much the same way as advocated for cataract surgery.[7] Such a policy avoids the disorientating effects of a hospital admission and allows elderly patients to return to familiar surroundings as soon as possible.

I agree with the authors that patients need to be fully informed about available treatments. They should also be told that, in this instance, opting for surgery need not necessarily entail being subjected to either general anaesthesia or inpatient admission.

B R P BIRCH

Department of Urology,
Southampton University Hospitals,
Southampton SO9 4XY

1 Chadwick DJ, Gillatt DA, Gingell JC. Medical or surgical orchidectomy: the patients' choice. *BMJ* 1991;**302**:572. (9 March.)
2 Fitzpatrick R, Hinton J, Newman S, Scambler G, Thompson J, eds. *The experience of illness*. London: Tavistock, 1984:132-53.
3 Rosin AJ, Boyd RV. Complications of illness in geriatric hospital patients. *J Chronic Dis* 1966;**19**:307-13.
4 Rassam S, Thomas HF. Local anaesthesia for cataract surgery. *Lancet* 1989;i:110-1.
5 Birch BRP, Anson KM, Clifford E, Miller RA. Day-case surgery: enhanced recovery with flumazenil. *J Roy Soc Med* 1990;**83**:436-8.
6 Birch BRP, Anson KM, Miller RA. Sedoanalgesia in urology: a safe, cost-effective alternative to general anaesthesia. A review of 1020 cases. *Br J Urol* 1991;**66**:342-50.
7 Watts MT, Pearce JL. Day-case cataract surgery. *Br J Ophthalmol* 1988;**72**:897-9.

Amantadine for influenza A

SIR,—We would like to endorse the views of Drs Karl G Nicholson and Martin J Wiselka concerning the use of amantadine in addition to immunisation for influenza in high risk groups[1] and add a further point about the duration of prophylaxis. The meeting in Vienna convened by the World Health Organisation in 1984 to discuss the use of amantadine alone could find no alternative to a possible five to six week course of amantadine, which is approximately the course of an epidemic.[2,3] If high risk groups were not protected from influenza beforehand then immunisation could be performed after an outbreak but the response to this would take several weeks. Amantadine would therefore offer protection during this "window" period.

We have recently reported the findings of a pilot study to investigate how rapidly an antibody response occurs after immunisation.[4] Six healthy volunteers (age range 25-60) were immunised with the 1989-90 influenza season vaccine, and blood samples were taken before immunisation and on days 2, 6, and 8 after immunisation.

The six volunteers showed a good response to the vaccine by three weeks, but, interestingly, within eight days a twofold to fourfold rise in titres of haemagglutination inhibition antibody was found, which boosted the pre-vaccination levels considerably. In addition, two volunteers had detectable rises in haemagglutination inhibition antibody from ≤10 to ≥40 within two days and one within six days of being immunised (table). A haemagglutination inhibition antibody titre of ≥40 is deemed to confer protection.[5]

Haemagglutination inhibition antibody titres v homologous vaccine virus and an epidemic strain before and after vaccination with influenza virus

Subject	Virus	Titre before vaccination	Titre after vaccination		
			2 days	6 days	8 days
1	A/Taiwan/1/86	40	80	240	1920
	A/Shanghai/11/87	160	160	1280	2560
	A/England/308/89		80	120	320
	B/Yamagata/16/88	40	40	40	480
2	A/Taiwan/1/86	40	40	60	240
	A/Shanghai/11/87	160	160	160	1280
	A/England/308/89		40	40	40
	B/Yamagata/16/88	20	20	60	60
3	A/Taiwan/1/86	40	40	40	240
	A/Shanghai/11/87	320	320	320	640
	A/England/308/39		30	80	120
	B/Yamagata/16/88	20	20	20	20

A/Taiwan/1/86 (H_1N_1—vaccine component); A/Shanghai/11/87 (H_3N_2—vaccine component); A/England/308/89 (H_3N_2—epidemic strain); B/Yamagata/16/88 (vaccine component).

These responses were against influenza A/England/308/39 (H_3N_2 strain), which was a representative epidemic strain in the United Kingdom from the 1989-90 season. This is presumably an anamnestic immune response to previously circulating H_3N_2 viruses, but nevertheless the antibodies reacted with that year's epidemic virus. The study has been extended this winter season to encompass four different age groups, including the elderly, to assess which immunoglobulin subclasses are important in the first days after immunisation and whether this rapidly produced antibody is protective. This could have important implications for immunising people at risk after the outbreak of an epidemic if they have inadvertently missed the autumnal immunisation recommended by the Department of Health. In last year's epidemic 26 000 excess deaths were recorded; this warns us that influenza as a disease can still surprise us and we need to exploit preventive measures to the full.

