Preference is given to letters commenting on contributions published recently in the *JRSM*. They should not exceed 300 words and should be typed double spaced

Coronary heart disease in Africa

The review by Dr Walker and Dr Sareli (January 1997 *JRSM*, pp 23–27) confirms the rarity of coronary heart disease (CHD) in South African blacks despite a high prevalence of diabetes and an increasing prevalence of other accepted risk factors. This paradox applies also to Afro-Caribbeans in Britain, Pima Indians and Sri Lankans, who have a high prevalence of diabetes but are not excessively prone to CHD[1–3].

These four populations have in common a low prevalence of persistence of intestinal lactase activity in adulthood (<40%) and a low or relatively low intake of milk[1,4]—or, in the case of some South African urban blacks, a preference for sour (i.e. low-lactose) milk. In contrast, the north Indian and Pakistani populations, who are prone to CHD as well as to diabetes, have a high prevalence of persistent lactase activity (67–100%) and a high intake of milk[1].

In populations not unduly prone to diabetes, CHD is similarly uncommon in those who have a low prevalence of persistent lactase activity and a low intake of milk, notably the Chinese, Japanese and Greenland Eskimos[1].

In Europe, the Mediterranean prevalence of persistent lactase activity is mainly intermediate (40–70%), and the 'Mediterranean diet' is low in milk and lactose compared to northern Europe, where the average prevalence of persistent lactase activity is about 90%. With regard to the French paradox, the prevalence of persistent lactase activity in France is 60–80% and the intake of milk is much less than in Britain, while the intake of butter and cheese, which have negligible content of lactose, and of dairy fat are much higher[1].

I suggest that Walker and Sareli, and other researchers on interethnic variations in the occurrence of CHD, should include lactase-activity status and intake of dairy products, with an estimate of lactose intake, in their investigations on the occurrence of the condition both within and between ethnic groups.

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Table 1 **Changing proportions of heart disease (HD) in Shanghai**

Types	*1948–1958 (%)*	*1959–1971 (%)*	*1972–1979 (%)*
Ischaemic HD	6	13	29
Rheumatic HD	50	40	26
Congenital HD	4	9	15
Myocarditis	1	2	8
Chronic cor pulmonale	8	11	6
Hypertensive HD	16	12	5
Cardiomyopathies	1	1	3
Pericarditis	2	2	1
Syphilitic HD	7	2	1
Thyroid HD	2	1	1
Others	3	2	5

Table 2 **Mean serum cholesterol of free-living Chinese, 1958 and 1981**

	1958		*1981*	
Age group (year)	*No.*	*Mean (mg/dL)*	*No.*	*Mean (mg/dL)*
Men	43	142.7	59	168.41
20–29				
30–39	68	154.9	39	180.80
40–49	97	154.4	50	205.64
50–59	54	168.9	42	205.11
Over 60	23	155.4	21	212.48
Total	285	155.3	211	191.21
Women	56	151.5	199	173.87
20–29				
30–39	52	149.1	109	176.98
40–49	55	160.1	152	197.73
50–59	45	173.3	57	213.40
Over 60	16	168.4	—	—
Total	224	158.64	517	185.9

Dr Walker and Dr Sareli describe several observations that are very reminiscent of what has happened in China. In 1941 Snapper[1] pointed out the rarity of coronary artery disease in China. But this is no longer the case in modern China, where among types of heart disease ischaemic heart disease moved from fifth place in 1948–1958 to number one in 1972–1979 (Table 1)[2]. Although part of the explanation lies in longer lifespan, less malnutrition and fewer infectious diseases, the principal reason is the increasing access to the harmful habits of Western society—i.e. high fat and high cholesterol food, cigarette smoking, lack of physical exercise due to increased mechanization, and stresses of urban living. That diet plays a major part is evidenced by the progressive rise of 'normal' serum cholesterol levels of free-living Chinese from 1958 to 1981 (Table 2)[2].

One shudders at the alarming statistics cited by Peto[3] that one of every three cigarettes manufactured in the world is consumed by the people in China. According to the Minister of Health, People's Republic of China, China ranks as the first in the world in its population and also in output of tobacco products[4]. Although all the cigarettes in China now carry a health hazard warning on their packages[5], smoking is still rampant, especially among the young (as high as 40% among high school students[4]).

Thus, one can either die from a heart attack due to coronary atheroma or succumb to malnutrition through poverty.

Can we achieve a utopian stage in the 21st century in which the Africans and the Chinese retain their ancestral low rates of cardiovascular disease while preserving the positive aspects of a Western lifestyle?[6].

