(hypercholesterolaemia, hypertension, smoking, obesity, lack of exercise, and diabetes mellitus) and ischaemic heart disease I concluded that, for each factor, the association is largely or wholly genetic in origin. Trends in the sex- and age-specific mortality from ischaemic heart disease in US whites and non-whites during the period of falling rates, 1968-75, were also studied. They were compared with those for mortality from all causes—which are uncomplicated by errors of death certification. From the close parallels between the two sets of trends we can be reasonably confident that the recorded fall in mortality from ischaemic heart disease is largely genuine.¹ The special characteristics of the fall, especially in whites, imply that changes in the conventional "risk factors"—which on independent grounds appear to be non-causal—are most unlikely to have made any appreciable contribution. However, the trends are consistent with the hypothesis that the impact of one or more extrinsic precipitators of ischaemic heart disease has fallen.¹ (Mortality statistics for the Australian population² show that a similar interpretation can be given to the fall in ischaemic heart disease recorded over the period from 1965-7 to 1977-8.) Agents that can act as precipitators include allergens and micro-organisms of all kinds, although viruses must be regarded as promising candidates for the precipitation of ischaemic heart disease because of the apparent exacerbating action³ of influenza viruses. (Precipitators start the active disease process by freeing "forbidden clones" of lymphocytes from the host's defence system; pathogenic forbidden clones proliferate and attack endothelial cells of coronary arteries. Exacerbators increase the severity of attack by already active forbidden clones.)

If this interpretation is substantially correct then changes in diet, smoking, and exercise will have no appreciable influence on mortality from ischaemic heart disease unless they happen to change the prevalence, transmissibility, or effectiveness of the hypothetical precipitators. Confirmation and identification of these precipitating agents is much to be desired if we are to prevent ischaemic heart disease through planned control.

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¹ Burch PRJ. *Cardiovasc Res* 1980;14:307-38.

² Dobson AJ, Gibberd RW, Wheeler DJ, Leeder SR. *Am J Epidemiol* 1981;113:404-12.

³ Rogot E, Fabsitz R, Feinleib M. *Am J Epidemiol* 1976;103:198-211.

⁴ Bainton D, Jones GR, Hole D. *Int J Epidemiol* 1978;7:231-9.

SIR,—In his article "Strategy of prevention: lessons from cardiovascular disease" (6 June, p 1847) Professor Geoffrey Rose contrasts the favourable trends in male mortality rates from ischaemic heart disease for the period 1968-77 in the United States and Australia with the absence of much change in England and Wales during these years. His statement about a large market shift away from butter and towards soft margarine could be interpreted as meaning that a falling consumption of butter made an important contribution to the ischaemic heart disease mortality trend in the United States and Australia.

The food data published by the Organisation for Economic Co-operation and Development¹ 2

show that in the United States from 1965-9 to 1975 the daily per caput intake of butter fat declined only from 5.8 g to 4.8 g. The total intake of dairy fats (including butter) fell 2.2 g (8.7%), from 25.2 g to 23.0 g. In the same period the intake of lactose fell 3.3 g (10.3%) from 32.1 g to 28.8 g.

In Australia the consumption of butter did not start to fall until 1972, several years after the decline in ischaemic heart disease mortality had begun, and did not fall below the UK level until 1974. On the other hand, the consumption of whole milk and of total dairy products excluding butter fell steadily from 1969 to 1974 (except for small rises in 1972). The intake of dairy fat excluding butter fell 1.1 g (6.3%), from 17.4 g to 16.3 g. The intake of lactose fell 4.4 g (13.3%), from 33.0 g to 28.6 g.

In Switzerland a decline in cardiovascular mortality has been associated only with decreasing milk consumption, while the intakes of butter, cream, cheese, and animal fats—as well as the consumption of cigarettes—have increased.³

Moreover, populations with a high prevalence of physiological lactose malabsorption are much less affected by ischaemic heart disease than are populations of predominantly lactose absorbers, as I have reported.⁴ The coronary hazard of dairy foods might lie more with lactose than with the dairy fats.