M A ZUCKERMAN
J S OXFORD

Department of Medical Microbiology,
The London Hospital Medical College,
London E1 2AD

1 Nicholson KG, Wiselka MJ. Amantadine for influenza A. *BMJ* 1991;**302**:425-6. (23 February.)
2 National Institute of Allergy and Infectious Diseases. Amantadine: does it have a role in the prevention and treatment of influenza? A National Institutes of Health consensus development conference. *Ann Intern Med* 1980;**92**:256-8.
3 Galasso GJ, Bektimirov TA, Douglas RG, Krylov VF, Oxford JS. Current status of amantadine and rimantadine as anti influenza agents: memorandum of a WHO meeting. *Bull WHO* 1985;**63**:51-6.
4 Zuckerman MA, Wood J, Chakraverty P, Taylor J, Heath AB, Oxford JS. Serological responses in volunteers to inactivated trivalent subunit influenza vaccine: antibody reactivity with epidemic influenza A and B strains and evidence of a rapid immune response. *J Med Virology* 1991;**33**:133-7.
5 Hobson D, Curry RL, Beare AS, Ward-Gardner A. The role of serum haemagglutination inhibition antibody in protection against challenge infection with influenza A and B viruses. *J Hygiene* 1972;**70**:767-7.

Asymptomatic hypercholesterolaemia

SIR,—Ischaemic heart disease is now the commonest form of death in British adults, and coronary disease is one of the most important modern epidemics. We were disappointed that at a time when the risk factors for this condition are at last gradually being elucidated that Dr Gilbert R Thompson should use the negative image of "time bomb."[1] He is right to refer to developments in Canada and the United States, but he has omitted any reference to some of the most important and exciting research findings from North America—namely, those of Blankenhorn that reduction of blood cholesterol concentration can be associated with regression of coronary disease.[2]

It is not correct to refer to "the costs of treating perhaps one in four adults with lipid lowering drugs." Asymptomatic hypercholesterolaemia is a condition for general practice, where it will mainly be found and managed. We currently have 371 patients identified with this condition (cholesterol concentration ≥6·5 mmol/l) in our general practice (5% of the total population and 13% of patients over 40). We are doing this work in ordinary consultations with one full time equivalent practice nurse for 7035 patients. We find that nowhere near "one in four adults" needs drug treatment, and only 3·5% of all our patients are being prescribed a lipid lowering drug of any kind.

We agree that epidemiologists naturally take a population view, and as clinicians we do indeed take a different view—namely, that the very high contact rate of patients with British general practitioners (currently five consultations per patient per year[3]) makes case finding and management by opportunistic means both possible and logical in primary care.

There are advantages in generalists reviewing the whole patient and all relevant risk factors. Estimating cholesterol concentration is a relatively cheap test, and before commenting on "huge increases" and costs it is also necessary to estimate and balance the savings in premature loss of lives. Quality adjusted life years (QALYs) are one way of doing this.

Peto estimated that a 1% reduction in serum cholesterol will lead to a 3% reduction in coronary heart disease.[4] On this basis the average reduction of 7% that we are currently achieving through general practitioner advice without drugs is likely to lead to a 21% reduction in coronary disease.

DENIS PEREIRA GRAY
RUSSELL STEELE
KIERAN SWEENEY
PHILIP EVANS

Exeter EX1 1SF

1 Thompson GR. What should be done about asymptomatic hypercholesterolaemia? *BMJ* 1991;**302**:605-6. (16 March.)
2 Blankenhorn DH. Angiographic assessment of atherosclerosis during lipid-lowering therapy. *Cardiology* 1989;**76**:75-82.
3 Office of Population Censuses and Surveys. *General household survey 1987*. London: HMSO, 1989:11.
4 Standing Medical Advisory Committee. *Blood cholesterol screening: the cost effectiveness of opportunistic cholesterol testing*. London: SMAC, 1990.

Idiopathic first seizure in adult life

SIR,—Dr Cees A van Donselaar and colleagues suggest that the decision to initiate or delay anticonvulsant treatment after a first idiopathic seizure in adults should be based on electroencephalographic findings.[1] They report that those with electroencephalographic abnormalities are more likely to have further seizures. First seizures in adult life are common causes of referral to neurology clinics. Before departments of clinical neurophysiology expand to meet this suggested need several points should be considered.

Dr van Donselaar and colleagues cite three older studies in which those with electroencephalographic abnormalities had an increased risk of recurrence after a first seizure. They do not, however, cite a recent study by Hauser *et al*, who reported that no electroencephalographic abnormality other than generalised spike and wave discharges (unusual in adult life) was associated with recurrence.[2] The Royal College of Physicians' study on first seizures[3] recruited many more adult patients than did Dr van Donselaar and colleagues (408 *v* 165) and found no significant relation between recurrence and electroencephalographic abnormalities. What can account for the different findings of the two studies?

In the study by Dr van Donselaar and colleagues electroencephalography was performed a mean of 6·7 days after seizure, which may make a difference, but this interval is unlikely to be attained in the United Kingdom, bearing in mind the long waiting lists for neurological consultations and then appointments for electroencephalography. The authors report that "If the first electroencephalogram did not show epileptic discharges (spikes or spike wave complexes), electroencephalography was repeated after partial sleep deprivation in all but three eligible patients." They do not record how many patients required this second investigation. It is well recognised that a single electroencephalogram is only a time sample of cerebral activity and that more time spent in recording multiple electroencephalograms will increase the numbers of abnormalities found.[4] Such repetitive investigation is unlikely to find