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Euthanasia

Although Dr Black (December 1996 *JRSM*, pp 722–3) describes involuntary euthanasia in the Netherlands as 'worrying' and possibly signalling the beginning of a slippery slope, he does not seem really to object. I find this very surprising. Indeed this involuntary euthanasia is contrary to the oath of Hippocrates and leads to a new definition of the function of medical doctors. So far the medical doctors always fought for the life of the patient. In the Netherlands the doctor may now administer life or death. The term 'involuntary euthanasia' is for me totally unacceptable. It is murder.

I am perplexed by Dr Black's exegesis on German doctors. Because I am a German doctor in the fourth generation these statements are addressing me in a very personal way. First, I would like to point out that the generalization that 'German doctors progressed from involuntary euthanasia to the gas chambers of Auschwitz' can not be accepted. It appears as though the German Medical Association was in charge of Auschwitz. It is a sad truth that mentally ill persons were murdered during the Hitler regime and that this was based on a repulsive pseudo-medical reasoning. However, Auschwitz was a mass murder without medical implications.

With reference to the situation in the Netherlands, a recent evaluation showed that 60% of all cases of euthanasia were not reported to the authorities as required by law and it remains uncertain whether the other conditions for euthanasia were met, such as the repeated and explicit desire of the patient to die and consultation with a colleague (*Deutsches Ärzteblatt* 1995, B2160 and 1996, B2588–89). The slippery slope begins here.

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Haemoglobin and ESR

The paper by Dr Kanfer and Ms Nicol (January 1997 *JRSM*, pp 16–18) brought back happy memories of a student project I undertook in Newcastle in the early 1970s, under the supervision of Professor David Kerr. The task was to determine why patients with uraemia had high erythrocyte sedimentation rate (ESR). It had been known from the classical studies of Robin Fahreus[1] that anaemia increased the blood sedimentation rate, and since patients with uraemia commonly have a normochromic, normocytic anaemia, it was first necessary to define the contribution of a reduced haematocrit to the ESR in individual patients. Blood samples in EDTA taken from healthy individuals were centrifuged gently to separate red cells and plasma, and calculated volumes of cells or plasma were removed to produce blood with haematocrits between 10 and 50%. ESRs were then measured by the Westergren method[2] and plotted against the haematocrit. The relationship (Figure 1) proved to be a negative exponential function. While the relationship between ESR and [Hb] shown by Kanfer and Nicol is shown as linear, the extrapolation to low haemoglobin levels may be erroneous. Indeed, their graphs indicate many data points above the regression line at haemoglobin concentrations of < 10 g/dL.

I also studied the reason that the normal ESR is so low. Theoretical studies by Ponder[3] in the 1920s suggested that, when blood begins to settle in a tube, it passes rapidly from an initial turbulent state in which the red cells are discrete, to a state in which rouleaux of 20 or so cells form. Using a specially constructed ESR tube with rubber-sealed ports, I was able to sample

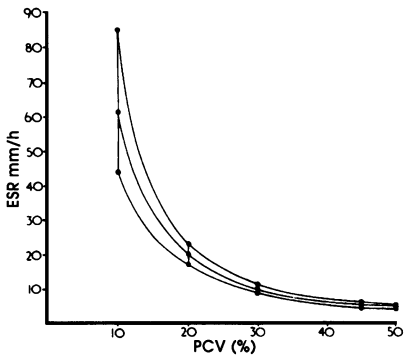

Figure 1 **Regression line relating erythrocyte sedimentation rate (ESR) and packed cell volume, with 95% confidence intervals**

and photograph cells in the tube at various depths and times and found that, under normal circumstances, the standing column of red blood cells in an ESR tube appears as a 'scaffolding' of branched rouleaux which then settles to a degree, giving the low, normal ESR. In conditions such as uraemia, the 'suspension stability of the blood' decreases; the process of orderly rouleaux formation breaks down, the red cells clump together and sediment rapidly, giving an increased ESR. The increased ESR in uraemia, incidentally, seemed to result mainly from an increase in plasma fibrinogen concentration.

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The paper by Dr Kanfer and Ms Nicol purports to show a meaningful inverse correlation between the Hb and the ESR for non-anaemic patients. May I criticize their interpretation of the correlation coefficients?

First, the authors have not used 95% confidence intervals for the correlation coefficients. The procedure is discussed by Altman and Gardner[1] and tables relating to Fisher's z statistic are available in *Documenta Geigy*[2]; their use involves only simple