($p < 0.01$) between values for high sucrose diet and those after return to usual diet (Col 3).

‡ ($p < 0.05$) Compares values on initial diet (Col 1) with those on high sucrose diet.

Comment

The results of this study suggest that a high sugar intake may cause kidney damage in man and may ultimately lead to the nephropathy we have found in rats.

We thank Miss P Mumford for her help with the diets and calculations of sugar intakes.

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Correction

Rifampicin-associated pseudomembranous colitis

We regret that an error occurred in this article by Mr S P Borriello and others (1 November, p 1180). Mr Borriello's name was wrongly spelt and should have appeared as it does here.