therapy. After a dose of viprynium with conventional purgation on the preceding day she was given whole-gut perfusion with piperazine added to the lavage solution in a dose of 0·25 g/l. Many dead threadworms were recovered. The patient remained symptom free for six months and no threadworms have since been identified in her stools.

Discussion

Whole-gut perfusion is a safe, simple, and comfortable procedure in patients who are neither obstructed nor constipated. After 30 to 45 minutes of perfusion regular, comfortable bowel action is established. The stools soon become rice-water in type, indicating that nearly all faecal material has been evacuated. The treatment saves nursing time. It deserves wider use whenever thorough cleansing of the bowel is required or slowly absorbed poisons need to be rapidly evacuated.

We thank the nursing staff for skilled help, and Drs A J Levi and J M Gumpel for encouragement and guidance.

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Severe anaphylaxis due to passive sensitisation by donor blood

There are many reports of "immediate-type" hypersensitivity reactions of varied aetiology associated with blood transfusion.[1] We report here an unusual example of this type of reaction which followed a transfusion of blood from an atopic donor.

Case report

A 63-year-old man was admitted to hospital because of epistaxis during the preceding four days. Bleeding was controlled by local measures; however, his haemoglobin had fallen to 9·1 g/dl and he was transfused with two units of whole blood. Thirty minutes after receiving the second unit of blood he developed severe generalised pruritus and urticaria, together with central chest pain and hypotension, (his systolic blood pressure was 80 mm Hg). He responded rapidly to 10 mg chlorpheniramine given parenterally, though he remained hypotensive for a further four hours, thereafter making an uneventful recovery. He had no history of previous blood transfusions or allergic episodes.

Routine investigations of a transfusion reaction were undertaken, all of which showed no abnormality. Antibody screening of the patient's pre-transfusion serum showed a weak anti-P_1, acting only with enzyme-treated cells, and a weak anti-Gm (1). One donor (A) was Gm (1)-negative; the other (B) was Gm (1)-positive. HLA and anti-IgA antibodies were not detected. The patient's serum immunoglobulin levels were normal. The two donors involved were traced and interviewed. Donor A had always been perfectly healthy. Donor B had suffered from asthma in childhood but more recently such episodes had been infrequent and had developed only after exposure to fish odours or high concentrations of house dust. Since early childhood, whenever he ate fish, donor B had experienced swelling of the pharynx associated with periorbital oedema and urticaria. Similar, but less severe, reactions occurred after eating peas and beans. Donor B was skin-tested and gave very large wheals to herring, sardine, cod, plaice, and shrimp; a modest reaction to beans; and only a minimal response to peas. He also reacted to several allergens, including grass pollen and *Dermatophagoides pteronyssinus* and *D farinae*, which may well have been related to his asthma. Further questioning disclosed that the patient had twice eaten fish in the two days before transfusion, during which time he had also eaten broad beans and peas.

Discussion

Anaphylactic transfusion reactions are usually manifested by urticaria, swelling of the mucous membranes, and pruritus. These usually occur when atopic patients are passively transfused with common allergens in donor blood—for example, milk or eggs.[2–4] Mild reactions—that is, a few urticarial wheals—are uncommon but have been described in as many as 3% of transfused patients; more severe anaphylactic reactions with shock and dyspnoea, however, are rare.[3] Exceptionally, however, a normal recipient may be passively sensitised by a transfusion of blood which contains a high titre reaginic antibody. The classic reaction of this type was described by Ramirez.[4] A man, without an allergic history, developed asthma during a carriage ride two weeks after receiving a transfusion of blood from a donor who was subsequently found to be allergic to horse dander. Apart from this celebrated case there are few recent reports of reactions of this type, probably because atopic individuals are not accepted as blood donors.

In this case the usual serological investigations showed a weak anti-P_1 and a weak anti-Gm(1), which were considered to be of no clinical importance. The anti-Gm(1) was assumed to be naturally occurring and, three weeks after transfusion, the persistence of antibody in the presence of Gm(1)-positive material from donor B indicated the low affinity of this antibody and supports the view[1] that Gm antibodies rarely cause transfusion reactions. Certainly, these antibodies do not satisfactorily explain this transfusion reaction with such striking anaphylactic manifestations.

