America, Australasia, Africa and Asia. This is perhaps one of those 'teachable moments' for physicians to bring their own knowledge on prion diseases up to date so that they can help their family, friends and patients to weigh the risks, assess the data provided by the CJD surveillance unit in Edinburgh (do other countries have one?), and themselves be able to evaluate and implement the results of research on prion diseases when they become available.

ROBERT MAHLER
Emeritus Editor
Journal of the Royal College of Physicians

Statistical Malpractice

Editor—The comment cited by Charlton (March/April 1996, page 112), that 'if statistics are needed, you should go back and do a better experiment' reminds me of a delightful anecdote about the late Dame Harriet Chick FRS, who put it another way when she remarked that 'our results were not significant, so we had to use statistics'.

LESLIE COLLIER
Regent's Park, London

Screening for hyperlipidaemia in childhood

Editor—The recommendations of the British Hyperlipidaemia Association (March/April 1996, pages 115–8) note that there are few published data on age and sex related norms for lipid levels for children in the UK. We draw to their attention some data we have collected on this issue.

During the course of a project on iron deficiency anaemia in childhood we measured the cholesterol levels on 579 children between the ages of 4 and 13 years of age. The specimens were taken at mid-morning in schools. None of the children was known to have a history of hyperlipidaemia. The mean serum cholesterol level was 4.46 mmol/l (range 2.43–7.00). There was no sex difference and no correlation between age and serum cholesterol, (r=0.007). There were not enough children from individual racial groups to establish race specific norms. The 95th centile for non-fasting cholesterol was 5.71 mmol/l.

We are also aware of other publications on this question which may help to establish the likely effect of implementing the committee's recommendations [1,2]. The committee recommends selective screening of non-fasting cholesterol and further investigation if the result is >5.5 mmol/l. These give no indication as to the likely prevalence of children at risk. Our findings suggest that 6.4% (33/579) of children fulfil this criterion. The likely response to such a screening programme would need to be clarified and the respective responsibilities of family members and professionals is important [3]. It is not clear who it is hoped might implement such screening and without some indication of the magnitude of the task in hand it may be that the committee's objectives will be frustrated. The issues of implementation have been summarised recently [4]. The second stage of the screening programme includes a fasting cholesterol cut off point of 6.7 mmol/l. Since fasting cholesterol is likely to be lower than non fasting, our findings would suggest that the percentage of children who fulfil this criterion may be as few as 0.3% (2/579) of children, supporting the relative specificity of this stage of the screening procedure.

The practical implications of this diagnosis are only briefly alluded to. Personal experience as a school doctor (JO'D) suggests that the management of such problems must take account of the reality of school life and particularly teachers' difficulties in understanding medical conditions. Questions asked under these circumstances have included: 'are such children able to participate normally in physical activity without extra risk of myocardial infarction?' and 'what should I do when the inevitable 'cheating' on diet occurs?' Diet in school is often fashion-led. When parents become aware that a child at risk of cholesterol problems takes a particular diet they may conlude that it is a 'healthy' diet and impose it on normal children, not necessarily to their advantage. It would be of greater value if some of the practical implications were to be spelt out in greater detail. Paediatric involvement might also be useful. For example there are efforts being made to ensure the appropriate administration of medications in schools and some of the lessons learnt from this might be applicable.

References

1 Armstrong N, Balding J, Gentle P, Kirby B. Serum lipids and blood pressure in relation to age and sexual maturity. *Ann Hum Biol* 1992;**19**:4477–87.
2 Azad K, Court S, Parkin JM, Laker MF, Alberti KG. Lipid levels in schoolchildren in North East England: effects of feeding and age. *Ann Clin Biochem* 1994;**31**:2333–9.
3 Torstal S, Volleback LE, Ose L. Screening for familial hypercholesterolaemia in relatives. *Lancet* 1995;**346**:1438.
4 Boulton J. Screening of hypercholosterolaemia in childhood. *Annales Nestle* 1994;**52**:14–24.

J M O'DONOHUE
Queen Mary's Hospital, Roehampton, London
M R HANCOCK
G K BANERJEE
Chelsea and Westminster Hospital, London

Editor—We have read with interest the recommendations of the British Hyperlipidaemia Association but question whether they have made a case for screening the substantial groups they propose—all children with a first or second degree relative with either familial hyperlipidaemia or early onset coronary artery disease—particularly at an age of two years. Such screening ought to meet widely agreed criteria—the test should be easy to perform, accurate and reproducible and should lead to a

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.

K E PEARCE
J S CHARLTON
The Elmcroft Surgery, North Harrow

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.

S L KEIR
P J MURPHY
M MacMAHON
Directorate of Medicine
Bristol General Hospital

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