safe and practicable intervention in those whose test result is positive.

Venepuncture in small children is not easy and many general practitioners would balk at performing it. Serum cholesterol measurements are not known for their high reproducibility from day to day. However my main concern is the intervention, which is scarcely mentioned in the report. What would the committee recommend?

Diet? Imposing a strict diet on young children is very difficult; as affected children grow up their diet tends to be inflicted on the whole family, whether they need it or not. Teenagers wish to be like everyone else of their own age and are liable to break their dietary restrictions, and whilst there is some circumstantial epidemiological evidence there is no actual evidence that reducing childhood cholesterol reduces IHD in later life.

Drugs? None is recommended in children of two years upwards since none has been adequately tested in this age group. We need a large and long term controlled trial before assuming that the expected benefits are delivered and are not outweighed by the side effects. It must be continued long enough to show (or exclude) difference in mortality. Some have dismissed the increased incidence of violent death during drug therapy of hyperlipidaemia as non-significant but it has been observed in several trials and cannot be lightly dismissed; the biggest lump of cholesterol in our bodies is in our brains. Even lowering cholesterol in the upper normal range could conceivably have ill effects. New diabetics can become hypoglycaemic with a blood dextrose over 7 mmol/l; can we be sure that the brain is not similarly sensitive to a change in the plasma cholesterol to which it is accustomed?

I suggest that until such trials have been conducted we recommend screening of those at risk in the Easter term before GCSE when it is easy to take an adequate sample and to repeat it in a couple of weeks. A full lipid profile would be necessary to avoid missing those with a very low HDL. We need separate guidelines for the Asian population in whom a serum cholesterol over 5.2 mmol/l in adults is distinctly dangerous.

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Treating high blood pressure following acute stroke

We were interested to read the acute stroke management guidelines by Bath *et al.* proposing that high blood pressure (BP) should be treated two days post stroke (January/February 1996, pages 13–7). High BP post stroke is a difficult area. BP rises after stroke, this rise being greatest in those with previous hypertension and those with intracerebral haemorrhage. Britton *et al.* measured BP in patients with acute stroke and matched controls and found the incidence of high BP was almost double in the stroke patients. They also noticed a spontaneous decline in BP over the first four days [1]. Harper *et al.* also found BP dropped for up to one week after stroke [2]. In Britton's observational study no correlation was found between severe levels of high BP and the progression of the symptoms of stroke.

Cerebral auto-regulation is impaired in ischaemic tissue [3]. Animal models have shown that there is an 'ischaemic penumbra' of potentially salvageable neurones around the area of severe ischaemia provoked by arterial occlusion where cerebral blood flow (CBF) is dependent upon systemic arterial pressure. In these regions, small reductions in systemic arterial pressure may be sufficient to lower CBF to lethal levels [4]. Although the extent of such a penumbra in human studies has not been established there is at least a theoretical argument for the avoidance of lowering systemic arterial pressure in the acute phase post stroke.

References

1 Britton M, Carlsson A, de Faire U. Blood pressure course in patients with acute stroke and matched controls. *Stroke* 1986;**17**:861–4.
2 Harper G, Castleden CM, Potter JF. Factors affecting changes in blood pressure after acute stroke. *Stroke* 1994;**25**: 1726–9.
3 Strandgaard S, Oleson J, Skinhoj E. Autoregulation of brain circulation in severe arterial hypertension. *Br Med J* 1973;**1**:507–10.
4 Powers WJ. Acute hypertension after stroke: The scientific basis for treatment decisions. *Neurology* 1993;**43**: 461–7.

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In response
Treating high blood pressure following acute stroke

We thank Keir *et al.* for raising the controversial issue of blood pressure management following acute stroke and fully agree with their message. Our practice for managing blood pressure has changed since our paper describing the King's College Hospital Acute Stroke Unit (January/February 1996, pages 13–7) was originally written, partly as a result of the editorial by O'Connell and Gray on this subject [1]. However, in spite of the observational, and therefore indirect, evidence that Keir and colleagues, and O'Connell and Gray quote, no large randomised controlled trials of blood pressure management in acute stroke have been undertaken and it remains unclear whether we should actively lower, or even elevate, blood pressure.

In an attempt to rectify this deficiency, we are co-ordinating a collaborative systematic review ('Blood pressure in Acute Stroke Collaboration', BASC) within the Stroke Review Group of the

Cochrane Collaboration of trials in acute stroke where vasoactive drugs were administered and where blood pressure and outcome were measured [2]. We hope that this project will identify whether actively reducing or increasing blood pressure influences outcome, and if beneficial what trial protocol needs to be assessed in a definitive study.