We believe that this non-allergic patient was passively sensitised by a transfusion of blood from the atopic donor, to allergens (probably fish and beans) which he had previously eaten. The atopic donor had previously avoided donating blood because of his allergic history but he had done so on this occasion after strong persuasion from a colleague. Unfortunately, he had omitted to mention his atopic history during routine questioning before phlebotomy. This unpleasant and dangerous transfusion reaction serves as a stern reminder of the importance of excluding such individuals from donor panels.

We wish to thank Dr G Taylor for skin-testing donor B and Mr D Devine for allowing us to publish details of his patient.

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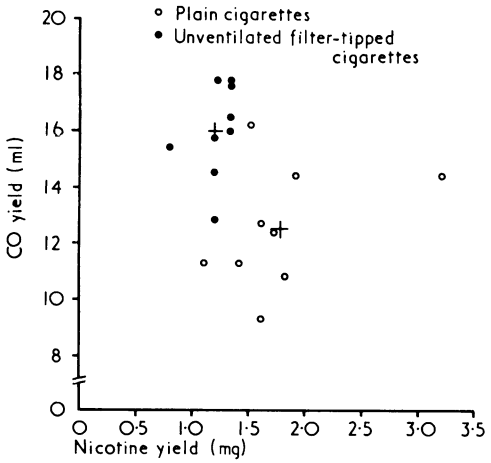
Smoking tables for carbon monoxide?

It has been suggested that the carbon monoxide (CO) yields of cigarettes should be published in Government Smoking Tables together with figures for nicotine and tar.[1] Nevertheless, the inclusion of CO yields would make the tables confusing to smokers who wished to select a less harmful brand, as some cigarettes with high CO yields have low tar and nicotine yields, and vice versa.

Methods and results

The CO yields of 18 popular brands of cigarette were measured using a smoking machine under standard conditions.[2] Nine brands were of the plain (non-filter) type and nine had ordinary unventilated filters. Nicotine yields were taken from Government Smoking Tables.

The average CO yield of the filter cigarettes was 28% higher than that of the plain ones (see figure), a highly significant difference ($P<0·001$ by Wilcoxon rank sum test.) In contrast, nicotine yields were significantly



Carbon monoxide and nicotine yields of nine plain brands and nine non-ventilated filter brands of cigarette. Each CO value is the mean of two to four assays. The crosses represent the means of the two types of cigarette. The plain brands were: Capstan Full Strength, Capstan Medium, Churchmans No. 1, Embassy Plain, Gauloise Caporal Plain, Kensitas Plain, Piccadilly No. 1, Senior Service Plain, and Woodbine Plain. The filter brands were: Consulate Menthol, Dunhill International, Gitanes Caporal Filter, John Player Special, Peter Stuyvesant King Size, Piccadilly King Size, Players No. 6 Filter, Rothmans King Size Filter and Senior Service Tipped.

lower in the filter than in the plain cigarettes ($P<0.005$). When plain and filter cigarettes were considered separately there was, within each type, a suggestion of a positive correlation between CO and nicotine yields. These complex relationships between CO and nicotine yields were obscured when the type of cigarette was ignored.

Discussion

Ordinary filter cigarettes have unventilated filters, and have higher CO yields than plain cigarettes because the paper surrounding the filter is less porous than that surrounding the tobacco—so that less air can enter through the wall of the cigarette to dilute the smoke. These cigarettes now occupy about 80% of the UK market, whereas in 1955 most cigarettes were of the plain type and filter cigarettes occupied only 2%. Alternatively, cigarettes may have ventilated (perforated) filters, through which air can enter and reduce the concentration of all the constituents of tobacco smoke. Hence it would be expected that the CO and nicotine yields of these cigarettes are highly correlated.[1] These deliver less tar, nicotine, and CO than either plain or unventilated filter cigarettes but occupy only about 5% of the UK market.[3]

A recent report on the examination of three plain and four filter brands showed that among cigarettes with nicotine yields over 1·0 mg (a level which automatically excludes all those with ventilated filters) there was no apparent relation between nicotine and CO yields, and that the filters of these cigarettes did not appear to reduce CO delivery.[1] Our results suggest they may actually increase it, but data on more brands are needed before the size of this increase can be reliably estimated.