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¹ Organisation for Economic Co-operation and Development. *Food consumption statistics 1955-73*. Paris: OECD, 1975.

² Organisation for Economic Co-operation and Development. *Food consumption statistics 1970-75*. Paris: OECD, 1978.

³ Guberman E. *J Epidemiol Comm Health* 1979;33:114-20.

⁴ Segall JJ. *Intern J Epidemiol* 1980;9:271-6.

"Caucasian"

SIR,—Dr M G Selson is right (2 May, p 1480) in objecting to the use of the term Caucasian as an epithet for the more-or-less white indigenous inhabitants of Europe and of those countries to which they have migrated, but he is right for the wrong reason. His obvious indignation at a supposed "racist" basis for the term is misdirected. "Caucasian" was coined by J F Blumenbach of Göttingen in 1795 for reasons that may have been acceptable in the context of what was known and believed in the field of anthropology in the late eighteenth century. The objection to the term arises from the fact that it appertains also to the inhabitants of the Caucasus area of the Soviet Union. We need an unambiguous term. Baker has adopted the term *Europid* (en suite with *Negrid*, *Australid*, etc.).¹ Perhaps you, Sir, would consider the adoption of "Europid" (or some analogous term) instead of Caucasian in communications published in the journal.

As to Dr Selson's assertion that it is "nonsense to talk about races in any scientific context," I would ask to what definition of race he objects. It is undeniable that the indigenous inhabitants of different parts of the globe differ in their anatomical characters, and that there are systematic differences between, for example, a Bushman, an African pygmy, an Eskimo and Dr Selson's next-door neighbour. It is convenient to have a word to designate the fact of these differences and, if we did not already have the word race, it

would be necessary to invent a word. The merging of adjacent races yielding intermediate types in no way lessens the need for this term.

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¹ Baker JR. *Race*. London: Oxford University Press, 1974:204.

Dietary fat and coronary artery disease

SIR,—I am amazed by the statement (6 June, p 1865) that the "importance of dietary fat intake in the pathogenesis of coronary disease has recently been confirmed by the long-term follow-up results of the Western Electric Study."¹ That study of an initial 2107 men followed over 19 years has been severely criticised by other experts.² The data indeed show no significant relationship of coronary mortality to saturated fat intake. A significant relationship of coronary deaths to dietary cholesterol was claimed but the authors now admit³ that their figures were calculated incorrectly. Recalculation shows no significant difference in death rate between low, medium, and high cholesterol intakes.

No doubt the reconvened DHSS Committee on Medical Aspects of Food Policy⁴ will examine the corrected figures.

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¹ Shekelle RB, Shryock AM, Paul O, et al. *N Engl J Med* 1981;304:65-70.

² Oster KA, el Lozy M, Olson RE, Blum LD. *N Engl J Med* 1981;304:1168-9.

³ Shekelle RB, Shryock AM, Paul O, et al. *N Engl J Med* 1981;304:1169-70.

⁴ Anonymous. *Lancet* 1981;i:597-8.

Bladder cancer

SIR,—With reference to your leading article on "Bladder cancer" (28 March, p 1012), we believe that it is worthwhile to record the pattern, behaviour, and management of bladder carcinoma in a district hospital. Bladder cancer has traditionally been treated at district general hospitals by general surgeons. This may change with increasing specialist urology units. But for the near future bladder cancer will still be treated in Britain at district hospitals by general surgeons with a special interest and training in urology. In such units radiotherapy and oncology facilities are not always immediately available, dictating a different approach to bladder cancer management than that recorded in your article. This prompts the question: "Should all bladder cancers be referred to specialist urology units?" However, without knowledge of the results obtained in both situations this question is difficult to answer.

