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 fluid[3] 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 disease[1] 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 (±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±0·06	1·07±0·06*†	1·42±0·08
Total serum cholesterol (mmol/l)	4·15±0·30	4·12±0·29	4·25±0·39
Urinary NAG (units excreted in 24 h)	31±6	51±6‡	36±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.