In the meantime, we support the view that blood pressure should not be therapeutically altered for the first week after stroke unless co-existing hypertensive encephalopathy, heart failure or ischaemia, or aortic dissection are present, or continued intracerebral bleeding occurs [3]. However, mechanisms need to be set-up by those caring for stroke patients to ensure that patients who remain hypertensive are adequately treated long-term to prevent further vascular events.

References

1 O'Connell JE, Gray CS. Treating hypertension after stroke. *Br Med J* 1994;**308**:1523–4.

2 Bath P, Bath F. Blood pressure management in acute stroke. [revised 27 July 1995] In: Warlow C, Van Gijn J, Sandercock P (eds) Stroke Module. In: The Cochrane Database of Systematic Reviews [database on disk and CD-ROM]. The Cochrane Collaboration; Issue 2, Oxford: Update Software; 1995.

3 Yatsu FM, Zivin J. Hypertension in acute ischaemic strokes. Not to treat. *Arch Neurol* 1985;**42**:999–1000.

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In response
Senescence, cancer and endogenous parasites

Editor—Professor Kay-Tee Khaw takes me to task in the Letters column (March/April 1966, page 189) for the alleged 'nihilism' of my article entitled 'Senescence, cancer and endogenous parasites' (January/February 1996, pages 10–2). I had always imagined that the criterion for judging a scientific hypothesis was its truthfulness, rather than its cheerfulness.

On such a basis we would presumably be required to reject Darwin's theory of natural selection itself, as my own work is based on this. Bernard Shaw famously did so in his preface to *Back to Methuselah*. Shaw declared that Darwin had 'banished mind from the universe', that natural selection was morally repugnant and therefore couldn't be true, and that we should instead subscribe to Shaw's own optimistic and progressive creed of Creative Evolution. Shaw was a dramatist of genius, and his views may be sound metaphysics or politics, but they are bad science nevertheless—natural selection has proved an unmatched source of detailed and counter-intuitive predictions of phenomena at many levels of biological organisation, from the single gene right up to complex forms of social organisation in animals.

I was also vexed by Professor Khaw's statement that my hypothesis of 'endogenous parasitism' was a mere platitude, stating no more than that 'we all have to die of something sometime'. In fact, the theory makes highly specific predictions concerning age-related changes in replicating cells and organelles, which are amenable to test by experiment.

Endogenous parasitism represents the difference between rapid replication and wear and tear, between cancer and degeneration, between parasites and debris. These seem large and significant differences to me, and ones that are readily measurable too; although either or both might be cause of ageing in any given instance.

The issue of 'when, how, and what we can do to improve things' for people suffering the effects of ageing is heavily dependent upon our understanding the nature of the processes of senescence. Endogenous parasitism is a real phenomenon with the potential to be a cause of ageing in humans and other organisms. Whether or not it turns out to be an *important* cause is a matter to be determined by scientific investigations—not a matter to be pre-decided by an expression of moral disapproval.

Reference

1 Maynard-Smith J, Szathmary E. *The major transitions in evolution.* Oxford: W H Freeman Spektrum, 1995.

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Medicine and elderly people: over-investigation or under-treatment

Editor—The surgical management of age-related aortic stenosis is long overdue for decision analysis [1], if only on the basis of the issues raised by Professor Treasure in the above conference [2] (March/ April 1996, pages 170–3). Hitherto, operative mortality has been the only consideration [3], despite the fact that this is much less than the two-year mortality risk attributable to the natural history of clinically significant aortic stenosis [2]. It is therefore illogical to argue that co-existing manifestations of aortic valve-related systemic hypo-perfusion, such as mental and physical lassitude, render aortic valve replacement needlessly hazardous, because, as in *Hamlet*, 'disease desperate grown, by desperate appliance are relieved, or not at all' [4]. Contrariwise, the risk of operative intervention is not to be dismissed when the stigmata of systemic hypoperfusion are absent, because the patient who is in otherwise excellent mental and physical condition stands to lose much more when untimely curtailment of life expectancy supervenes. Therefore, in the absence of significant comorbidity (unrelated to aortic valve disease), the indications for aortic valve replacement should apply to all age groups with aortic stenosis, since the two year mortality risk of 80% attributable to conservative management [2] vastly exceeds the current statistics