The Government publishes smoking tables to encourage smokers to avoid brands with high tar or nicotine yields. Evidence of toxicity is much less secure for nicotine than for tar, but since the yields of both tend to vary in parallel, this is of little practical importance. The toxicity of CO in tobacco smoke is largely unknown, but present evidence suggests that it may be more harmful than nicotine.[4] For this reason it has been recommended that CO yields should be included in smoking tables, but this proposal raises a special problem: some brands with "medium" tar yields have relatively high CO yields, and others with lower CO yields produce more tar. Smokers who cannot stop smoking and who wish to base their choice of cigarette on information in tables giving figures for tar, nicotine, and CO may, therefore, find themselves in a dilemma, since the only cigarettes which are low in all three are not generally regarded as satisfying. We believe, therefore, that it would be unwise to include CO yields in official tables until medical advice can be given about the relative risks of exposure to the different components of cigarette smoke.

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Hypertriglyceridaemia and abdominal pain

Hypertriglyceridaemia has long been known to be associated with abdominal pain. Such cases are usually Fredrickson types I and V,[1] with massive rises in the serum triglyceride concentration. We report an example of pain associated, at least between attacks, with modest hypertriglyceridaemia.

Case report

A 33-year-old South African Indian had first been admitted as an emergency in October 1974 complaining of central abdominal pain. He had been well until six weeks previously, when he had started to get bouts of central colicky abdominal pain occurring once a week, lasting 20 minutes accompanied by nausea. Over the previous few days the pain had become continuous. The only abnormal physical sign noted was tenderness in the right iliac fossa. Appendicular or ureteric colic had been diagnosed. The pain had subsided and the patient had been discharged. His attacks had continued and as an outpatient, the results of intravenous pyelography, barium meal and follow-through, and cholecystogram had all been normal. Fasting serum lipids had shown a serum triglyceride concentration of 5·3 mmol/l (469 mg/100 ml) (normal 0·3-2·0 mmol/l (26-178 mg/100 ml)) and a cholesterol of 7·4 mmol/l (286 mg/100 ml) (normal 4·0-7·5 mmol/l (154-290 mg/100 ml)) with the presence of chylomicrons.

On admission for investigation he stated that he was now getting two attacks per week and had lost two jobs as a result. It was noted that there was no family history of similar attacks or of premature vascular disease, and xanthomata or arcus senilis were not present. The results of extensive investigation were all normal. An oral glucose tolerance test gave normal results. Serum uric acid was 441 μmol/l (7·4 mg/100 ml) (normal 100-400 μmol/l (1·7-6·7 mg/100 ml)). Fasting serum triglyceride concentration was 3·0 mmol/l (266 mg/100 ml), cholesterol 6·5 mmol/l (251 mg/100 ml), without the presence of chylomicrons. Lipoprotein electrophoretic strip[2] showed a densely staining pre-beta band indicating type IV hyperlipoproteinaemia. Plasma post-heparin lipolytic activity was normal.[3] He was challenged with a high-fat diet and the same evening developed severe abdominal pain. A blood sample taken 10 pm that night showed that the serum was loaded with chylomicrons; the triglyceride concentration was then 14·2 mmol/l (1257 mg/100 ml), and serum amylase 70 Somogyi units/100 ml (normal 0-200).

The pain subsided during the night, and fasting serum triglyceride levels the following morning were 4·3 mmol/l (380 mg/100 ml) and the morning after 4·6 mmol/l (407 mg/100 ml) both samples showing presence of chylomicrons. He was started on a low-fat diet but continued to have attacks of abdominal pain, and six weeks later fasting triglyceride was 4·2 mmol/l (372 mg/100 ml) without chylomicrons. He was then changed to a 120-g carbohydrate diet (previous intake 200 g) with substitution of polyunsaturated for saturated fats. His attacks of pain ceased and two months later the fasting serum triglyceride was 3·4 mmol/l (301 mg/100 ml) without chylomicrons. Though he was free of symptoms we felt it necessary to further reduce his serum triglyceride level, and gave him clofibrate, 1 g twice daily. He was seen again at the end of August 1975, three months after starting his low-carbohydrate/polyunsaturated-fat-substituted diet, and one month after starting clofibrate. He felt well, had had no further attacks of pain, and was holding down an important executive post. Fasting serum triglyceride was 2·0 mmol/l (177 mg/100 ml) with a normal lipoprotein electrophoretic strip. He was last seen on 13 October, when he remained well and symptom free; the fasting serum was clear, with a triglyceride concentration 1·5 mmol/l (133 mg/100 ml).