this might not be the whole story: "The answer may well be more complicated." A very recent review¹ of the epidemiology of heart disease supports both comments.

In the course of the review I analyse the sex and age patterns of mortality from "ischaemic heart disease" (8th ICD 410-414) in five populations: England and Wales, Scotland, US whites, US non-whites, and Japan. Defined in this way, ischaemic heart disease is heterogeneous, comprising three main components. Most deaths over the age range 30-75 years can, however, be attributed to one component, "acute myocardial infarction" (8th ICD 410). I deduce that genetic factors play two distinctive roles where acute myocardial infarction is concerned. Firstly, genes determine whether or not a person is at risk to acute myocardial infarction but, once the disease process has started, other genes are the main factors involved in determining its average duration, or latent period, from initiation to death. In males the average latent period ranges from about seven years in US non-whites to 13 years in England and Wales; in females these intervals are twice as long. Seasonally dependent environmental factors can exacerbate pathogenesis and hasten death but they have little influence on the average duration of the latent period.

A most interesting feature of the epidemiology of ischaemic heart disease, and one that has attracted much attention, is the recent fall in mortality recorded in several countries, including the United States. By comparing the secular trends in overall mortality with those recorded for mortality from ischaemic heart disease in US whites and US non-whites, I conclude that the fall is probably largely or wholly genuine. If it is, then the characteristics of the decrease in relation to sex and age implicate one or more extrinsic precipitators, whose impact has been declining for a decade or more. Classical precipitators are infective micro-organisms and allergic agents.

The evidence can be readily interpreted in terms of autoaggressive theory.¹ Once random intrinsic changes have occurred in a genetically predisposed person to produce a mutant stem cell, a precipitator appears to be needed to trigger the growth of a forbidden clone, the peripheral cells of which attack the endothelial lining of coronary arteries. A search for the precipitator or precipitators of ischaemic heart disease might well prove rewarding.

My analysis implies, I should add, that the "risk factors"—hypertension, hypercholesterolaemia, obesity, smoking, and lack of exercise, have little or no causal action; their association with ischaemic heart disease appears to be predominantly genetic in origin.

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

Hyperlipidaemia advances and retreats

SIR,—As co-ordinator of the Persantine (dipyridamole) Aspirin Reinfarction Study and as co-principal co-ordinator of the Aspirin Myocardial Infarction Study referred to in your leading article entitled "Hyperlipidaemia advances and retreats" (2 August, p 340), I would like to offer the following corrections to

the statement that "Both the Aspirin Myocardial Infarction Study and the Persantine Aspirin Reinfarction Study had failed to show any benefit, and the main positive finding was a surprisingly high incidence of aspirin-related side effects." The Aspirin Myocardial Infarction Study showed no benefit in total mortality over a period of 42 months but did show modest benefits in coronary mortality and coronary incidence that were not statistically significant.¹ The Persantine Aspirin Reinfarction Study showed favourable effects both for the pure aspirin group and for the combination group for total mortality and coronary mortality of 17% and 25% respectively.² By our conservative statistical standards—that is, $z \leq 2.6$ ($p \leq .01$)—these differences were not significant. For coronary incidence, however, the Persantine plus aspirin combination was highly significantly different from placebo for the first two years of treatment for the endpoint coronary incidence (coronary mortality plus definite non-fatal myocardial infarction). Aspirin alone, while less effective, showed significant differences for the same endpoint at months 8 and 24 of treatment.

Side effects due to a daily dose of 1 g of aspirin were indeed not inconsiderable, mainly owing to increased bleeding tendency and some laboratory indications of kidney damage. However, no patient's life was lost because of that. Side reactions attributable to Persantine were limited to an excess of headaches.

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¹ Persantine-Aspirin Reinfarction Study Research Group. *Circulation* 1980;62:449-61.

² Aspirin Myocardial Infarction Study Research Group. *JAMA* 1980;243:661-9.

Sodium and potassium intake and blood pressure

SIR,—One of the conclusions of Dr Peter Brustyn and colleagues (23 August, p 537) was that small reductions of sodium intakes by Western populations are unlikely to lower blood pressure levels. Although agreeing with this opinion it should be pointed out that this conclusion was not derived from their experiments, for these did not include the decreasing of sodium intakes.