In the district of Ealing (population 220 000) the majority of bladder cancers during the past 20 years have been treated by one general surgeon at the district hospital. During that period 215 patients with bladder cancer were seen (135 males, 80 females; mean age 69 years). All patients underwent an initial staging procedure, which included urography, cystoscopy, bimanual examination of the bladder under anaesthesia, and tumour biopsy. On this basis, three groups of patients could be identified: (i) patients with superficial papillary tumours (110 patients)—treated by endoscopic diathermy resection; (ii) patients with infiltrating (T2 or T3) tumours (51 patients)—treated by total simple cystectomy

TABLE I—Pattern of disease in patients with superficial tumours

Category of patient	No followed more than 5 years	No of recurrences			Deaths from bladder cancer
		1	1	≥2	
Patients without residual or recurrent tumour at 3 months	33	20	3	10	1 (3%)
Patients with residual or recurrent tumour at 3 months	38	0	3	35	9 (24%)

TABLE II—Results of treating 51 patients with invasive bladder carcinoma by total cystectomy and urinary diversion

No of years since cystectomy	No of patients still alive	No of patients who died	Cause of death		
			Disease	Treatment	Non-related
0-1	2	16	3	10	3
1-5	3	14	5	4	5
5-10	7	3	0	1	2
10-20	4	2	0	1	1

without either preoperative or postoperative radiotherapy; (iii) patients with clearly advanced extravesical disease (54 patients)—treated by radiotherapy or terminal care as indicated.

In patients with superficial papillary tumours the five- and 10-year survival rates were 76% and 71% respectively—figures similar to those reported by specialist urology units here and abroad.^{1,2} Two distinct subgroups of patients could be recognised (table I) in this category: (1) Patients who were free of tumour at the first three-monthly check cystoscopy (that is, had neither residual nor recurrent tumour); these patients had quite a good outlook in terms of tumour recurrence and long-term survival, 61% having no further recurrences of tumour during a minimum follow-up period of five years and only 3% dying of bladder cancer during a five-year period. (2) Patients who had residual or recurrent tumour at three months; 92% of these patients developed multiple (more than five) recurrences during the follow-up programme and 24% eventually died of bladder cancer. The patients with infiltrative tumours who were treated by total cystectomy had a high perioperative mortality rate (17.6%), related to cardiovascular and respiratory complications of surgery in an elderly group. However, the five- and 10-year survival rates were 31% and 12% respectively (table II). The patients with advanced extravesical disease had a very poor prognosis, and all of these patients were dead within two years of diagnosis.

Although the overall results given above are poor, they differ little from those reported by specialist urology units, including units which strongly favour radiotherapy.^{3,4} It seems hard to justify any claims that patients with bladder cancer may fare less well in district general hospitals or centres without immediate access to radiotherapy facilities. Clearly there is much room for improvement in the survival figures for bladder cancer, but in the light of present knowledge we feel that a primarily surgical approach to this difficult problem is not inappropriate, but that adjuvant or possibly only cytotoxic chemotherapy might lessen the poor results in patients with advanced disease.

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¹ Pryor JP. *Br J Urol* 1973;45:586-92.

² Barnes RW, Dick AL, Hadley HL, et al. *Cancer Res* 1977;37:2895-7.

³ Prout GR jun. *Cancer Res* 1977;37:2764-70.

⁴ Whitmore WF, Batata MA, Ghoneim MA, et al. *J Urol* 1977;118:184-7.

Assisting the police

SIR,—In describing the duties of a doctor examining one of his own patients after death,

Dr Stuart Carne (6 June, p 1839) stated that the coroner must be notified "in cases where the doctor had not seen the patient within 14 days of death." Leaving aside the somewhat pedantic argument as to whether this compulsory duty falls to the doctor or the registrar, I still feel that the instruction is incorrect.