A recent comprehensive review¹ of population sodium intakes suggested four groups having a different range of consumption levels: (1) Very low sodium intakes, less than 30 mmol sodium (NaCl 2 g) per day, consumed only by hunter gatherers and primitive peasant groups who add no salt to their food; among them blood pressures do not rise with age and essential hypertension is virtually unknown. (2) Customary sodium intakes, ranging from 90 to 200 mmol per day, as in all Western and almost all Eastern populations who add salt to their food; among them blood pressures rise with age. This range of sodium intake permits the expression of hypertension in the genetically susceptible minority, roughly 10-20% of middle-aged persons. (3) Very high sodium intakes of 600-800 mmol per day; these levels have been consumed only for a few days as experimental procedures; they cause blood pressure levels to rise. This review had less to report concerning group 4, the intermediate group having sodium intakes ranging from 30 to 90 mmol per day—that is, intermediate in sodium consumption between groups 1 and 2. This intermediate level of sodium intake has been reported in Samburu and Maasai herdsmen who drank much sodium-containing milk, in Tarahumara Mexican Indians, in Ojong Java Pacific islanders, in Hadza Tanzanian newly

settled nomads who took salty drinking water, and formerly in Kikuyu Kenyan women who added plant ashes from brackish swamps to a no-added-salt diet.² All these groups had virtually no hypertension and no rise of blood pressure in adult life.

Considerable interest must therefore be taken in the results of experiments to study hypertensive patients who had eaten the customary sodium intakes of group 2 and were switched to the lower (intermediate) sodium intakes of group 4. They were not switched to the intolerable low sodium levels of group 1; these are known to reduce hypertensive blood levels. Three reports have recently stated that the majority of essential hypertension patients who had intermediate sodium intakes reverted to normal diastolic blood pressures and ceased all hypotensive drugs, usually within four to eight weeks, while consuming 50-70 mmol of sodium a day.³⁻⁵ These diets involved the exclusion of a short list of highly salted foods. Patients came to accept the change, and even refused to revert to their previous dietary habits when offered the chance; eventually most came to dislike highly salted foods. It is hoped that these preliminary data will be assessed in more sophisticated trials and also will investigate protective factors such as potassium intake, supported by one reference to an experimental study in the review.¹

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¹ Tobian L. *Am J Clin Nutr* 1979;32:2739-48.

² Trowell HC. *E Afr Med J* 1980;57:167-73.

³ Magnani B, Ambrosioni E, Agosta R, Racco F. *Clin Sci Mol Med* 1976;51, suppl:625-6.

⁴ Dodson PM. *Br Med J* 1980;280:564.

⁵ Mannerberg D. *Chest Heart Stroke J* 1979;3:62-5.

Three types of health education

SIR,—Ever since St Gregory the Great ("Non angli sed angeli"), it has been traditional in this country to debate classifications proposed by others. In this vein may we question the typology of health education proposed by Dr Peter Draper and others (16 August, p 493), which we find confusing? Surely it is important to distinguish clearly the target group, the individual or society, from the objective—be it primary, secondary, or tertiary prevention. Rather than three types of health education, conceptually are there not a minimum of six?

Whatever the terminology, we are in total agreement that "social" rather than "personal" health education, promoting social responsibility in policy affecting health, is an unexploited activity. Part of the reason may be that the ground rules have changed. One-man campaigns in the nineteenth century public health tradition are insufficient against the increasingly resourceful forces at work today. New approaches are certainly required and perhaps one should take the form of highly sophisticated regional initiatives.

The authors call for a reappraisal of the most effective means of organising health education. In Wessex a multidisciplinary regionwide positive health team has just been formed on an experimental basis. Utilising the media to the full, it aims to emphasise individual and collective responsibility for good health. As a first exercise, it has investigated the effectiveness of the very best "personal" health education in promoting the voluntary use of car seat belts in a defined community.¹ The disappointing response observed confirms the need for "social" health education