Regulation 51(1) of the Registration of Births, Deaths and Marriages Regulations 1968 states that the relevant circumstances are when the deceased was seen by the certifying medical practitioner "neither after death nor within 14 days before death." The words neither and nor are emphasised by Davies.¹ Thus the carrying out of either examination would satisfy the requirements for direct death certification and registration.

It has always seemed to me that the English coroner's system suffers in comparison to that operating in Scotland by overwhelming the necropsy service with patently natural deaths which, if dissection is to be carried out, would be better routed through academic departments of pathology. To apply a "14-day rule" which is not dictated by regulation would seem to make things even more difficult.

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¹ Davies MRR. *The law of burial, cremation and exhumation*. 4th ed. London: Shaw and Sons, 1976:78.

Plasmapheresis in the haemolytic-uraemic syndrome in children

SIR,—The case report by Dr T J Beattie and others (23 May, p 1667) prompts us to report a similar case of oliguric haemolytic-uraemic syndrome (HUS) in a child who recovered fully after plasma exchange.

At the age of 4 years she had her first episode of HUS manifest by anaemia (haemoglobin 4.7 g/dl), thrombocytopenia (platelet count $50 \times 10^9/l$ ($50000/mm^3$)), and renal failure (plasma creatinine 345 $\mu mol/l$ (3.9 mg/100 ml)). With prednisolone, antibiotics, heparin, and dipyrindamole she recovered fully without the need for dialysis.

Four years later, at the age of 8 years, she had her second episode of HUS, occurring two weeks after treatment for an ascaris infestation (facial oedema, urticaria, and eosinophilia). Her progress is shown on the accompanying figure.

In view of the debate about the presence of circulating triggers,¹ an as yet unidentified plasma deficiency,² or both, we carried out sequential plasma exchange. Initially plasma exchange was carried out replacing 1.5 l of the patient's plasma with 1.5 l of human serum albumin (HSA)

Effect of plasma exchange using human serum albumin (HSA) followed by fresh frozen plasma (FFP) on plasma creatinine and platelet count in haemolytic-uraemic syndrome.

Conversion: SI to traditional units—Creatinine: 1 $\mu mol \approx 0.0113$ mg/100 ml.

(Buminate) on four consecutive days. After three days there had been no improvement, suggesting no circulating agent perpetuating the condition. The plasma exchange was then repeated using fresh-frozen human plasma, which was followed by prompt recovery of renal function (such that dialysis could be withdrawn) and recovery of the platelet count. This suggests that repletion of a missing factor (capable of allowing PGI_2 (prostacyclin) synthesis) present in fresh-frozen plasma but not in Buminate was responsible for recovery.

Although we have not tested the ability of the patient's plasma to support synthesis of prostacyclin in vitro, we feel that plasma exchange may be helpful in HUS and related conditions such as thrombotic thrombocytopenic purpura and postpartum acute renal failure if only as a convenient method of infusing large volumes of fresh-frozen plasma into an oliguric patient to replete deficient plasma factors. We feel that plasma exchange was responsible for the clinical improvement in this patient, who falls into a poor prognostic group in that she was older and had had a previous attack. She had failed to respond to aspirin and dipyrindamole given for 10 days prior to plasma exchange and then subsequently became oliguric.

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¹ Anonymous. *Lancet* 1979;ii:890-1.

² Remuzzi G, Misiani R, Marchesi D, et al. *Lancet* 1978; ii:871-2.

University budgets and medical education

SIR,—I have believed for a number of years that the cost of clinical education for undergraduates must be more expensive than that of preclinical, but reading the article by Dr Anne Grüneberg on university budgets and medical education (13 June, p 1987) persuaded me to reassess the reality of that statement.

From the staffing point of view there are remarkably few people who are exclusively retained for the purpose of clinical teaching. The vast majority, particularly in peripheral hospitals, have a strictly clinical role and teaching is a very minor consumer of time. This is very different from preclinical teaching, where each specialty has a number of lecturers whose central role is that of educating the undergraduates. There are comparatively few teaching-orientated clinical departments, and